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# A SEROLOGICAL - GENETIC STUDY OF IRANIAN AND NEIGHBOURING POPULATIONS

By

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A Thesis Submitted for the Degree of

Doctor of Philosophy

Department of Anthropology

University of Durham. U.K.

1983

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ABSTRACT

A total of 1709 blood specimens collected from ten population groups of Iran have been studied for genetic variations of some blood group, serum protein and red cell enzyme systems. The results were combined with those of the previous studies on other Iranian groups and compared with reported frequencies for neighbouring populations.

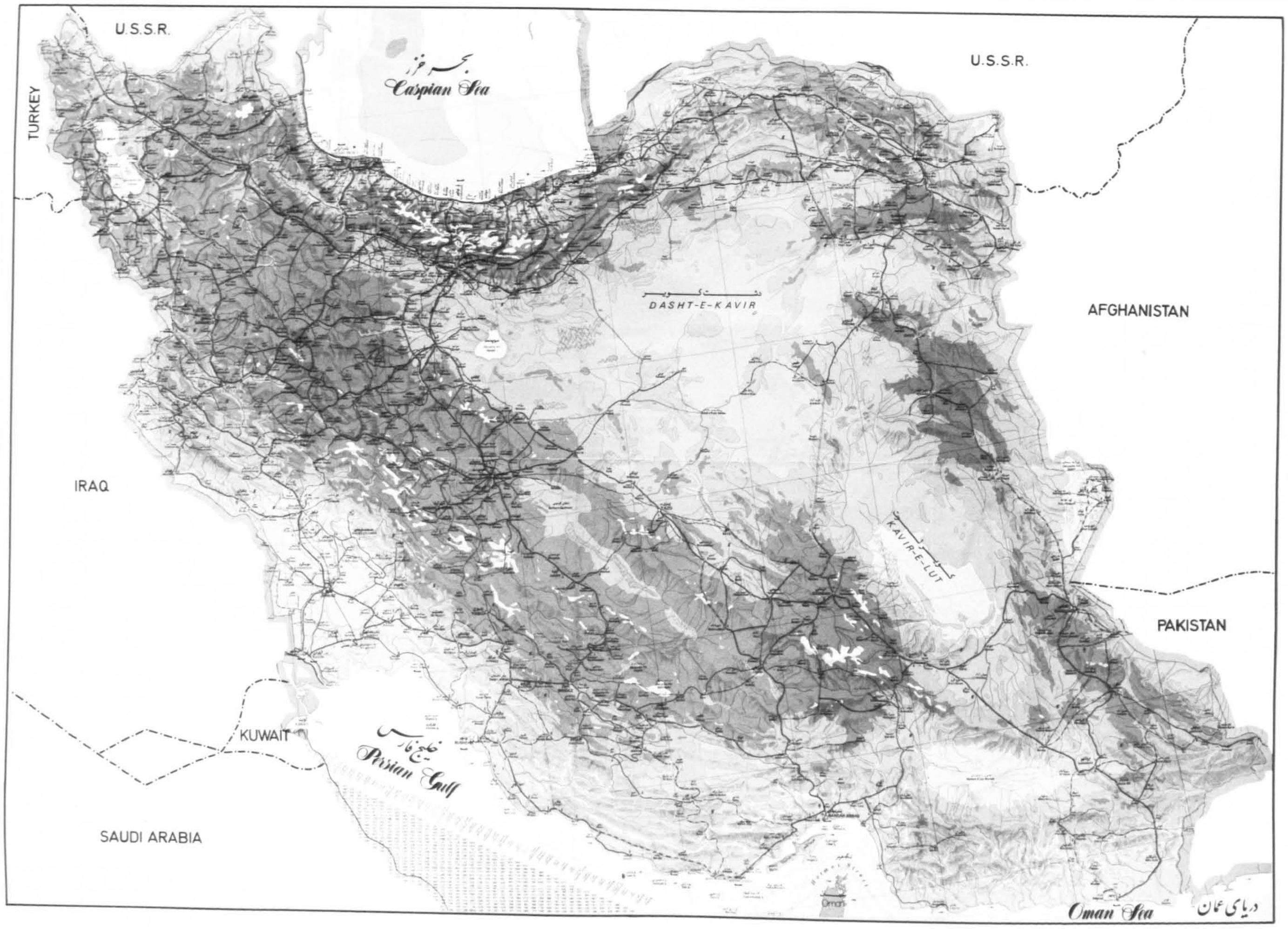
The average gene frequencies for Iran, in general, show a departure from the values found in the countries to the West and an approach to those of the Indian region. By contrast, the generalized feature of gene frequencies for both the Caucasus and Turkey show connections rather with the West than with the East.

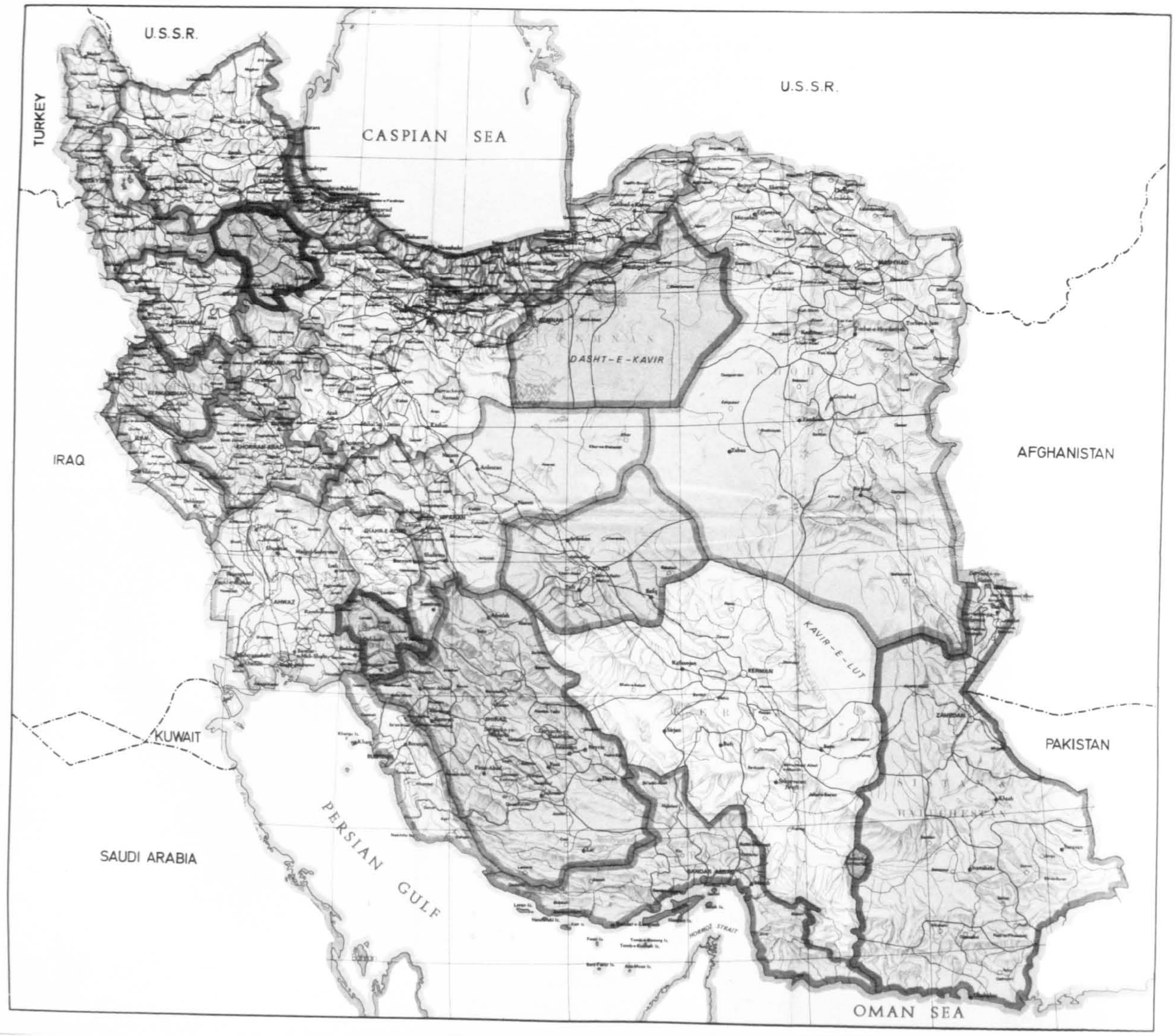
Taken as a whole the gene frequencies of the population of Iraq suggest Eastern rather than Western connections. The Arabs of Kuwait, Saudi Arabia and the United Arab Emirates, having high frequencies of African marker genes, show a considerable degree of admixture from Africans. It is notable that the Arabic speakers of South Western Iran show little resemblance to the Arabs of Arabia. On thewhole, the serological evidence suggests that over a long period of time there has been little migration into Iran across the Persian Gulf. The generalized feature of gene frequencies for Pakistan show relationships rather with India than with the neighbouring countries to the West. The average gene frequencies for the Afghans suggest both Caucasoid and Mongoloid characteristics.

*To :*

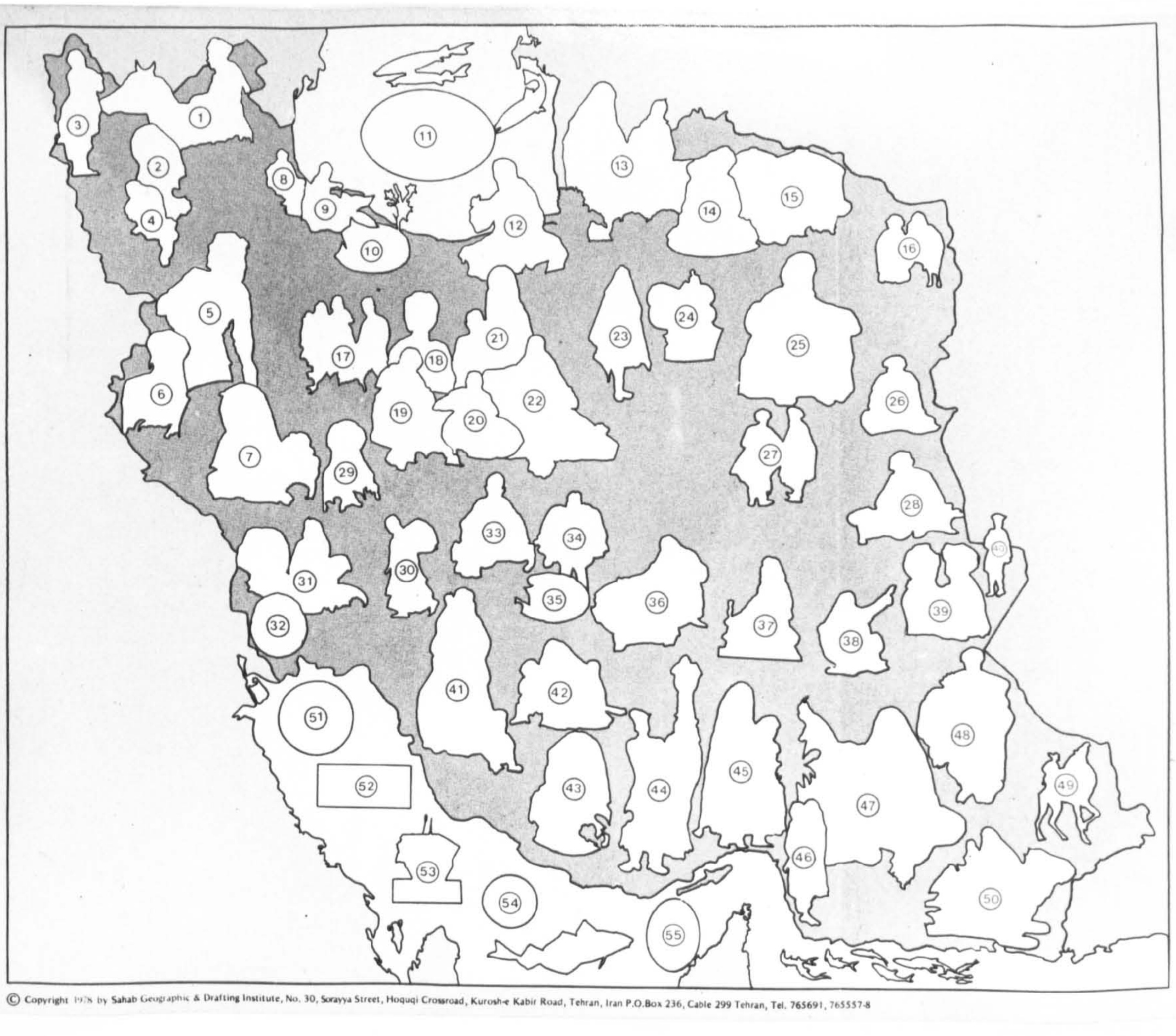
*My Country*




U.S.S.R.
U.S.S.R.
TURKEY
بحر خزر
Caspian Sea
دشت کویر
DASHT-E-KAVIR
AFGHANISTAN
IRAQ
KAVIR-E-LUT
PAKISTAN
KUWAIT
خلیج فارس
Persian Gulf
SAUDI ARABIA
Oman
Oman Sea
دریای عمان


U.S.S.R.
U.S.S.R.
TURKEY
CASPIAN SEA
DASHT-E-KAVIR
IRAQ
AFGHANISTAN
KAVIR-E-LUT
KUWAIT
PAKISTAN
SAUDI ARABIA
PERSIAN GULF
OMAN SEA





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# Ethno Composition

## LOCAL COSTUMES – HANDICRAFTS – CERTAIN AGRICULTURAL PRODUCTS

1. Shahsavan horse-rider
2. Rural girl from Eastern Azarbaijan
3. Rural woman from Western Azarbaijan
4. Old man from Oshnoviyeh strings tobacco for drying
5. Kurdish woman from the area around Sanandaj
6. Kurdish man from Javanrood area
7. Kurdish woman from the environs of Kermanshah
8. Kaviar-production, Bandar Pahlavi
9. Girl from Lahijan picking tea leaves.
10. Riceplanting by Gilani women
11. Fishing in the Caspian Sea
12. Beautiful local costume of the Northern Provinces
13. Carpet-weaving Turkomans (Yamut tribe)
14. Turkoman girl, sorting wool
15. Turkoman Yurt
16. Shepherd
17. Children of the Central Province
18. A city-girl (Tehran)
19. Qalamkar-maker (Isfahan)
20. Engraving a tray (Isfahan)
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23. Woman from Sanguesar-e Shahmirzad
24. Traditional transportation of straw
25. Man in local costume of northern Khorasan
26. Costume of the Barbar and Teymouri tribes of eastern Khorasan
27. Carpet pedlar in eastern Khorasan
28. Shoemaker from southern Khorasan
29. Woman of the Lor tribe from the environs of Khoramabad
30. Young Bakhtiary mother carries her baby
31. Bread-baking in the environs of Shushtar
32. Lovely girl from southern Khuzestan
33. Rural man from Isfahan area, spinning wool
34. Transportation of dyed wool for carpet-weaving, near Isfahan
35. Carpet-weaving girl, Isfahan
36. Woman from Yazd area spinning with an old spinning wheel
37. Woman from a village near the desert
38. Baluchi woman spinning wool
39. Two men from Sistan
40. Man from Zabol
41. Qashqa'i woman spinning wool in the old traditional way
42. Rural woman from the environs of Shiraz with her children
43. Woman from southern Fars Province
44. Qashqa'i camel driver
45. Woman and children from Bandar Abbas
46. Young girl from around Minab
47. Baluchi-woman weaving a net for the transportation of straw
48. Pedlar from Zahedan
49. Dromedary-rider
50. Two girls riding on a camel
51. Diving in the Persian Gulf
52. Trade-Vessel
53. Motor-Launch on the Persian Gulf
54. Sunset on the Persian Gulf
55. Pearl-diver

For my husband and my two children
Amir Ali and Nazli without whose
moral support, patience, love and
understanding this research could
never have been under taken.

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## NOTE ON THE TABLES

1- All the tables are arranged by order of gene frequencies.

2- All the tables of each serological system for nine countries in chapter 5 will have the same number and each country will take a code as follows:

| | |
|---|---|
| I | Iran |
| II | Caucasus |
| III | Turkey |
| IV | Iraq |
| V | Kuwait |
| VI | Saudi Arabia |
| VII | The United Arab Emirates |
| VIII | Pakistan |
| IX | Afghanistan |

3- The population numbers in the tables correspond to those on the maps.

## INTRODUCTION

There are obvious phenotypic differences between human populations throughout the world. Physical characteristics, such as skin, hair and eye colour, stature, and the shape of the cranium have long been used for purposes of classification of humanity. The differences between populations with regard to such characters are obvious to the layman, but the anthropologist has reduced the results of examining them to mathematical form. It is, however, well known that, with one or two possible exceptions, such characters are not inherited according to any simple rule, and geneticists have shown that, in the case of certain of these characters, the quantitative expression of any one of them is controlled by a number of independent genes. Furthermore, most of the measurable features of human beings bear the marks not only of heredity but in a varying degree of environment, and especially of the state of nutrition during early life. Though the relationship is not a simple one, it is generally true that characters whose individual expression tends to be modified by the environment are also particularly subject to natural selection by the environment in the population as a whole. For instance,it is probable that the individual who is genetically small-boned will be at an advantage in a lime-poor environment, and so will tend to have more surviving offspring than a large-boned person.

In spite of these combinations and the apparently continuous grading of the characters concerned, morphological anthropologists have come to realize that the underlying factor in their classifications of human beings consists in the dis-

crete genes which are reproduced and so transmitted from generation to generation in a given population, and which, taken together, determine its innate physical composition.

Although obvious phenotypic features distinguish one population from another, it is also recognized that genotypic factors, such as blood group gene frequencies, differ from population to population. In the case of the blood factors we are dealing with unitary and obviously discontinously characters which are determined at conception and remain fixed for life that is to say,with the separate effects of individual genes which are sharply differentiated from one anther and which are therefore less subject to personal errors of observation than are many of the characters hitherto used.It appears likely that natural selection due to the external environment is slight for most of the blood factors(though certain combinations of blood groups are known to be intrinsically unstable).Moreover,the observed characters are a much more direct consequence of the genetic constitution than are the directly observable characters of the body.

It is evident that regional differences have biological significance and the functioning of the physical organism towards its environment is determined by the genes responses. Physical anthropologists are devoted to the understanding of these differences and their biological significance.

The study of population structure can show the interrelationship of biological and cultural variation with reference to a variety of models. Since the genetic structure of a population refers to the distribution and relationship of gene and genotype frequencies, it is ultimately a reflection of the population's mating structure(Yasuda and Morton, 1967).

In studies of population structure, the effect of barriers to gene flow,which create a subdivided population, are of major importance. These barriers may be geographic and/or cultural in nature. Man has complex rules about marriage and mating which can affect the amount of inbreeding in any population. In some small groups, the prohibition of mating with a close relative may force a man to seek a bride from another community, thus expanding the gene pool. Many thousands of years ago the movement of both large and small groups of people and their consequent admixture have made it difficult for any clear classification of human types to be made. In fact, much of today's research into human population variation seems to emphasise the need to examine the various factors contributing to human diversity.

The ABO blood groups were discovered at the beginning of the century( Landsteiner, 1900), their Mendelian inheritance indicated in 1911 by Von Dungern and Hirszfeld. Hirszfeld and Hirszfeld(1919),introduced the distribution of human blood groups as a new basis of classification. An enormous mass of information has accumulated since then on the distribution of ABO blood groups, the results of which were collected by Mourant et al(1954 and 1976) whose investigations suggested their variation in frequency differed from one population to another( Sunderland, 1973; Papiha,1979). Sunderland(1973), considers that,"...from an anthropological view point the most interesting matter is that the distribution of the A,B and O genes is extremely variable among the World's People." Distribution of other genetical characters such as MNS, Rh and taster reactions to phenylthiocarbamide and the later discovered blood groups are still being studied. These charact-

ers are susceptible to accurate statistical analysis. Their mode of inheritance is by Mendelian laws.

Apart from blood group systems, the new biochemical markers such as serum proteins, red cell isoenzymes and haemoglobins are of great importance in anthropological research. Their frequencies change slowly and the mode of heritance is relatively simple. Surveys from various parts of the world have shown that most of these markers are polymorphic,that is, most of them occur in two or more forms,maintaining a frequency of greater than one percent and less than ninety nine percent. Not all characters determined by single genes can be used for anthropological classification. To be of value, the gene frequency must vary significantly from one population to another, and the frequency of the second most common gene in a given system must be sufficiently high in some populations to enable a useful estimate of its value to be made by exami ning or testing only a few hundred perons. This eliminates nearly all congenital pathological conditions as being too rare, but a few inherited haematological abnormalities, such as thalassaemia, the sicklecell trait and glucose-6-phosphate dehydrogenase deficiency, are sufficiently common in some populations to render them taxonomically useful.

At the present time the study of genetic markers,such as the blood group systems can provide very useful information on the differentiation of human populations.

The aim of the present study is to add to the genetic data already existing, to attempt to appreciate the genetic structure of Iranian population as a whole and to compare it with those of the neighbouring groups.

# CHAPETER 1. INTRODUCTION TO IRAN

## 1.1. Geographical setting.

### 1.1.1. Physical features.

Iran means " Land of the Aryans", what the Greeks and then the west called Persia. It is situated between 44° and 63° longitude and 25 and 40 latitude. It has an area of 628,000 square miles (1,648,000 sq.Km) and is the fourth largest country in Asia.

The Iranian plateau is a triangle set between two depressions, the Caspian sea to the north and the Persian Gulf to the south. Further, between central and western Asia, it forms a link between the steppes of inner Asia and the plateau of Asia Minor and, beyond, Europe.

The country is bounded on the north by the Soviet Union and the Caspian sea, on the east by Afghanistan and Pakistan, on the south by the Persian Gulf and the sea of Oman, and on the west by Turkey and Iraq. Geography can thus account for the historic past which the plateau was called on to play in the course of thousands of years of history.

Iran is a land of high mountains, deep valleys, great flat - lands and vast arid deserts. The average altitude is 3,300 feet, while many peaks rise as high as 10,000 feet. Mount Damavand, the highest point in the Alborz range, rises to nearly 19,000 feet.

Two major ranges divide Iran in an east-west direction: the Alborz, beginning in the Caucasus, passes through northern Iran and continues into Afghanistan. The second range, the Zagros begins in Anatolia and continues right down to Baluchistan. Iran has two big deserts: the salt Desert ( Kavir-e-Namak) south east of Tehran and the Lut Desert ( Kavir-e-Lut )

further to the south-east. There are several major lakes in Iran, apart from the great in land sea, the Caspian, which lies to the north. The largest is Lake Rezaiyeh in west Azarbaijan, followed the Hamun in Sistan and the Bakhtegan in Fars. The heart of the country is the Tehran-Esfahan area.To the south is the important province of Fars, with its principal city of Shiraz. Further to the south-east is the province of Kerman. In the north-east is the province of Khorasan with its largest city,Mashhad. Further to the south and east is the barren land of Baluchistan.

1.1.2 Climate

Almost all of Iran lies in the north temperate Zone,but the plateau as a whole offers a variety of climates which,in turn, yield a great variety of agricultural products. Iran has great extremes of climate, the Caspian littoral is humid and sub-tropical while the central deserts are almost completely dry. Annual rainfall varies from an average of 40 inches in Gilan and Mazandaran to an average of less than two inches in the desert regions. Temperatures also vary greatly; in parts of the deserts and the areas of the Persian Gulf temperatures are rise to 130 degrees Fahrenheit,while in certain parts of the north-west temperatures of less than $18^{o}$ F. are not unusual in winter. Rain generally falls between October and May, the heaviest being recorded in December and March. The heihgt of the plateau and the presence of high mountains contribute to cold winters; frost occurs all over the country with the exception of southern Khuzistan and the coastal areas of the Persian Gulf and the sea of Oman. In central and north-eastern as well as in north-western regions, he-

avy snows fall from November to early March.

## 1.2.Historical review

Although Iran has more than her share of prehistoric mounds and sites, far too little archaeological work has been done to permit a reconstruction of her history before the advent of the Achaemenids. Excavations in one Cave at Behistun and in the caves called Belt and Hotu near Behshahr on the Caspian coast represent a splendid beginning. The finds indicate an active flint industry in the middle palaeolithic period as well as skeletons and Skulls of the Mesolithic period.This material has been dated to about 10,000 B.C. by the Carbon-14 method. About 6,000 B.C. some of the inhabitants of Iran discovered agriculture, practised animal husbandry, and made painted pottery and polished stone implements. The physical characteristics of these people are not known. However, the finds at Susa and Khuzistan showed Indo-European names in the cuneiform tablets of the Mittani Period in Mesopotamia and indicate the presence of these people in Iran. About 2,000 B. C., the Aryans came to Iran from the plains of the southern U.S.S.R. One stream of this great migration moved into present day India and the other stream came into Iran. Gradually these people replaced the original inhabitants. In the 19th Century B.C., nomadic tribes like the Medes, Persians (Parsa) and Parthians entered Iran from the north of the Caspian sea and by way of the Caucasus and mixed with the former inhabitants. The Medes settled in what is today north-west Iran; the Persians lived further south, roughly in the modern provinces of Fars and Khuzistan. The inflow of small communities of Greek origin after 331 B.C. also changed the ethnic map of Iran. Following the Greek period, the Sassanian dyna-

stry (226-641 A.D.) was established. The next major movements which resulted in the present diversity was the inflow of Arabs, Turks and Mongols. Arabs came to Iran in the 7th Century A.D. The transfer of tribes and the incursion of wandering Arab groups from Iraq had an effect on the racial mixing within the country. The Turkish invasion (1050 A.D.) changed the ethnic face of Iran. All of Anatolia, Azarbaijan and other areas were Turkified, the Turkish speaking tribes being scattered throughout the area. The north-east and north-west saw strong intrusions of Turkish and Tartar elements. The Mongols (1200 A.D.) ruled Iran for a century and strongly influenced the population. Eventually the Mongols were converted to Islam and this facilitated the mixing with earlier established Persians.

Persian history falls naturally into two distinct periods: the pre-Islamic and the Islamic, the dividing point being the overthrow of the Sassanians by the Arabs.in the third decade of the Seventh century A.D.

The Persians themselves always speak of their country as Iran, an ethnic name implying the habitat of the Iranians - a branch of the Indo-European family. The term Persia is derived from the name of the south-westerly province of Pars ( Modern Fars), where the first kings of united Iran, the Achaemenids, had their Capitals Pasargadae and Persepolis(Parsa).

As far as we know, the earliest Iranians to settle in what we now call Persia were the Medes who, migrating from the region of Transoxania or Turkistan,established themselves in the country lying between Rhages and Ekbatana (now Hamadan) in the north-west, probably in the eighth century B.C. Of the early Medes we know very little beyond what may be learnt from the

Greek historians and occasional allusions in Assyrian inscriptions for the Medes had frequent intercourse with their neighbours the Assyrians, from whom they acquired the art of writing, and much else besides.

Three general factors characterize Persian history: general continuity, periodic cataclysms, and the extraordinary influence of physical environment. For 2,500 years Persia had possessed an educated ruling class and a tradition of civilization which had survived the most barbarous invasions and the most alien conquests. From about 600 B.C. to the Moslem conquest there existed a native Persian civilization which nearly reached its perfection, modified by Hellenic influence after the conquest of Alexander and later, Islam. This civilization blossomed again in the Abbasid period, and was modified a third time by Chinese influences after the Turkish and Mongolian conquests. Persian civilization produced the safavid culture in the sixteenth and seventeenth centuries, the artistic productions of which are well known in Europe. In the twentieth century Persian life has again become subject to violent external influence with the introduction of European thought and mechanical techniques, and at present the old way of life and the new are still imperfectly blended. During her long history, Persia has always preserved her frontiers more or less unchanged, and she even emerged from the great war without loss of territory or status.

During certain periods, notably after Alexander the Great's conquest, and for more than two hundred years after the invasion of the country by the arms of Islam, Persia lost her position as an independent empire, and during the remaining periods of her history, though she has often formed merely one

of several states in the dominions of some world-conqueror, she has always retained her national individuality.

## 1.3. Ethnic groups

The Iranian population is among the most heterogeneous in the world. The region, now called Iran, has been invaded by people of many different races, and each has left its contribution to the gene pool of the local population. Kirk et al (1977) have reviewed the pertinent literature and indicate that the invaders include Assyrians, Greeks, Arabs, Turks, and Mongols. They point out also that many trade routes, including one from China, have crossed Iran, and that Africans were brought as slaves during the Moslem conquest. The invasions and the trade routes have provided ample opportunity for admixture.

Around the rim of Persia are group after group of people who are organized into pastoral tribes. Many of them live near settled folk who speak their language and often have a tradition of having formerly been tribesmen themselves. Such an ehtnic group is likely to be dominated by the tribesmen rather than the settled folk even through the latter are more numerous. The tribesmen are better organized for war and hence have more political influence.It is not surprising therefore that geneticists have made many studies of the people of Iran.Field (1939 and 1959) has reviewed extensively the studies in prehistory and physical anthropology. More recently there has been an increasing number of investigations into the distribution of blood genetic markers in Iranians. These offer a means of dissecting the varied genetic structure of the modern populations and relating these populations to others out-

side the country.

The inhabitants of Persia have neither racial nor linguistic unity. The principal elements are commonly called Persian but there are also Kurds, Lurs, Turks, Baluchis,Arabs, Georgians, Dravidian Brahuis, and Afghan Hazaras of Mongolian origin, whilst religious minorities include Armenians, Jews, Nestorian Christians or " Assyrians" and Zoroastrians.

Exact measurements have been made of only a few small groups of the population, but it seems that the bulk of the Persian population is derived from the Indo-European Aryan-speaking Iranian invaders of the second millennium B.C., and from their intermarriage with the previous inhabitants. The latter belonged to the 'Caucasian' branch which originally colonized the territories from the Caucasus to the Arabian Peninsula. There are several widely different constituents within the 'Persian' block. In the most recent anthropological work on Persia the term " Iranian" is reserved for a widespread physical type, on the border of the medium- and long -headed classifications, that occurs Persistently in Persian-speaking areas. But within the Persian block there are some large groups which may prove to be racially distinct, and possibly purely Caucasian, such as the Bakhtiari tribes of the Central Zagros, who are an extremely short-headed people..The Kurds, too, tend to be short-headed, and the exact affinities of the Persian-speaking stock of Khorasan, commonly called Tajiks, and of the Baluchis, are not yet determined. In general, racial mixture is greatest in those part of Persia which have formed the historical Zones of passage for invading armies and wandering hordes, namely Azarbaijan,

Khorasan, and the intermediate provinces, and also in the Zones of Sistan and Baluchistan which are open to the east. The traditional Persian policy of transferring troublesome tribal groups from one part of the country to another, and the persistent wandering of Arab tribes, have also complicated the racial map.

Thus Arab, Turkish, and Kurdish tribes appear in relatively homogeneous areas such as Fars and Mazandaran and add to the confusion of Khorasan and east Persian Baluchistan With these notable exceptions, there is a block of Iranian peoples speaking Persian dialects or allied languages throughout the Zagros regions and the populated basin west of the central deserts, and in the Alborz and Caspian provinces, while a similar element is considerable in the cultivated areas of Khorasan and continues through the Birjand highlands to Sistan; of specific elements Kurds live in the northern Zagros and north Khorasan, Lurs in the Central and Southern Zagros. The principal area of the Turkish element, descendants of the Seljuk and later invaders, is from Azerbaijan eastwards to Tehran and Saveh districts; there is also a large Turkish group in eastern Fars and northern Khorasan. Arabs are found in the plains of Khuzistan where they form the eastern fringe of the principal body of Arab peoples, in southern Khorasan, in south - eastern Fars, and along the southern Coasts, where they arrived by sea continuously in ancient and recent times. Armenians are most numerous as peasants in Azerbaijan, in the Burujerd and Faridan districts of the north-eastern Zagros, and as townsmen in many cities of north-western and northern Persia, particulary in the Julfa suburb of Esfahan. Jews are widely scattered in towns throughout Persia.

Georgians exist in much the same areas as the Armenians, but have generally been assimilated to Persians.

1.3.1.The Turks

The Turks of Persia have few racial affinities with the Ottoman Turks of Anatolia, and are generally grouped with the Uzbek and other Turkemans of the Adjoining Russian Republics as ' Iranian Turks '. They derive from central Asiatic Stocks, but probably contain by intermarriage a considerable Iranian element, especially in Azarbaijan.

1.3.2. Th Kurds

The Kurds are an *ethnic* group inhabiting adjacent mountainous regions in western Iran, northern Iraq and southeastern Turkey. There are also small numbers of Kurds in Syria, Jordan, the Soviet Union, Afghanistan, and Pakistan. There are today about 9,000,000 Kurds of whom over 3,000,000 are in Iran.

It is only about the beginning of the Christian era that the Kurds become recognizable as a distinct people. There is considerable disagreement as to their origin and privious history. Nikitin (1943) has summarized the views of numerous authorities and supports the theory of Minorsky (1945) that the Kurds originated as an amalgam of two related tribes, the Mardoi and the Kyrtioi, speaking related Median dialects. If we accept these views then the Kardoukhoi whom xenophon encountered in the country which is now Kurdistan were not the main or sole ancestors of the Kurds (but migrated to become the Kartvelians of the Caucasus). We may, however, regard the Kurds as descended at least in part from the Medes whose king Cyrus had his capital at Sanandaj, now the chief town of Iranian

Kurdistan. No adequate anthropometric survey of the Kurds exists, and we are dependent almost entirely on verbal descriptions from which it is clear that there are considerable variations of physical type from tribe to tribe. It is certain that at least some of the Kurds are blond and blue-eyed while others are much darker. In view of the growing relative importance of the blood groups and other inherited blood factors in characterizing populations and defining their inter relationships, it had for many years been realized that such a study of the Kurds would be of great importance. There was, however, a further reason for wishing to obtain such data. A numerous and important group of Jews, the Kurdish Jews, formerly lived in Kurdistan. Some still remain but many have migrated to Israel.

The Kurds follow the sunnite branch of Islam, an important feature which sets them apart from most of the remaining population of Iran. On the whole, however, linguistic affinity may have been a greater force than religious differences in the relation between the two people. The Iranians seem to have had less trouble with the Kurds than have their sunnite neighbours, the Turks and Iraqis. However, the Kurds have been a difficult element in Iranian history. In the seventeenth century Shah Abbas found it necessary to move a part of the tribe to Khorasan, partly to defend the area against Turkman raiders, but partly also to break up a dangerously concentrated force in the west. The Kurdish communities of today vary from highly nomadic tribal groups to settled town dwellers.

### 1.3.3. The Lurs

South of the Kurds in the Zagros chain, the Lurs still pursue a largely tribal existence. Together with the closely

associated Bakhtiari, they are said to number somewhat less than half a million. They live in Luristan. Their language is a special dialect of Persian and they are shiites. The origin of the Lurs is still unknown. Henry Field (1939) thinks that they are a part of the original Iranian stock that migrated from the regions to the east of the Caspian sea during the first half of the first millenium B.C.

1.3.4. The Arabs.

Several Arab tribes are found in the Zagros and many settled Arab villages and towns are found along the coast of Persian Gulf. The Arabs scattered through Persia are, like the Turks, no longer a racially pure element. In Khuzistan, where they are most homogenous, their affinities are with the Arabs of southern Iraq, themselves a mixed people with a preponderance of Caucasian elements. The Arabs of Khuzistan region are most probably the original population of the region. They are not mixed with other local population groups. However, in the course of history they must have become intermixed to a certain degree with negro elements, as can still be observed from the form of hair, nose, and skin colour.
The existance of a negrito type in southern Iran, said to be of 'Sumerian' or Dravidian origin and distinct from imported negro stocks, is still a matter of dispute. There are about two hundred thousand Arabs in Iran. The Iranian Arabs are Sunnite by religion.

1.3.5. The Bakhtiaris

All along the Zagros mountain-range from Azarbaijan to the Persian Gulf, pastoral tribes move with their herds back and forth every year over the spine of the mountains between summer ranges in the high valleys of the east and winter ran-

ges in the low foothills of the west. A segment of the Zagros about 150 miles long and centered due west of Esfahan is the home land of the Bakhtiari tribe. Some Bakhtiari tribesmen speak a dialect containing many words of Arab origin not found in other persian dialects; others speak a Turkish dialect differing little if any from that of the neighbouring Ghashghai tribesmen.

The Bakhtiari were until 1942 an autonomous political unit: they still have a common tradition of unity; and they reveal their feeling of belonging together in legends of a common origin. The Bakhtiaris are nominally shi'ah Moslems.

1.3.6.The Ghashghais

The Ghashghais occupy a region south of Shiraz. They are a group of Turkish origin with a language of their own, and a Mongol-like (very often a Bakhtiari-like) habitus. Their Mongoloid features are stronger than for instance those of the Ottoman Turks in the N.W. of Iran, who are perhaps more a product of intermixing with the original ancient population of Asia Minor. According to the tribal leaders, the Ghashghais were brought to the province of Fars by Shah Abass in the 17th century from what is now Russian Azarbaijan. Some Iranians state that they are of Seljuk Turkish origin; however, the Khans emphasize that although their language is a Turkish dialect, it was probably aquired in the not too distant past,and that their music and legendary tales are non-Turkish. Other Iranians believe that the Ghashghais originated from one of the Mongol tribes of Turkemanistan in Russia.

The Ghashghais are primarily a Turkish-speaking group,though a few Arab and perhaps even some Persian-speaking bands have allied themselves to the group. They are reported to have about

thirty-three thousand families in the area of Fars, though their numbers may have been much reduced lately by defection to other groups. Their annual migration from winter to summer pasturage is the longest among Iranian nomads, extending almost from the area south of Esfahan to the Gulf. They follow the shi'a branch of Islam.

### 1.3.7. The Mamassanis

The Mamassanis are a branch of the Lur tribe in southern Iran. According to the Khan, this tribe migrated from the Kurdistan region more than 800 years ago.

### 1.3.8. The Basseris

The Basseris are a tribe in the province of Fars near Shiraz. Their history is sparse with the exception of the belief that they have always occupied their present lands. They are variously described as Arab and Persian.

### 1.3.9. The Baluchis

The Baluchis of Iran are part of the larger group which forms the major part of the population of Baluchistan in west Pakistan. They speak an Aryan language akin to Persian and are quite conscious of bonds with Iran. However, they are sunnites by religion (though with several shiite modifications.) They may number as many as a million in Iran, in an area streching from near the Gulf of Oman to the province Sistan on the Afghan border.

### 1.3.10. The Brahuis

The Brahuis in Iran represent a very small fragment, under one hundred thousand, of a much larger group in Pakistan and Afghanistan. They live in the neighbourhood of Sistan and speak a Dravidian language, unrelated to any other language or dialect in Iran.

## 1.3.11. The Turkmans

Historically, Iran has been much troubled by the nomadic, raiding Turkmans, who have crossed the Russian border for many centuries, wreaking havoc on the Persian inhabitants of the area. With rather broad heads, Mongoloid eye folds, and straight hair, not always dark, they present a markedly different appearance from other Iranians. No accurate estimates of their numbers are availabe; their distribution is throughout most of northern Khorasan and down into the eastern part of Mazandaran.

## 1.3.12. The Assyrians

The Assyrians were originally an Arab-semitic agricultural people, who in ancient times occupied (several thousand years B.C.) the area between the Euphrates and Tigris rivers. They conquered extensive areas of the Middle East and became the elite class in this region, who did not intermix with the conquered peoples. In 175 A.D. approximately they were converted to Christianity. In 650 approximately their country was conquered by the Moslems, and the Assyrians fled, partly to the mountains where they became perhaps the ancetors of some of the Kurdish tribes, and partly to Persia where they maintained themselves as a separate group. Upto now this group has maintained its own language and script, quite different from Iranian and related to Arabic. The Assyrians are probably not a pure "race" as Christian Iranians are called " Assyrians" and taken up in the Assyrian Community. The christian Assyrians, live chiefly in the neighbourhood of Lake Urumieh, numbering about twenty thousand. Their language, of the semitic group, is distantly related to Arabic; their religion, of the

Nestorian branch of Christianity, provides a great Cohesive force.

### 1.3.13. The Armenians

Some Armenians, as mentioned, live in north western Iran, near the borders of their traditional native land. Others were settled in Julfa by Shah Abbas in 1604 across the river from Esfahan where they engaged their skills in the production of brocades and rugs in the royal factories. Still others live in Tehran and other major cities. Their total number is probably about sixty thousand. Their language is of the Indo-European group, but not at all closely related to Persian. They were not encouraged to intermarry with the Moslem group. Even today, those Armenians who have intermarried live outside the area of Julfa.

### 1.3.14. The Jews

Very little is known about the history of the Jews in Persia and about the social and cultural conditions of their life. It is definite, however, that a Jewish community was established there during the Babylonian exile( Levy, 1948 , Fishel, 1948). This community appears to have existed continuously since then. Its members sought to guard their Cultural individuality, carefully guarded themselves against all foreign influences and observed all of their religious laws, despite many vicissitudes during which many of them were killed or converted to Islam. Nevertheless, it is difficult to assume that the Persian Jewish community isolated itself so successfully that it can be considered anthropologically pure. The mother tongue of the Jews today is Persian but most of them jealously preseve their ethnic group integrity (including religion) through inbreeding and segregation.Jews in

Iran number perhaps seventy-five thousand with the greatest concentration in Tehran, Hamadan, Esfahan, and Tabriz.

1.3.15. The Zoroastrians

The Zoroastrians are the closest to the original Persians now in existence and today number only 15 to 20,000. Most Zoroastrians were born in Yazd and now live in Yazd, Kerman and Tehran. The Parsis of India, of similar origin, are descendants of Zoroastrians who fled to India at the time of the Islamic conquest. They are probably different ethnically from the Zoroastrians of Iran in that, according to local belief, those who left Iran were mainly soldiers.

Zoroastrians have a strict unwritten code:

They do not proselytize, outsiders are not accepted into the religion, and if a Zoroastrian marries into another group,neither he nor his children are considered Zoroastrian.

Zoroastrians believe that they were untouched by the Mongol invasions of the 13th-14th centuries. They most certainly were not the defending forces. If this is true, it suggests that the Zoroastrians, with their extensive Empire, were also reservoirs of the blood group, B gene. The importance of this group for future studies is incalculable. Unfortunately, if the present rate of loss of its members continues, they will probably be extinct within the next 50 years. The followers of the Zoroastrian religion in India form two distinctive groups, the Parsis and Iranis, numbering respectively just over 100,000 and approximately 6,000. The Parsis migrated to Gujarat at the end of the 7th century AD after the fall of the Sassanian Empire in Persia under the impact of the Moslem invasion. Since that time they have maintained a high degree of sociocultural isolation from other Indian communities, but

they have spread from Gujarat to other areas, chiefly in the north-west. Today approximately 70% of the Parsis in India live in Bombay having migrated there from the end of the 17th century. By contrast the Iranis are derived from the remnants of the Zoroastrian. Communities in the Yazd region of Iran who have migrated to India in the last 150 years and now live predominantly in Bombay, Poona and Dahanu regions.

1.3.16. Other ethnic groups.

The north-west corner of Iran is a potential trouble spot because of its proximity to Turkey and Russia. This largely mountainous area, Azarbaijan, before the nineteenth century included what is now the Soviet Republic of Azarbaijan in the USSR. The dominant people are called Azari or Azarbaijani. A small part of the area, near the Turkish border, is inhabited by Assyrians.

The Azari are a Turkish-speaking group, numbering about two million, indentical in language and certainly origin with the inhabitants of the Soviet Republic of Azarbaijan, immediately across the Russian border.

1.4.Demography and population density.

Iran is located in a region of complex population movement throughout the whole historical and protohistorical period. Some of this movement is documented, other parts we can only infer: the result has been to leave the population of Iran divided into many religious and ethnic groups of great interest not only to the Iranian people themselves but also to the researchers of human population structure and evolution.

We know little of the earliest inhabitants of what is now called Iran though there is evidence of human occupation from

palaeolithic times onwards, and increasingly abundant information is being discovered about occupation during the post-pleistocence or proto-neolithic era (Field,H.,1939,1959;Braidwood et al., 1961; sunderland,E., 1968; wilber, 1973).At some time during the second millennium B.C., Aryan invaders from the north or north-east entered the area and left their mark in myth and language and in the physical appearance of the peoples. Olmstead (1948) interprets the archaeological and traditional history of the Iranians as indicating that Aryans settled the southern shores of the Caspian sea in what was called Hyrcania ( later Mazandaran ) the capital of which was not far from Asterabad Bay at the south east corner of the Caspian. From the coastal area the Aryans crossed the barrier of the Alborz Mountains and spread out over the Iranian Plateau.

The Aryan invaders were succeeded by many others including Assyrians, Greeks, Arabs, Turks and Mongols. The present day populations are descended from the remnants of these successive waves of peoples who became assimilated with the original inhabitants. This process of assimilation seems to have progressed further in the more settled groups: it has had the least effect on nomadic groups (Arasteh, 1964). In addition to many invasions, northern Iran, particularly, has been on major trade routes and its people have been influenced, at least in part, by the repeated passage of caravans. With their assortment of exotic wares and of people bringing new ideas, and from time to time, new genes. of these trade routes, by far the most important was that running east to west passing close to modern Tehran and forming the end of the old "silk" road which connected China with Europe.

The total population of Iran is about 36 million.since Iran covers a vast territory, this means there is a fairly low numerical density. Movements of population in and out of the country (i.e. external migration)are not of great importance.But, on the other hand, the rural exodus, the drift from the country to the towns, presents a highly intractable problem; and the seasonal migrations of nomads are also considerable. It is the disposition of relief which could be held mainly to determine the geographical distribution of the population.

The centre of the country is largely empty and life has been driven either towards the exterior or towards the interior of the mountains, to the points where there is an adequate water-supply; people live among the foothills of all three mountain ranges, and each group has settled half in the plain and half on the mountain side. There are extraordinary variations in density. The most heavily populated cities are:Tehran, the shores of the Caspian sea, Tabriz,Shiraz, Esfahan, Mashhad, etc.

By way of simplification, the most heavily populated areas may be classified as follows:

the shores of the Caspian sea, the watered mountain regions, the naturally watered or irrigated oases of the plateau.

The elements which determine this distribution are the same that influence every dispersal of men throughout the world: natural factors and human factors(sociological, economic, and historical). The most important natural factors affecting the population distribution throughout Iran,as in a number of other developing countries, are water and temperature.

## 1.5. Consanguinity

Consanguinity and endogamy are among the characteristic features of marriage in Iran. Apart from considerations of convention such as the very frequent marriages between relatives,geographical, ethnic, religious, cultural, social and economic factors exert considerable influence.

There exists different rate of consanguineous matings in Iran as the results of variations in the above mentioned factors. The types of consanguineous marriages also differ among Iranians. First cousin matings occur more than the second cousin marriages among most population groups. Uncle-niece matings happen only among the Jews. This type of mating is forbidden in Islamic and Christian rules.

The incidence of consanguineous marriages ranges from 24.46 (Esfahanis) to 31.59 (Tehranis) percent in Urban; 30.59 (Azaris) to 65 ( Bandaris ) percent in rural; 18 (Turkmans) to 73.46 ( Ghashghais) percent in tribal; and 2.81(Christians) to 25.39( Jews) percent in religious groups of Iran (Farhud et al, in preparation).

On the whole the observed frequencies of the consanguineous matings in Iran appear to be higher than that of 30 percent in the Middle Eastern populations ( Frazer Roberts and Pembrey, 1978).

## 1.6. Religion

In religion, however, Persia has played an important part, a part not wholly laid aside, and an outline of her religious history is here appropriate. Zoroastrianism was the ancient faith of Iran and is important because of its similarity to Judaism and Christianity. A phase of this religion known as Mithraism penetrated into the Roman world during the early Christian ages and spread so rapidly in many parts of Europe that altars were set up and cave-temples built to celebrate the mysteries of the Persian divinity Mithra and to glorify this personification of light, the sun, and truth. Furthermore, the system of Manichaeism, which sprang up on Persian soil, was powerful enough to compete for a time with Neo-platonism and Christianity for the religious and intellectual supremacy of the Roman Empire. Islam is the religion of Persia to day, but Persia belongs to the Shiite sect of the faith and acknowledges Ali, Mohammed's first cousin and son-in-law, as the prophet's successor in opposition to the Sunnite branch of Islam. Iran is in fact the chief representative of Shiism and has been largely instrumental in the growth of this factional movement which divides the Islam world.

Except for one or two hundred thousand Christians, Jews, and Zoroastrians, all the inhabitants of Persia are Moslems, and Shia Islam of the" Jafari"form is the official religion of the state.

Persia is peculiar in that the dominant form of Islam is

not the Sunnism which is supposed to be "orthodox" in other Moslem countries. Most of the Kurdish, Turkomans, Baluchis and the Arabs people inhabitants in the Kurdistan, Gonbad, Baluchistan and Khuzistan, are Moslem Sunnis, More than two million of the 36 million Persians being Sunni Moslems.

Since the Islamic conquest in the seventh century A.D., vast areas of Iran have been overrun by Arabs, Turks, Mongols and Afghans. These peoples arrived as Moslems or were afterwards converted to Islam. Large numbers of the invaders remained and their descendants today form the tribes in Iran ; however, many of their progeny are now mixed with the Moslem population.

## 1.7. Language

Persian or Farsi is the official national and literary language of Iran, spoken by three quarters of the population. Arabic, a number of Turkish dialects, and a few other languages are spoken in Iran, in addition to Persian, which is ,of course, the most important language. It is also, as the official language of the country, known one way or another by most of the other inhabitants. Persian is a member of the Indo-European family of languages and belongs to the Indo-Aryan(Indo Iranian) branch of this family. It is spoken in various dialect forms by a majority of the population. It is an aryan language, derived from Middle and old Persian, but has been modified by Arabic in general vocabulary and in the constructions of written prose. The present tendency is towards the expulsion of Arabic words and forms in favour of Persian terms. In sound, Persian is sonorous and has been called the most musical of all languages. It is written in Arabic characters, but in an elementary form it is capable of Romanization.

Inhabitants of various cities can be identified by minor variations in pronunciation and choice of words. Most Persians believe that the "purest" Persian is spoken in Shiraz. These regional differences do not seriously affect mutual understanding.

Several provincial dialects are spoken,especailly in Gilan and Mazandaran, and there are several small communities speaking patois of their own. The speech of Gilani( Gilaki) and Mazandarani is far enough from standard Persian as to be almost incomprehensible to a Persian.

Two other languages, that of the Kurds on the west and the Baluchis on the east, are related languages of the Indo-European group, Kurdish is distinct from Persian, being directly connected neither to old nor to middle Persian. Of the three principal forms of the Kurdish language two are spoken in Persia, Kurdi and Gorani, but these two have numerous dialects.

Baluchi is another aryan language akin to old Persian and often modified by modern Persian. Luri, the language spoken by most Faili Lurs of Luristan proper, by the Bakhtiari,Mamassani tribes is closer to old Persian than is Kurdish. Brahuis of the eastern border speak a Dravidian language,unrelated to any other language of Iran.

The Arabic dialects spoken by the Arab tribes of Khuzistan, along the coast of the Gulf and of Mokran, and,with varying mixture of Persian or Turki elements, by Arab groups of the interior such as the Khamseh tribes of Fars, are modern variants of the same older Arabic that formed the base of the classic literary language and all the dialects from Morocco to India. Arabic is a semitic language,related to Hebrew,

Syriac, and Ethiopic. There is thus no clear relationship between Arabic and Persian, but conquest and the legacy of the Islamic religion and culture have resulted in an enormous infiltration of Arabic words into Persian. Arabic words actually incorporated into Persian have been modified to fit the Persian sound pattern.

The Turkish languages, unrelated to Arabic or Persian , belong to the Uralic-Altaic family, which includes languages spoken as far west as the European shores of the Bosphorus and east to Mongolia. Turkish dialects are spoken in Iran: Azari, the Turkish language spoken by the Turks of Azarbaijan, north west of Iran, is grammatically close to the Ottoman Turkish of Anatolia. Various tribes, including the Ghashghai and Khamseh, in the south, speak Turkish dialects. The nomadic Turkmans of Khorasan in the north east, also speak a form of Turki, which is much closer to east Turki.

These languages are not written to any extent in Iran . In Azarbaijan, however, the language is written as well as spoken, and though the educated classes understand Persian, they prefer to be addressed in their own language. Many Turkish words have been incorporated into Persian, and vice versa.

Of minor nationalities, the Armenians retain their native language. Armenian is an Indo-European language, but is entirely incomprehensible to a Persian. Almost all Armenians in Iran speak Persian in addition to their native tongue.
The Assyrians around Lake Urumieh speak Aramaic dealects related to the older East Syriac, an ancient language of the Semitic group, intermingled with Persian, Turki and Arabic words. With their religion, this is a great binding force, which

unites them with the Assyrians of Iraq, and makes them a foreign minority in both countries.

Jewsspeak Persian and also a Persianized Hebrew.

# CHAPTER 2.

# AN ACCOUNT OF POLYMORPHIC SYSTEMS EMPLOYED IN THE PRESENT INVESTIGATION

During the last fifty years, and especially over the last twenty years, the discovery of new techniques has led to the detection of many genetically determined polymorphisms in man, and the frequency of the genes and phenotypes can be used in classifying and comparing populations. Genetic traits now commonly investigated in population surveys include the major blood group antigens and the serum protein and red cell enzyme polymorphisms. Search for genetic variation in human populations is important because genetic marker systems constitute a prerequiste for several kinds of genetic studies, particularly for the attempts to map the human genome.If genetic variants governed by one locus shall be efficient as a maker system, more than one of the alleles at the locus in question must be relatively frequent in the population under study. Recent research has demonstrated that approximately 10 percent of randomly selected gene loci controlling biochemical traits in man show polymorphic variation and that nearly all gene loci reveal the presence of rare mutant forms with frequencies of 1 per 1000 or less (Harris, 1970; Harris and Hopkinson, 1972). The ability to detect such genetic variants provides a powerful tool for tracing population affinities.

The study of serum protein and red cell enzyme polymorphisms by electrophoretic techniques has become a valuable tool for the modern physical anthropologists. It contributes to the description of genetic stocks, helps to trace migrations and exchanges, and may confirms the genetic isolation of some

groups. As more refined techniques have developed, a large number of new alleles have been demostrated in many serum protein and red cell enzyme systems. This permits a more precise definition of a population's genetic composition. When many populations have been studied, maps can be drawn showing the distribution of the various alleles. If these maps show that clines of gene frequencies cross linguistic, cultural, and racial barriers but are correlated with geographical factors, one can suppose an environmental (selective) effect which may be as important as migration and genetic exchanges(Bodmer and Cavalli-Sforza, 1976).

Little or nothing is known regarding the adaptive value of erythrocyte enzyme polymorphisms, except for the recognized role of G-6-PD. But if the allelic frequencies of most of these systems are represented geographically, it is observed that their distribution within mankind cannot be considered merely as random phenomenon. Furthermore, it is known that, in general terms, for each polymorphic enzyme of differing phenotype there exists a different enzymatic activity, as a result of which it is reasonable to suppose that, in the presence of certain biotic and/or abiotic factors, an adaptive role attaches to these alleles. Since the first step in ascertaining whether this is true is obviously to gather the largest possible volume of data regarding genetically and/or geographically heterogeneous populations; a number of surveys are now being conducted to that end, especially of populations still heavily exposed to the natural environment.

## 2.1. Blood group antigens.

Since Landsteiner's work (1900-1901) on the ABO groups, other separate blood groups systems have been discovered, in

all of which inheritance has been shown to conform to Mendelian principles. The ABO blood groups of man have been intensively studied and the distributions well-mapped because of the dramatic ill-effects arising from incompatibel transfusion. Remarkably, the incidence of haemolytic disease of the newborn arising from ABO incompatibility is low, but some specialists are doubtful of the sensivity of the standard techniques used for its detection. The Rhesus group in all its complexity has received much attention in connection with haemolytic disease of the newborn, whilst other systems such as Duffy, Kell and Kidd are known to incite haemolytic antibody formations but the specific conditions for this to occur are unknown. All these serological systems and others besides, are variable in human populations.

## 2.1.1. The ABO blood group system

Three of the ABO blood groups were discovered in 1900 by Landsteiner. He described the agglutination which occurred when red cells of one individual were exposed to the action of serum from another (Landsteiner, 1900, 1901). He found that in some cases agglutination occurred, whereas in others there was no reaction.On the basis of these agglutination reactions Landsteiner was able to divide human beings into three distinct groups.Indication of a fourth group ( AB) were found in 1902 by Decastello and Sturil but they tested only the serum which they described as ' without group '. We have been unable to ascertain who first described the reactions of AB red cells (Mourant, et al. 1976), but the situation which emerged within a few years was that human

beings are divisible into four classes according to the reactions which their red cells show with normal human sera. The classification of four groups- A, B, AB and O, is based on the presence upon the surface of the red cells, of either, both, or neither of two substances known as A and B substances, the active parts of their molecules being of a polysaccharide nature. In serological terms the A and B substances are described as antigens or agglutinogens.

The red cells of an individual possess either one or both, or neither of the antigens A and B. A cells carry only A substances, B cells only B; AB cells carry both and O cells neither. All or very nearly all the red cells of an individual are qualitatively indentical in blood group constitution and reactions..

Human sera may contain either,both, or neither of the corresponding antibodies or haemagglutinins, anti-A and anti-B. Red cells containing antigen A are agglutinated by anti-A, cells containing antigen B by anti-B. Both anti-A and anti-B agglutinate AB cells, while neither of them react with O cells. The anit-A in a serum containing it can attach itself to the A substance on A or AB cells, and it then alters their properties in such a way that they stick to each other, or agglutinate. Anti-B behaves similarly with B or AB cells.

No blood contains one of these antibodies which will cause its own red cells to agglutinate, but subject to this restriction all possible antibodies are present in all bloods after the first few months of extra-uterine life. Thus all normal human sera contain those antibodies which do not react with the individual's own red cells, The serum of an A person contains anti B, that of a B person, anti-A. The serum of an O

person contains both antibodies, and that of an AB person, niether of them. The relations between genotypes,phenotypes, and antibodies are summarized in Table 2.1.1.a., which forms the basis of AB testing, known sera containing anti-A and anti-B, and know A and B cells, being used as reagents to test unknown bloods.

TABLE 2.1.1.a.

THE ABO BLOOD GROUPS ANTIGENS AND ANTIBODIES

| Blood groups (Phenotype) | Blood groups substances on red cells | Antibodies present in plasma(or serum). | Genotype |
|---|---|---|---|
| O | none | anti-A,anti-B | OO |
| A | A | anti-B | AO or AA |
| B | B | anti-A | BO or BB |
| AB | A and B | none | AB |

The inheritance of blood group characters, based on Mendelian principles, was reported by Dungern and Hirszfeld(1911). But the genetics of the ABO system were first correctly worked out by Bernstein(1924) who showed that the four groups were inherited by means of a set of three allelomorphic genes A,B and O(for the frequencies of which the symbols p, q and r are commonly used). Of these, A and B each gives rise to a characteristic antigenic (and ultimately biochemical) structure, the A or the B antigen, while the O gene behaves as an amorph, not giving rise to any antigen peculiar to itself. These antigens are usually

sought on the surface of the red cells, but are in fact found widely distributed elsewhere in the body.

Thus, the A gene, when present at all, on one or both of the chromosomes concerned, determines the presence of the A substance on the red cells. The B gene similarly determined the presence of B antigen. The 0 gene give rise to neither antigen, and in the heterozygote with an A or B gene it has little influence on the expression of that gene in producing the A or B antigen, but homozygous 00 bloods carry neither antigen. As the 0 gene is really an amorph, therefore group A and B behave as dominant traits. Thus group A blood may be of genotype AA or AO, B blood BB or BO, but group 0 blood can only be of genotype 00 and AB blood of genotype AB. Thus, the four blood groups represent six genotypes as shown in Table. 2.1.1.a. Because of the amorph status of the 0 gene, the genotypes AA and AO are indistinguishable by any routine tests on the cells of the individual, as are the genotypes BB and BO.

The anti-A and anti-B antibodies in human serum are still often described as 'naturally occuring', but it is now generally accepted that they are the result of immunization in infancy by substances closely related to the A and B antigens, and derived from the environment through the respiratory or alimentary tracts. It may appear at first sight surprising that these antibodies, the formation of which is stimulated by a variety of environmental antigens, should apparently be so precisely tailored to the blood group antigens of other human beings. It must, however, be remembered that the normal individual is tolerant of, (or does not make antibodies to), antigens forming part of his own body,( or

at any rate parts which are accessible to plasma antibodies). Thus, for example, any antibody produced in response to an A-like antigen derived from the environment will be so constituted as to react as well as possible with A-like antigens but subject to the restriction of not reacting with the individual's own antigens, (which may include substances which are more or less A-like, present in all individuals, whether or not they possess the gene-determined and biochemically sharply defined blood group A antigen). Such an antibody may also react with non-human A-like substances, such as red cell antigens of other mammals, but this will not show in tests restricted to human red cells and human plasma or serum. Similar considerations apply to B and anti-B.

The sub-groups of A and B

Considerable numbers of variants of the A antigen are known, most of which are rare; the B antigen is less variable but several rare variants have been described.

At the variant level, the most important distinction is between $A_1$, the commonest antigen, and $A_2$ which has a frequency of several percent in most European, African, and West Asiatic populations. Though the distinction has been known for about 50 years, its basic nature is still not completely understood, but most of the facts are covered by the following conventional explanation. Thomsen et al(1930) observed that there were two varieties of the A antigen, $A_1$ and $A_2$, allowing the blood groups A and AB to be classified respectively as $A_1$ and $A_2$, and as $A_1B$ and $A_2B$. Both types of antigen react with the ordinary antibody anti-A, but only $A_1$ reacts with anti-$A_1$, while $A_2$ fails to do so. Thus, red cells classified as $A_1$ are agglutinated by anti-A and anti-$A_1$, whereas cells classified

as $A_2$ are agglutinated by anti-A and do not show any reaction with anti-$A_1$. Reaction of antigens is shown,as usual, by agglutination. Anti-$A_1$ is present in the serum of most B persons together with ordinary anti-A.

The $A_1$ and $A_2$ antigens are produced by corresponding allelomorphic genes, so that what we have called the A gene is really of two possible kinds, $A_1$ and $A_2$. $A_1$ is dominant to $A_2$ and 0; $A_2$ is dominant to 0; neither $A_1$ nor $A_2$ is dominant to B. In the genotype $A_1A_2$, the $A_1$ gene causes the production of $A_1$ antigen, and thus the genotypes $A_1A_2$, $A_10$ and $A_1A_1$ are indistinguishable, by methods at present available, since all react both with anti-A and anti-$A_1$.

Thus, the recognition of four alleles -$A_1$, $A_2$, B and 0 - leads to the expectation of ten possible genotypes and six distinguishable phenotypes.

The complete genotypic system of the $A_1A_2B0$ groups is shown in Table 2.1.1.b.

TABLE 2.1.1.b.

THE $A_1A_2BO$ GENOTYPES AND GROUPS

| Genotypes | Groups |
|---|---|
| OO | O |
| $A_2O$ | $A_2$ |
| $A_2A_2$ | |
| $A_1O$ | $A_1$ |
| $A_1A_2$ | |
| $A_1A_1$ | |
| BO | B |
| BB | |
| $A_2B$ | $A_2B$ |
| $A_1B$ | $A_1B$ |

A considerable number of other variants of blood group A are known; they are mostly very rare but at least two of them are not uncommon in Africans, and possibly in other non-European populations.

A type intermediate between groups $A_1$ and $A_2$ ($A_{1,2}$ or $A_{int}$), reacting positively but weakly with anti-$A_1$, was described by Landsteiner and Levine(1929) and first shown by Wiener et al

(1945) to found especially in Negroes. In addition, many sub-groups of A have been described which are negative with anti-$A_1$ and weaker than $A_2$ in their reactions with anti-A. There has been some confusion as to their identity and nomenclature. One such group has been shown to be present in South African Negroes (Bantu). Its distinctiveness has now been established by Brain (1966) who has called it $A_{bantu}$. Though he is able to define four fairly distinct types, $A_1$, $A_{int}$, $A_2$, and $A_{bantu}$, among the Bantu, the sharp distinction between the A subgroups, found among Europeans, certainly breaks down in the Bantu; a similar finding has been made by Vos and Comley(1967) in West Australian aborigines. As group $A_2$ is intermediate in several of its properties between $A_1$ and O, it would be of great interest to know whether this applies to its associations with disease, but unfortunately very few investigations of associations have determined the sub-groups of A in their patients.

2.1.2. The Rhesus blood group system

The discovery of the Rhesus or Rh blood group system by Landsteiner and Wiener in 1940 was the most important discovery in the blood group field since the ABO groups. They showed that the antibodies, produced by injecting red cells of the monkey Macacus rhesus into rabbits and guinea-pigs( produced by immunizing them) not only agglutinated the red cells of the monkey but also about 85 percent of the people of European descent. They classified these 85 percent whose red cell were agglutinated by rabbit anti-rhesus serum as Rh-positive and those showing no agglutination as Rh-negative. The antibody was shown to be indistinguishable from that reported in Levine and Stetson's case of 1939.Finally, it was shown by Levine et al in 1941 that the destruction of the red cells

in the new-born was the result of Rh blood group incompatibility between a Rh-negative mother and a Rh-positive child.

Intensive work on this system led to the view that the Rh groups were not as simple as they seemed at first. Soon after the discovery of the original anti-Rh (the anti-rhesus monkey-guinea pig serum actually defines a different antigen to D as found in man ) other reactions were noted and antibodies clearly connected with the Rh system, but having different specificities from the original one, were discovered, and it became necessary to recognize subtypes of the Rh groups.

The nomenclature used to communicate the various findings of the Rh system was based on two classic hypotheses concerning the genetics of the system, one of which was suggested by Fisher and Race (cf. Race and Sanger, 1962), the other by Wiener (cf. Wiener and Wexler, 1963).

The Wiener hypothesis suggests that the Rh antigens are produced by a series of multiple alleles of one gene. He postulates that a gene gives rise to an agglutinogen and this in turn possesses a number of blood factors, whereas the Fisher-Race hypothesis makes no such distinction between genes and antigens. The relationship between the Wiener and Fisher-Race notation is set out in Table 2.1.2. a .

TABLE 2.1.2.a

COMPARISON OF THE FISHER-RACE LINKED GENE THEORY AND THE WIENER MULTIPLE ALLELE THEORY

| Gene Complex | Symbol | Anti-gens | Gene | Agglu-tinogen | Blood Factors |
|---|---|---|---|---|---|
| CDE | $R_z$ | C,D,E | $R^z$ | $Rh_z$ | rh',$Rh_o$,rh" |
| CDe | $R_1$ | C,D,e | R' | $Rh_1$ | rh',$Rh_o$,hr" |
| cDE | $R_2$ | c,D,E | $R^2$ | $Rh_2$ | hr',$Rh_o$,rh" |
| cde | r | c,d,e | r | rh | hr',hr",hr |
| cDe | $R_o$ | c,D,e | $R^o$ | $Rh_o$ | hr',$Rh_o$hr",hr |
| cdE | R" | c,d,E | R" | rh" | hr',hr" |
| Cde | R' | C,d,e | r' | rh' | rh',hr" |
| CdE | $R_y$ | C,d,E | $r_y$ | $rh^y$ | rh',rh" |

The system of antigenic and genetic notation most commonly used is the CDE system based on the work of Fisher and Race (1944); but Wiener and others describe it in terms of Rh and R with indices and suffices. As Race and Sanger (1968) point out, disagreement is almost entirely on interpretation and not on the observed facts. They also say, with reference to the CDE notation, ' we think that this is at present the only notation in which a detailed account can be communicated'. The Rh groups are thus regarded as determined by a set of alleles at three very closely adjacent loci on each of a pair of chromosomes, which may be called the C, D, and E loci.

The principal antigen of the system is know as D, determined by a gene D, the allele d of which behaves as an amorph. Very closely linked to the Dd locus are two other loci each

characterized by a pair of major alleles, Cc and Ee respectively. Each of these four genes gives rise to a correspondingly named antigen.

As only one of each pair can be carried on each chromosome, there are thus eight possible chromosomic combinations of genes, all of which are known to exist, but of which CDe, cDE, cDe and cde are the commonest. The eight alternative Rhesus gene combinations are shown in Table 2.1.2.b.

TABLE 2.1.2.b.

ALTERNATIVE RHESUS CHROMOSOMES

| | | | |
|---|---|---|---|
| | D | E | CDE |
| | | e | CDe |
| C | | E | CdE |
| | d | e | Cde |
| | D | E | cDE |
| | | e | cDe |
| c | | E | cdE |
| | d | e | cde |

When these letters were assigned by Fisher there was no evidence as to the serial order of the loci, but there is now strong evidence that it is DCE.

At each locus two common alleles may be distinguished as well as a considerable number of rarer ones. In general, each allelic gene gives rise to a distinct antigen, corres-

ponding to a distinct antibody which causes agglutination of the red cells containing that antigen. Some of the rarer alleles, however, differ only quantitatively in their effects from one or other of the common alleles.

The D locus can be occupied by either of two common allelic genes D and d. Of these, D gives rise to the D antigen which is the strongest antigen of the system, both in its immunizing potential and also usually in agglutination for which it is responsible. The d gene, on the other hand is, like O in the ABO system, an amorph, giving rise to no specific antigen. There are, in addition, a number of other genes which are known collectively as $D^u$( Stratton, 1946). The $D^u$ antigen produced by the genotypes $D^u d$ and $D^u D^u$, gives rise to agglutination by some but not all anti-D sera, and those agglutinations which do occur tend to be weak. Wiener and his colleagues (1959, 1962) have, however, described a series of specific weak variants of the D antigen which they call $Rh^A$, $Rh^B$, $Rh^C$ and $Rh^D$, of which the first three at least may be regarded as forming part of the $D^u$ series, but precisely definable by reactions with appropriate antisera. They give rise to the possibility of immunization of persons carrying one component of the D cc complex, by another component of the same complex, a phenomenon which has been reviewed by Tippett and Sanger (1962).

The Gonzales ($Go^a$) antigen of Alter et al (1962) is present in about 2 percent of American Negroes. It is recognized by a positive agglutination test with a specific human anti-$Go^a$ serum. The antigen was originally thought to belong to a completely new blood group system, but Lewis et al (1967) have shown that it is part of the Rh system, and is apparently produced by a variant of the D gene in the cDe complex. All $Go^a$-

positives are strongly positive with most anti-D sera, but a number of $Go^a$-positives have produced an antibody which agglutinates all D-positives except those whose only D gene is of the $Go^a$-variety. Tippett has shown that the antigen fits into the class of D variants described above and, according to Race and Sanger( 1968 ), 'The antigen maybe pictured as replacing part of the usual D structure previously thought merely to be missing'.

At the C locus there may exist either of two common alleles, C and c, each giving rise to a specific antigen. Rarer specific alleles are $C^w$ and $C^x$ and one quantitative one like $D^u$, known as $C^w$. A further variant, more nearly resembling c, has been described by Huestis et al (1964).

At the E locus the common alleles are E and e. The rare antigen $E^w$ ( Race and Sanger, 1968) is recongnized by a specific anti-$E^w$ antibody, and depends on a gene $E^w$.

However, with the exception of $D^u$ and $c^w$, all additional alleles are unusual or rare in Caucasian populations.

Although the Rh system is one of the most complex genetic systems investigated in man, it has been of great value in describing the gene pools of the various human populations. There are wide racial differences in the frequency of Rh gene complexes. Detailed information regarding racial differences has been reported by Mourant et al (1954, 1976).

From a clinical point of view the Rhesus or Rh system is by far the most important of the blood group systems other than ABO. Because of its greater clinical importance it has been the subject of much more extensive and detailed research, and also of intense controversy, mainly centring upon its genetics.

Rh incompatibility is the main cuase of haemolytic disease of the newborn, and a major cause of transfusion reactions. Incompatibility with respect to the D antigen is the main cuase of haemolytic disease of the newborn, but Cc and E**e** incompatibilities are rare causes, as they are also of transfusion reactions. Unlike the antibodies of the ABO system, each of which is universally present in persons lacking the corresponding antigen, antibodies of the Rh antigens are virtually never found except as a result of immunization by pregnancy or transfusion, and reactions appear only at a second exposure to the antigen. This applies also to all the other blood group systems except the ABO. Very few searches have been made for any other kinds of diseases associations of the Rh groups, and nearly all of these are confined to a comparison of the D-positive and D-negative types.

The Rh genetical complexes

Fisher's theory of the genetics of the Rh blood groups postulates the existence of closely linked sets of three genes, one from each of the sets of alleles mentioned above.The number of such complexes described in the literature is very large.

While the CDE formulae are essential for understanding both the genetics and the antigens, it is convenient to have a 'shorthand' notation for at least the commoner complexes, and for this purpose symbols based on those of Wiener are most convenient. In the Table 2.1.2.c the CDE genetical formulae of these selected complexes are set out, together with the 'shorthand' notation which is based on, but not indentical with, that of Wiener.

TABLE 2.1.2.c.

Rh GENE COMPLEXES FOUND IN POPULATION STUDIES, SHOWING THE CDE ( FISHER ) NOTATION AND THE USUAL ABBREVIATED NOTATION, BASED ON THAT OF WIENER.

| Fisher's notation | Abbreviated notation |
|---|---|
| CDE | RZ |
| CDe | $R_1$ |
| CdE | $r^y$ |
| Cde | r' |
| cDE | $R_2$ |
| cDe | $R_o$ |
| cdE | r" |
| cde | r |
| $C^w DE$ | $R_Z^w$ |
| $C^w De$ | $R^w_1$ |
| $C^w de$ | $r'^w$ |
| $CD^u e$ | $R_1^u$ |
| $cD^u E$ | $R_2^u$ |
| $cD^u e$ | $R_o^u$ |

Most of the antigenic manifestations of the Rh genes can be expressed in terms of separate antigens determined by allelic genes at three distinct though very closely linked loci. There are however, a number of antigens which cannot be attributed to any one of these three hypothetical loci. Nearly ev-

ery such antigen can, however, be explained as a joint product of genes at two adjacent loci on one chromosome.

2.1.3. The MNSs blood group system

There was a long interval between the discovery of the ABO blood groups and that of the next system, the MN, which was first described by Landsteiner and Levine(1927).This system depends upon a pair of allelomorphic genes which behave in nearly all respects as though they determined the antigens M and N respectively.

Antibodies against M and N are not usually found in humans, but are prepared by the injecting of human blood into rabbits. The serum of the rabbit injected with M blood agglutinates both M and MN erythrocytes, and the serum treated with N blood agglutinates N and MN erythrocytes. Thus, the two antigens,M and N were detectable on human red cells by means of immune rabbit sera, one or both of which were present on all cells.

The inheritance of these antigens is based on the two allele theory advanced by Landsteiner and Levine in 1928. According to this theory there are two alleles, M and N, either of which determines the presence of corresponding antigenes on red cells. Thus there are three genotypes MM, MN, and NN and three corresponding phenotypes M, MN and N.

It has, however, recently been shown that the N antigen is a precursor substance, and that the so-called N gene is an amorph which leaves the N antigen unchanged, while the M gene in the heterozygote converts part of the N antigen into M, and in the homozygote converts nearly but not quite the whole of it.

In 1947 Walsh and Montgomery discovered the existence of a new antigen, S, which Race and Sanger found to be related to the MN system (Race and Sanger, 1949).

The S antigen was shown to be serologically different from M and N ( Race and Sanger, 1947). Antigen S was shown not to be an allele of M and N but that it was related to MN as the alleles of Rh system are related. Using the analogy of Fisher's theory of the genetics of the Rh system, they showed that the S antigen was the product of one of a pair of allelic genes S and s, very closely linked to M and N.

Family studies suggested that persons who possess S are homozygous or heterozygous for one allele and persons who do not have S are homozygous for another allele. In 1951 the discovery of the expected antithetical antibody anti-s which agglutinated the red cells of homozygotes as well as heterozygotes was reported by Levine et al (1951), indicating that there are two antigens, S and s and three blood types, S, Ss and s. These findings confirmed the hypothesis that S and s form another pair of genes closely linked with M and N. The relationship of the M and N gene locus to that for S and s is considered to be very close, and it becomes difficult to postulate whether these are two gene loci close together on the same chromosome or whether complex alleles at a single gene locus are concerned in producing both MN and Ss substances. Table 2.1.3. demonstrates the genetical interpretation of serological reactions of the complete MNSs system.

TABLE 2.1.3.

GENETICAL INTERPRETATION OF THE REACTIONS OF ANTI-M, ANTI-N, ANTI-S AND ANTI-s SERA

( After Race and Sanger, 1950).

| Anti M N S s | All 4 sera: genotype | First 3 sera: genotype or phenotype |
|---|---|---|
| + - + - | MS/MS | MMS |
| + - + + | MS/Ms | |
| + - - + | Ms/Ms | Ms/Ms |
| + + + - | MS/NS | |
| + + + + | MS/Ns | MN.S |
| | Ms/NS | |
| + + - + | Ms/Ns | Ms/Ns |
| - + + - | NS/NS | |
| | | NN.S |
| - + + + | NS/Ns | |
| - + - + | Ns/Ns | Ns/Ns |

Race and Sanger (1970) suggested that the linkage between the loci must be very close since crossing over has been shown to occur only very occasionally.

On a given chromosome any one of the four combinations MS, Ms, NS, and Ns could exist, but S was more usually linked with M than with N. To explain this uneven association it was necessary to postulate very close linkage.

In addition to the common genes M and N, a number of rare alleles are known at the same locus. The only one which is of anthropological interest is $M^g$, discovered by Allen et al

(1958). The antigen $M^g$ reacts very weakly with anti-N and not at all with anti-M, but a specific anti-$M^g$ is present in one percent or more of normal human sera.

$M^c$ is an antigen intermediate in properties between M and N, reacting with some anti-M and some anti-N sera.The gene responsible is an allele of M and N( Dunsford et al, 1953).

The $M^k$ gene appears to be an allele at the combined MNSs locus, and gives rise to none of the antigens M,N,S and s.It does, however, gives rise to a specific antigen to which a very weakly reacting antibody can be prepared in rabbits(Metaxas and Metaxas, 1964).

Two kinds of antiserum are known which define subtypes of M.Anti-$M_1$, corresponding to anti-$A_1$ of the ABO system, was found by Jack et al(1960), to be present in some human anti-M sera, but not in rabbit anti-M. The $M_1$ antigen is commoner in Negroes than in Europeans. If the antisera were more readily available, the subtyping of M would probably be of considerable anthropological interest. The $M^v$ antigen is defined by a single human serum which reacts with all N and MN bloods and with about 1 in 400 M samples from Europeans. The latter are regarded as containing an M Variant, mostly of course in the heterozygous form. (Gershowitz and Fired, 1966).

A third allele, which determines the absence of both S and s, and is know as $S^u$, is not uncommon in Negroids.

The system is one of considerable complexity, involving numerous variants of M and N, and a variety of antigens determined by other closely linked genes. While the system is thus of considerable genetical and anthropological interest, it seldom gives rise to haemolytic disease of the newborn, or of transfusion reactions, and shows few disease associations.

Antigens due to genes linked with MNSs

A considerable number of antigens are known, each individually very rare, which in family studies have proved to be due to genes closely linked to the loci for MN and Ss. Because of their individual rarity they seldom occur together, and hence little is known as to possible allelomorphism among the genes concerned.

The Hunter ($H^u$) antigen was found as early as 1934 by Landsteiner, Strutton and Chase in 7 percent of American Negroes but only 0.5 percent of whites, all of whom were N or MN. Chalmers et al (1953) obtained evidence that the antigen was the product of a gene closely linked to Ns. It is possible that the anti-Hunter serum defines a sub-group of N rather than distinct antigen.

The Henshaw (He) antigen of Ikin and Mourant (1951) is found mainly in Africans. The He gene is almost invariably closely linked with NS in West Africans but with MS in South Africans. It is also sometimes linked with Ns or with Ms. The antigen does not appear to have been found, in tests controlled by comparison with known He-positives, in any populations not known on other grounds to be of African ancestry (Shapiro, 1956).

The Miltenberger complex of antigens ($Mi^a$,VW, Mur) is certainly due to a number of genes closely linked with MNSs, and may plausibly though not certainly be regarded as due to a set of alleles at a single locus closely adjacent to those for MN and Ss ( Levine et al, 1951).

Of the other antigens known to have close linkage relations with MNSs, several are actually or potentially of anthropological interest. They are as follows: the $st^a$(stones) anti-

gen, the $m^a$( Martin ) antigen, the Vr (Verdegaal ) and $N^a$ ( Nyberg) antigens, the Tm antigen, the sj antigen, the $Ri^a$ ( Ridley ) antigen.

2.1.4. The p blood group system

The p blood groups were discovered by Landsteiner and Levine (1927) in the course of the same investigations that led to the discovery of the MN groups. It was at first thought that only one antigen, p, was involved, determined by a gene p, the allele p being an amorph. Thus, bloods were classified as p-positive and p-negative, the positives possessing p gene. Thus, p-positives were either pp or Pp, and p- negatives pp. The antibody agglutinated approximately 75% of the population and these were termed.p+. Owing to the existence of bloods which reacted weakly to the early anti-p sera, the frequency of the two groups could not be established with certainty. Racial differences however, were recognized ( Landsteiner and Levine, 1927, 1929) and the p antigen was shown to be inherited as a mendelian dominant character ( Landsteiner and Levine, 1931).

Since then the $Tj^a$ antigen has been discovered by Levine et al (1951), coupled with the recognition by Sanger (1955 ) that the antigen is part of the p system. It became clear that p-persons shared a powerful antigen ($Tj^a$) with p+ people, and a third and extremely rare group is defined in which this antigen is lacking. This discovery led to a modification of the original notation of the p system. Race and Sanger (1954)gave a table of results of testing with anti-$p_1$ on the blood of 'Caucasians'. The frequency of negatives, now $p_2$, varied from 18-30 percent. Such differences Race and Sanger (1970) stated reflect serological and not anthropological differences.

Further investigation has disclosed a system of considerable complexity and has shown that p as originally defined is not in fact an amorph but a weak variant of the original p. By analogy with the ABO system the two genes are now called $p_1$ and $p_2$, and a third extremely rare amorph gene, the new p,has been recognized, the homozygous form of which fails to react with a very rare but extremely potent antibody, anti-p,formerly known as anti-$Tj^a$(anti-Tay) and then thought to belong to an independent system.

The antigen $Tj^a$ found by Levine et al (1951) was at first regarded as the product of a gene present in nearly all human beings, the extremely rare allele being an amorph, with the homozygotes usually having a strong anti-$Tj^a$ antibody. Sanger (1955) then showed that $Tj^a$ was part of the p system, with three alleles, $p_1$ (formerly p ), $P_2$( formerly p), and the new p (formerly regarded as the amorph allele of $Tj^a$). These relationships are similar to those existing between $A_1$, $A_2$ and 0 of the ABO system. $P_2$ bloods sometimes show anti-$p_1$ in the plasma, usually with a very low titre, but the rare bloods always have a high titre of anti-p+ anti-$p_1$. The p system has now taken the form shown in Table 2.1.4.

TABLE 2.1.4.

THE P BLOOD GROUP SYSTEM

| Phenotype under old system | modern phenotype | Genotype | Reaction with anti-sera | |
|---|---|---|---|---|
| | | | anti-P+ anti-$P_1$ (formerly anti-$Tj^a$) | anti-$P_1$ (formerly anti-P ) |
| P+ | $P_1$ | $P_1P_1$ | + | + |
| | | $P_1P_2$ | | |
| | | $P_1P$ | | |
| P- | $P_2$ | $P_2P_2$ | + | - |
| | | $P_2P$ | | |
| | P | PP | - | - |

The gene P, and hence all the genotypes containing it, are exceedingly rare.

The only recognizable genotype, PP, has never been found in any random population survey, and Race and Sanger(1968) estimate its frequency at about one per million. Population data are thus concerned only with the genes $P_1$ and $P_2$. The strength of the antigen $P_1$ in $P_1$- positives varies quite considerably. Fisher(1953)has shown that much of the variation can be explained as due to the difference between $P_1P_1$ and $P_1P_2$.By no means the whole of the observed variation can be explained in this way,but it is still uncertain whether,and if so how far, the remaining variation is due to a series of quantitative variants of the $P_1$ gene. Many papers by Japanese workers describe the incidence and other properties of blood group antigen Q. This has been shown by Henningsen ( 1954, 1955 ) to

be indentical with that here called $P_1$.

Two very rare antigens, $p^k$ and Luke, are related to the p system; as Race and Sanger (1968) show, $p^k$ and probably also Luke, behave as though controlled by a series of allelic genes separate from those of the $P_1P_2P$ series, but interacting with them in a similar manner to the interaction of the Hh genes with those of the ABO series. $p^k$ individuals (perhaps analogous in the above sense with those of Bombay type) seem to be almost confined to the Finnish people, but even among them have never been found in a random series of tests. The Luke system or subsystem remains rather obscure; the Luke antiserum fails to react with p(and also with $p^k$) people,but is negative also with about 2 percent of $p_1$and $p_2$ people.

The $p_1$ antigen is present in hydatidcyst fluid (Cameron and Staveley, 1957) and in a considerable variety of worms, both parasitic and free living. It is likely that the anti-$P_1$, not infrequently found in the plasma of $p_2$ individuals and in that of several species of mammals, is a response to worm infestation, but nowork appears to have been done on possible associations between the p groups and such infestation.

Women of genotype pp, who always have anti-p+ anti-$P_1$ in their plasma, are particularly subject to abortion, apparently resulting from the action of the antibody upon the almost invariably p-positive foetus.

Paroxymal cold haemoglobinuria is due to the presence in the patient's plasma of a cold-reacting auto-antibody. It was shown by Levine et al (1963) that this usually has anti-p specificity.

## 2.1.5 The Kell blood group system

The kell blood group system which initially, like all other systems, appeared simple, has gradually been shown to have a comparable complexity, and a similar organisation to the MNSs and Rh systems. The Kell antigen was first described by Coombs, Mourant, and Race in 1946. It was soon shown that it was the product of a gene K. The antigen K was found in only 9 percent of an English population sample, who were termed Kell-positive. The discovery of the expected antithetical antibody anti-K (Cellano) by Levine et al (1949) made it clear that the system was governed by a pair of allelomorphic genes, K and k and the inheritance of the Kell groups by means of two allelic genes without dominance was proved. The two genes K and k control the production of the corresponding antigens K and k. The groups of the system as defined by anti-K and anti - k are shown in Table 2.1.5.a.

TABLE 2.1.5.a.

PHENOTYPES AND GENOTYPES OF THE KELL SYSTEM

| Reaction with antisera anti-K | anti-k | Phenotype | Genotype |
|---|---|---|---|
| + | - | K | KK |
| + | + | Kk | Kk |
| - | + | k | kk |

This simple view of the Kell system lasted until 1957 when Allen and Lewis (1957, 1958) described two further antigens $Kp^a$ and $Kp^b$, associated with the Kell system and shown to be the products of a pair of allelic genes.

The sutter system of Giblett (1958) at first appeared to be an independent one. Again two antithetical antigens $Js^a$ (found by Giblett, 1958) and $Js^b$ (found by Walker et al,1963) are known, the products of a pair of allelic genes, first shown by Stroup et al 1965 to belong to the Kell system.

Finally, the Karhula or $Ul^a$ antigen of Furuhjelm et al (1968, 1969) has been shown to have a similar association.

By analogy with the MNSs and Rh systems, the Kell system may be regarded as determined by three or possibly four closely linked loci, occupied by the respective allelic genes K,k; $Kp^a$, $Kp^b$; and $Js^a$, $Js^b$. The details of the relationship with $Ul^a$ are not yet published. If we disregard $Ul^a$, about which little is yet known, only four of the eight theoretically expected gene complexes have been found, as shown in Table 2.1. 5. b.

TABLE 2.1.5.b.

GENE COMPLEXES OF THE KELL SYSTEM

| |
|---|
| $KK^b\ Js^b$ |
| $kKp^aJs^b$ |
| $kKp^bJs^a$ |
| $kKp^bJs^b$ |

Some very rare individuals are known whose red cells are negative with all six diagnostic antisera. They may be homozygous for a rare amorph gene complex, but it is possible that some of them are, as suggested by Race and Sanger (1968) , homozygous for a rare allele of a common gene which controls the synthesis of a basic substance from which the observed

antigens are elaborated.

The great majority of human beings in all parts of the world are homozygous $kKp^b$ $Js^b/kKp^b$ $Js^b$, the genes K, $Kp^a$, and $Js^a$ being everywhere relatively rare and none of them having yet been found occurring together in one gene complex. Thus most of the facts about the distribution of the complexes can be covered by separate consideration of the distributions of the genes K, $Kp^a$ and $Js^a$:

2.1.6. The Duffy blood group system

The discovery of the Duffy blood group system was first briefly reported by Cutbush, Mollison and Parkin (1950) and more fully reported by Cutbush and Mollison (1950). The antibody, anti-$Fy^a$, was discovered in the serum of an individual suffering from haemophilia and who had received a number of blood transfusions over a period of twenty years, and developed an immune antibody in response to transfusion. It soon became clear that the antigen was the product of a gene,giving rise to the recognizable antigen, called $Fy^a$ which must possess an allele $Fy^b$ though no product of the latter gene could at the time be detected. The first example of the antithetical antibody, anti-$Fy^b$, which did detect this product, was reported in 1951 by Ikin et al. They also found that the antigens were inherited by two allelic genes, $Fy^a$ and $Fy^b$, without dominance.

The allelic genes $Fy^a$ and $Fy^b$ account for nearly all the phenotypes found in European populations, but a third allele in the Duffy system was postulated when Sanger et al (1955 ) discovered that the red cells of most American Negroes did not react with either antiserum and that they belonged to a new phenotype which was like Fy(a-b-). They thought that they

were homozygous for a third gene, Fy. This allele is exceedingly rare in whites but Race and Sanger, from family data, consider that Fy may have a frequency as high as 3 percent among Europeans. Chown et al (1962, 1965) have reached a similar conclusion; they however, find that the third gene among Europeans is not a complete amorph but gives rise to weak $Fy^b$ reactions.

Table 2.1.6. demonstrates the genetical interpretation of the Duffy system.

TABLE 2.1.6.

PHENOTYPES AND GENOTYPES OF THE DUFFY SYSTEM

| Reaction with: anti-$Fy^a$ | anti-$Fy^b$ | Genotype | Phenotype |
|---|---|---|---|
| + | - | $Fy^aFy^a$ | Fy(a+b-) |
| + | + | $Fy^aFy^b$ | Fy(a+b+) |
| - | + | $Fy^bFy^b$ $Fy^bFy$ | Fy(a-b+) |
| - | - | Fy Fy | Fy(a-b-) |

An inspection of the latest tables of Mourant et al (1976) will show that, except in the South African Republic, over 95 percent of African Negroes are of this type, that is homozygotes of a third allelic gene, at first regarded as an amorph and so named Fy. It is now known that two distinct genes have in the past been confused under this term. One, $Fy^x$, with a frequency of approximately 1.6 percent in Euro-

peans, gives a product which is negative with anti-$Fy^a$ but which reacts feebly with anti-$Fy^b$( Chown et al, 1972). The other, almost universal in Africans, gives a product which completely fails to react with either of these antibodies, but is not a true amorph, for Behzad et al (1973) have shown its product to react specially with an antibody which they called anti-$Fy^4$; the gene should presumably be called $Fy^4$. Miller et al (1975) have shown that the homozygote of this type is probably specifically resistant to Vivax malaria, to which Africans have long been known to be resistant. It is known that Africans are mostly of the Duffy blood type Fy(a-b-), the homozygote of which was formerly regarded as the amorph gene Fy but is now recognized positively as $Fy^4$. They are also mostly highly resistant to the malarial parasite plasmodium vivax ( the formerly common European type.). Miller et al have now shown that the point of attachment and entry into the human red cell is the antigen $Fy^a$ or $Fy^b$, but that they will not enter cells of type Fy(a-b-). One may speculate that Africans developed resistance to p. vivax by natural selection of the 'amorph' Fy gene, biologically an efficient and 'cheap' process. When, however, p. vivax had thus almost disappeared from tropical Africa, the much more virulent p. falciparum was evolved, to which at first there was no inborn resistance. Thus when the haemoglobin S gene appeared, possibly introduced from outside Africa, or evolving in some limited focus, the fact that heterozygotes were resistance to p. falciparum led to the spread of the new haemoglobin by natural selection. This, however, was a much more 'costly' form of resistance, since it led to the early deaths of nearly all homozygotes.

## 2.1.7. The Kidd blood group system

The Kidd blood group system was discovered by Allen et al in 1951. The finding of this 'new' antibody, anti-$JK^a$, was the result of a haemolytic disease in a newborn infant. The antigen $JK^a$, was shown by these authors,in collaboration with Race and Sanger (1951) to be the product of a gene $JK^a$. The existence of the expected antibody, anti-$JK^b$, reacting with the product of its allele, $JK^b$, was described by Plaut et al in 1953. It has been suggested that the two genes, $JK^a$ and $JK^b$, are inherited as non-dominant autosomal alleles.At present, tests have been made with both antisera. A new phenotype like JK(a-b-) was reported by Pinkerton et al (1959) in Filipinos with some Spanish and Chinese ancestry. Such individuals were presumed to be homozygous for a third allele JK. The genetic background of the JK(a-b-) phenotype is not yet clear.

The phenotypes and genotypes of the system as defined by using anti-$JK^a$ antisera is shown in Table 2.1.7.a

TABLE 2.1.7.a.

PHENOTYPES AND GENOTYPES OF THE KIDD SYSTEM

| Phenotypes | Genotypes |
|---|---|
| JK(a+) | $JK^aJK^a$ |
| | $JK^aJK^b$ |
| JK(a-) | $JK^bJK^b$ |

The phenotypes and genotypes of the system as defined by both anti-$JK^a$ and anti-$JK^b$ antisera is presented in Table 2.1.7.b.

TABLE 2.1.7.b

PHENOTYPES AND GENOTYPES OF THE KIDD SYSTEM.

| Reactions with: | | Phenotypes | Genotypes |
|---|---|---|---|
| Anti-$JK^a$ | Anti-$JK^b$ | | |
| + | - | JK( a+b-) | $JK^a$ $JK^a$ |
| + | + | JK( a+b+) | $JK^a$ $JK^b$ |
| - | + | JK( a-b+) | $JK^b$ $JK^b$ |

Race and Sanger(1968), however, think that many sera used as anti-$JK^a$ reagents contain an additional unrecognized antibody, probably belonging to the JK system, since tests carried out in England have rather consistently shown an excess of apparent heterozygotes, above the frequency required by genetic equilibrium. Studies on Canadians by Chown et al (1965), however, show internal consistency, suggesting that a pure anti-$JK^a$ serum was used. The question of the purity of anti-$JK^a$ sera is at the moment mainly of theoretical interest in population studies, since such sera, pure or otherwise,are in extremely short supply. It could, however, have considerable practical importance if adequate supplies of anti-$JK^b$ as well as of anti-$JK^a$ should become available and show the JK (a-b-) phenotype, mentioned below, to be at all widespread. Even the antiserum anti-$JK^a$ however is rather rare, so that its use has to be somewhat restricted. It is moreover a difficult serum to use requiring either an enzyme technique or a complement-fixing anti-human-globulin test.The knowledge of the distribution of the genes of the JK system is based mainly

on tests with anti-$JK^a$ alone.

## 2.1.8 The Lutheran blood group system

The Lutheran blood group system was discovered in 1945 by Callender and Race who found the antibody now known as anti-$Lu^a$ in the serum of a patient suffering from Lupus erythematosus diffusus. The antibody was shown to be immune in nature. The system was more completely reported by Callender and Race (1946). Family studies indicated that the antigen $Lu^a$ was inherited as a Mendelian dominant character and the blood group system depends upon a pair of allelic genes, $Lu^a$ and $Lu^b$. The gene $Lu^b$ was only recognized as the absence of $Lu^a$ until 1956. The notation suggested for the system was as follows:

genes : $Lu^a$, $Lu^b$

phenotypes : Lu(a+), LU(a- )

antibodies : anti-$Lu^a$, anti-$Lu^b$ ( to be discovered).

Positive evidence of the existence of an $Lu^b$gene was first found by Cutbush and Chanarin (1956) when they indentified an expected antibody anti-$Lu^b$ which reacts with the product of this gene. The finding of anti-$Lu^b$ established the existence of the antigen and gene $Lu^b$. With the use of the two antisera three phenotypes can be identified each corresponding to one of the genotypes $Lu^aLu^a$, $Lu^aLu^b$ and $Lu^bLu^b$.

Table 2.1.8 indicates the correspondence of genotypes and phenotypes of the Lutheran system.

TABLE 2.1.8.

PHENOTYPES AND GENOTYPES OF THE LUTHERAN SYSTEM

| Reactions with: anti-$Lu^a$ | anti-$Lu^b$ | Phenotype | Genotype |
|---|---|---|---|
| + | - | Lu(a+b-) | $Lu^a$ $Lu^a$ |
| + | + | Lu(a+b+) | $Lu^a$ $Lu^b$ |
| - | + | Lu(a-b+) | $Lu^b$ $Lu^b$ |

In 1961, Crawford et al showed the system to be more complicated with the detection of a new phenotype that reacted like Lu(a-b-) to the known antibodies. This fourth phenotype fails to react with either of the anti-sera.

Most examples of this phenotype show dominant inheritance possibly due to a gene at a separate locus, but no such example has ever been found in a random population survey. Others show recessive inheritance and may be due to an amorph allele at the Lutheran locus; only three examples have been found in population surveys one by Darnborough et al (1963) in testing 18069 British blood donors, and two by Candanayingyong et al ( 1967 ) among 455 Thai donors.

The Lutheran system is notable genetically as having provided the first known example of ordinary autosomal linkage in man, between the loci for Lutheran blood group and for salivary secretion of the antigens of the ABO system (Mohr,1951). As, however, there is a recombination frequency of about 15 percent the linkage does not affect the independence of the two systems in population studies.

Both the diagnostic sera required for this system are in short supply, anti-$Lu^b$ extremely so. Thus only a very few population samples have been tested with both antisera, and only moderate numbers with anti-$Lu^a$ alone.

It has been shown that the $Sw^a$ ( Swann antigen ) gene is part of, or closely linked to, the Lutheran system. (Metaxas -Buhler et al, 1972).

## 2.1.9. The Diego blood group system

The Diego blood group system was first reported by Layrisse et al in 1955. The antibody which reacted with the antigen had been produced by a Venezuelan woman, Mrs. Diego, through pregnancy immunization, and had caused haemolytic disease of the newborn. The authors recognized that the family involved was probably partly of American Indian descent, and they showed the antigen to be present in various Indian and mixed populations, the phenotype frequency rising as high as 35 percent in Carib Indians. The antigen is the product of a gene $Di^a$ with dominant (or co-dominant ) expression.
The expected antithetical antibody anti-$Di^b$, detecting the product of the allelic gene $Di^b$ was found by Thompson et al (1967). It has been suggested that the system is controlled by two allelic genes, $Di^a$ and $Di^b$. No example has yet been found of a phenotype Di(a-b-) nor is there any other genetical evidence of the existence of a third allele.

Information about the distribution of the $Di^a$ gene has been studied extensively by numerous investigators, but especially by Layrisse and his colleagues (1960). According to them the Diego antigen appears to be confined to ethnic groups of mongoloid origin. Geographically, it is distributed widely, having been found in South American Indians, Mexican

Indians, Japanese, Chinese and in mixtures of Mongoloids with other ethnic groups. It is extremely rare in Caucasians,North American and African Negroes. Thus, the gene is essentially characteristic of Mongoloids and has never been found in any survey of unselected persons of presumed unmixed European descent.

Of the Diego blood groups more than of those of any other system it can be said that their main scientific interest lies in the field of anthropology.

2.1.10 The ABH secretion system

The antigens A,B and H of the ABO blood group system, as well as occurring in the erythrocytes, are also found in water soluble form in body fluids, including saliva (Yamakami, 1926; Lehrs, 1930, Putkonen, 1930). However, some individuals do and others do not secrete into their saliva antigens corresponding to their ABO blood group. Schiff and Sasaki ( 1932 ) found that the ability to secrete the ABH group-specific substances is inherited as an autosomal dominant trait. Groups A, B and AB persons who are secretors secrete the antigens corresponding to their blood groups. Group O persons who are secretors secrete the H substance, as do all other secretors to a somewhat less extent. The non-secretor individuals do not secrete their ABH substances, no antigenic substances being found in their body fluids. Secretion is controlled by a pair of allelic genes, Se and se, giving three possible genotypes SeSe, Sese and sese, the first two constituting the secretors, the third category the non-secretors.

2.2. Serum proteins

All immediate gene products are now known to be proteins, but the direct detection and examination of the products of

specific genes is a very recent development, beginning with the discovery by Pauling et al in 1949 of a physio-chemical difference between two genetically distinct haemoglobins.

From the standpoint of the methods used in identifying them,as well as from that of their innate functions the proteins of the blood are divided into two main classes by the surface membrane of the red cell. Outside are the plasma proteins. Inside are the red-cell proteins, of which by far the most abundant is haemoglobin. Of the others, present in smaller amounts, the enzymes are of the greatest interest to the geneticist, because they can be visualized by highly specific staining reactions, and because many of them show two or more readily distinguishable gene-controlled variants. Numerous enzymes, many of them genetically polymorphic are,however, also present in the plasma.

The object of a series of population surveys is not,except in rare instances, to determine in any absolute sense the properties and constitutions of the products of a series of allelic genes. This has in general already been done, or is being done, by workers in other disciplines. The object is rather to distinguish clearly in an empirical manner between known gene products, while always keeping an eye open for an unknown one. The geneticist must not,however, lose sight of the functions of the proteins with which he is dealing, from the standpoint of the body's total metabolism. In many cases the genetical variants of a single protein can be shown to differ quantitatively in their functional chemical activity, and such differences must be taken into account in studying interactions between genetical constitution and the environment.

However, the blood plasma contains in solution a great

variety of proteins, many of which show genetical polymorphism. Unlike the blood groups, these proteins mostly have known functions, for instance as enzymes, or as carriers of simple substances like metals and vitamins. As mentioned above the genetical variants of a particular protein often, and perhaps nearly always, differ quantitatively in their functional activity, thus allowing the possibility of natural selection, which may be the cause of the observed variability of gene frequencies between populations. Only in a few cases have the frequencies of the variants of a particular protein been ascertained in patients suffering from specified diseases ( Mourant et al, 1976, 1979).

2.2.1. The haptoglobin (Hp) system

Haptoglobin has long been known ( Polonovski and Jale, 1940) as a glycoprotein ( a protein with attached carbohydrate ) present in the plasma, and having the well defined function of combining with any dissolved haemoglobin entering the plasma as a result of the lysis of red cells. This prevents the haemoglobin from being excreted by the kidney, but the share of the haptoglobins in conserving the body's supply of iron is not fully understood. The literature of the biochemistry and genetics of haptoglobin is extensive and complicated. A full and excellent summary is given by Giblett (1969).

The first report of the existence of the plasma protein now known as 'haptoglobin' was made by polonovski and Jale (1938). Smithies (1955) and Smithies and Walker (1956) demonstrated that genetical variation occurred in the $\alpha_2$-globulin, haptoglobin, when sera were subjected to starch-gel

electrophoresis. The complex electrophoretic patterns could be attributed to three common phenotypes, known as Hp1-1, Hp2-1, and Hp2-2, which behaved genetically as though the proteins concerned were the products of two allelomorphic genes, $Hp^1$ and $Hp^2$, at an autosomal locus (Smithies et al 1962).Phenotypes 1-1 and 2-2 are the expression of the homozygous forms and phenotype 2-1 of the heterozygote of the $Hp^1$ and $Hp^2$genes. Extensive family studies (Smithies and Walker, 1955;Gala - tius-Jensen, 1958; Harris et al, 1959; Smithies et al, 1962) confirmed this hypothesis.

The $Hp^1$gene has a single product which yields a fast moving band, intensely stained and somewhat slower than free haemoglobin. The $Hp^2$gene gives rise to a series of polymers with different mobilities. It has no band in the position of the $Hp^1$ component, but slower bands of diminishing intensity towards the origin. These bands vary in width and extent to which they stain. The heterozygote of these two genes, HP2-1, shows a band in the position of the $Hp^1$band, but it stains less trongly, and also has multiple bands in the $Hp^2$ position which have a faster mobility than $Hp^2$ bands, due to the fact that they are polymers of the products of both the $Hp^1$ and $Hp^2$ genes ( Figure 2.2.1.a and b).

The haptoglobin molecule, like those of so many other proteins, consists of two types of polypeptide chains, $\alpha$ and $\beta$ ,presumably the products of two distinct sets of allelomorphic genes. Black and Dixon(1968) who have studied the structure in great detail point out resemblances with the immunoglobulins and suggest that the two types of protein have evolved from a common origin.

Variants of the $\beta$ chains are exceedingly rare; almost all

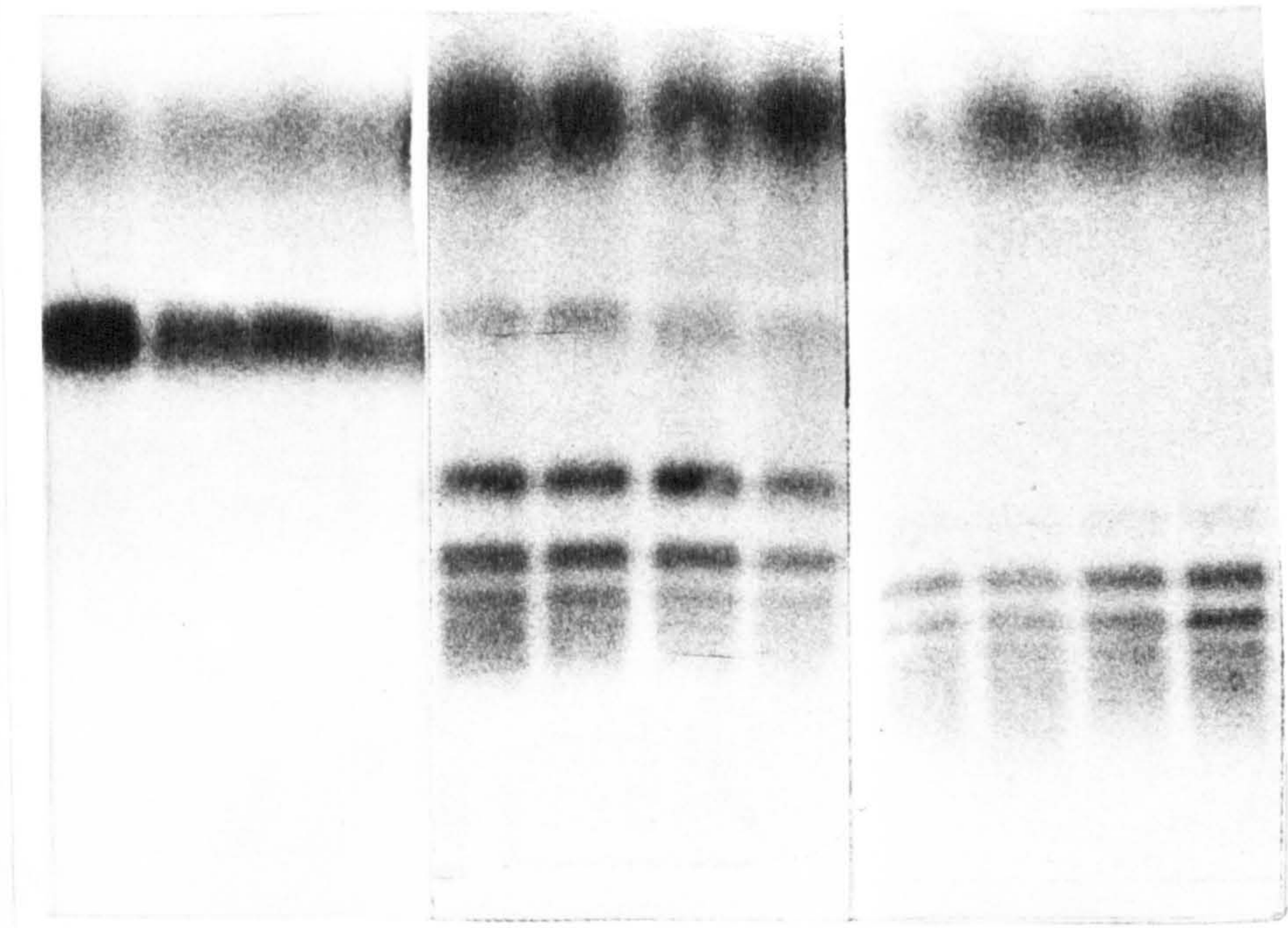


Fig.2.2.1.a. Starch gel electrophoretic patterns of the three common haptoglobin phenotypes (From left to right)HP 1, 2-1 and 2.

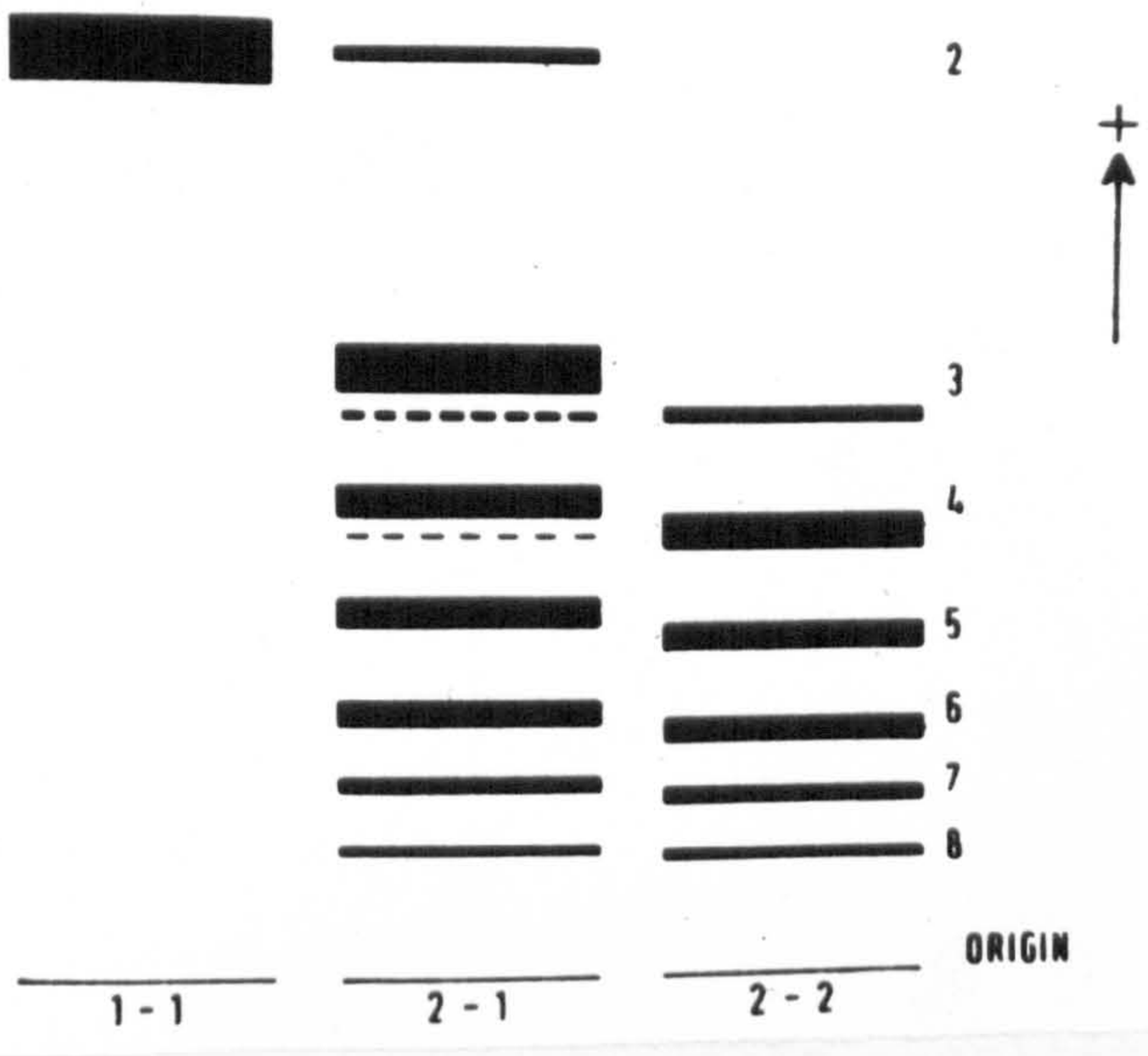



Fig.2.2.1. b. Diagram of the three common haptoglobin phenotypes, HP 1, 2-1 and 2.

the known variation is in the ∝ chains, the main polymorphism being determined by genes at the ∝ locus.

The β -chains are found to be the same in all the three haptoglobin types, whereas structural variations in the ∝ -chains show different electrophoretic patterns of the intact molecules. Thus, a series of phenotypes in the haptoglobin system can now be recognized.

When the haptoglobin molecule is cleaved into its constituent chains by means of 8 M urea and mercaptoethanol, the chains determining the apparently single Hp1 phenotype are shown to be of two alternative types, differing slightly in their speed of migration during electrophoresis, and by a single amino-acid substitution in their polypeptide chains, corresponding to alleles $Hp^{1F}$ and $Hp^{1S}$.Thus, individuals with Hp1-1 phenotype can have a fast migrating ∝ -polypeptide ($Hp_{1F-1F}$), a slow migrating ∝ -polypeptide($Hp_{1S-1S}$) or a mixture of both fast and slow migrating ∝ -polypeptide ($Hp_{1F-1S}$). Phenotypes Hp2-2 persons exhibit only one band which is considerably slower than Hp1F or Hp1S. Individuals with Hp2-1 phenotype have Hp2 band combined either with Hp1F or Hp1S. These subtypes are controlled by two alleles, $Hp^{1F}$ and $Hp^{1S}$. The single type of ∝ chain derived from the $Hp^{2}$ molecule is of nearly twice the molecular weight of that of Hp1 of either kind; it is in fact a combination of the greater part of the ∝ chains of the two types of Hp1, and appears to arise from a partial gene duplication resulting from unequal crossing over between $Hp^{1F}$ and $Hp^{1S}$.The retention of the original amino-acid sequences suggests that this crossing over was a comparatively recent event in the history of mankind, while the wide distribution of the $Hp^{2}$gene suggest that it

must in some circumstances have had a considerable selective advantage over both the $Hp^1$ genes.The single band of Hpl and the fastest band of Hp2 each correspond to molecules containing two $\alpha$ and two $\beta$ chains.

The molecular weights of all Hp2 molecules present in the plasma are thus greater than any of those of Hpl. Hp2-2 presumably for this reason,tends to bind less haemoglobin,weight for weight, than Hpl-1,but the former,because of the larger molecular size of the complexes,is more fully resistant to excretion by the Kidney: this is presumably the basis of the success of the mutation.Some more nearly direct evidence of the presumably balanced advantage enjoyed by the $Hp^1$ molecule has recently come to light,since Ritter and Hinkelmann(1966)and subsequently Kirk and his colleagues(Kirk et al,1970;Kirk,1971)and Ananthakrishnan et al (1973)have shown,on a wide variety of populations,that in matings where the father is incompatible with the mother with respect to the ABO groups, the children show a higher frequency of the $Hp^1$ gene than do those of families, drawn from the same population,inwhich the father is ABO compatible.This was explained as due to deaths from haemolytic disease of the newborn resulting from ABO incompatibility(though mostly unrecognized as such),and related to the fact that the product of the $Hp^1$gene is more efficient than that of $Hp^2$in removing dissolved haemoglobin from the plasma conserving the contained iron. Thus in cases where foetuses,or newborn are affected by haemolytic disease due to foetal A or B inherited from the father, a child who is of type Hp1-1 should have the best, and one of type 2-2 the worst chance of survival. Family studies by Vana and Stienberg(1975) confirmed the tendency towards raised $Hp^1$frequencies among the offspring of ABO incompatible matings, but fur-

ther analysis of the data has shown that the $Hp^1$ frequencies are intrinsic to the blood groups themselves,irrespective of the type of mating from which they come. They conclude that there is a tendency,which they cannot at present explain,for $HP^1$frequencies to increase with blood group in the order:O,A, B,AB and indeed in the order of genotypes:OO,AO,BO,AB,AA,BB. This explanation does not, of course, exclude some form of underlying natural selection either of gametes or zygotes.

In most populations only the three phenotypes 1-1,2-1, and 2-2 are commonly distinguished, a number which is increased to six if tests for the variants of $Hp^1$ are performed. Beside these, however, a few variant phenotypes are sufficiently common to be found in some random population surveys. One of these is known as HP2-1M.,the M standing for 'modified'. This phenotype shows the bands of Hp2-1, but with the slower bands (higher polymer) increasingly faint. This type, found mostly in Negroes, is probably due to variant of the Hp2 gene which, however, gives rise to a homozygote indistinguishable from normal $Hp^2/Hp^2$, or sometimes to Hpo.Another type,even commoner,and also found mostly in Africans,is that usually known as Hpo,and characterized by an apparent absence of haptoglobin; it was originally regarded as the product of an amorph allele. Though a very few cases may be due to such an allele, the majority are not directly due to genes at the Hp loci.There are in fact a series of grades of deficiency of haptoglobin, and by physicochemical methods of concentration it can often be shown to be present,and its type can be determined,when initial tests appeared to show complete absence.Haptoglobin is shown to be depleted or absent in cases of haemolytic anaemia.However,the type is found with undue frequency in children both of whose parents

are Hp2-1M. It is likely that Hp0 or ahaptoglobinaemia is the result of a combination of environmental and genetic factors, not necessarily all at the Hp locus.

Though the quantitative variant phenotype Hp0, may in most cases be of environmental origin,e.g. haemolysis, there seems no doubt that there also occur individuals with no detectable haptoglobin,or with only very minute amounts,in the absence of any haemolytic process (Harris,1961).

Factors affecting the maintenance of the $Hp^2$ gene have been attributed to selective advantage conferred by environment such as malaria(cited in Walter and Steegmuller,1969). It is also well established that the capacity of haptoglobin to bind haemoglobin varies considerably among Hp types.The Hp1-1 type has a greater capacity than other: Hp1-1 > Hp2-1 > Hp2-2. It is possible that the relatively high incidence of haemolytic conditions,especially anaemias,known to occur among tropical populations,is related to a possible advantage in haemoglobin binding capacity,which one haptoglobin type may have over others.From this it has been inferred that Hp2-2 is selected against in areas with prevalent haemolytic diseases(Baxi and Camoens,1969).Weitkamp et al(1972)also found a positive correlation between ahaptoglobinaemia and malaria infection.

A considerable number of rare phenotypes found in the haptoglobin system are classified as quantitative and qualitative variants. In addition to the Hp2-1M and Hp0 phenotypes, other rare phenotypes of quantitative category are Hp2-1'Carlber', Hp2-1 (Haw) and Hp2-1 ( Trans). The qualitative variants are very rare and the best known are Hp-1J, Hp-2J and Hp-Mb.

Despite much investigation the relation between genetics

and physiology in this system is by no means fully understood. The Hp and ABO genes appear to interact ante-natally through some form of selection, to affect the frequencies of various combinations of ABO and Hp phenotypes, as shown by numerous family studies.

## 2.2.2. The transferrin (Tf) system.

Transferrin or siderophilin is the iron-binding protein component of the plasma. It is a $\beta$ -globulin, containing 5.5 percent of carbohydrate, which combines with inorganic iron present in the plasma and transfers it to the receptor cells of the bone marrow and other storage depots. Like haptoglobin, trnasferrin has long been known as a well defined plasma protein when Smithies (1957) first demonstrated by means of Starch-gel electrophoresis, the existence of inherited variation in its molecular structure. It exists as a number of genetically determined variants, migrating at different speeds. The protein part of the molecule appears to be one long polypeptide chain coded by a single gene.

By far the most common type in all populations is known as C. The other variants have been labelled with regard to their electrophoretic mobility in relation to this type. Types which migrate faster than TfC are known as TfB with an appropriate suffix and those which migrate more slowly are similarly classed as TfD. Suffixes are sometimes geographical, or ethnographic, as in $D_{chi}$ and $D_{montreal}$. In other cases the relative speed of migration is indicated as in $B_{1-2}$, intermediate between $B_1$ and $B_2$. Gene notation is typographically awkward, involving suffixes to indices as $Tf^{D}chi$. Variants other than C are relatively uncommon, and have generally been found in combination with C, the usual variant being present in amounts

about equal to that of C.

Family studies by Horsfall and Smithies (1958) suggested that there exists a series of allelic genes, each of which determines the formation of a particular Tf type. Individuals with two of the above Tf genes appear to be heterozygotes, individuals with one, homozygotes. Smithies and Hiller (1959) reported that the formation of $\beta$-globulins B, C, and D is determined by three co-dominant autosomal allelic genes, $Tf^B$, $Tf^C$, and $Tf^D$, the C gene being much more common than B or D genes. The band or bands of transferrin can as a rule be recognized by inspection of a general-protein-stained electrophoretogram, lying between haptoglobin and albumin ( Figure 2.2.2.a).

For definite identification, however, one of a number of specific tests should be used. As a rule only a single band is seen, corresponding to the common TfC phenotype, representing the homozygote of the most common gene. A great many genetically determined variants of transferrin are now known, but all are rare, and most are very rare, so that they are found almost exclusively as heterozygotes in combination with the common type. Variant homozygotes, and heterozygotes of two variants combined, are very rare indeed. At present, the polymorphism of transferrin is attributed to 22 allelic genes. (Smithies, 1957; Giblett, 1969; Walter, 1975). ( Figure 2.2. 2.b).

But only four variant phenotypes are known to have frequencies above one percent in any population, these are the heterozygotes of $Tf^D1$, $Tf^Dchi$, $Tf^B2$, and $Tf^B0\text{-}1$.

The application of isoelectric focusing to the analysis of transferrin has revealed considerably more genetic hetero-

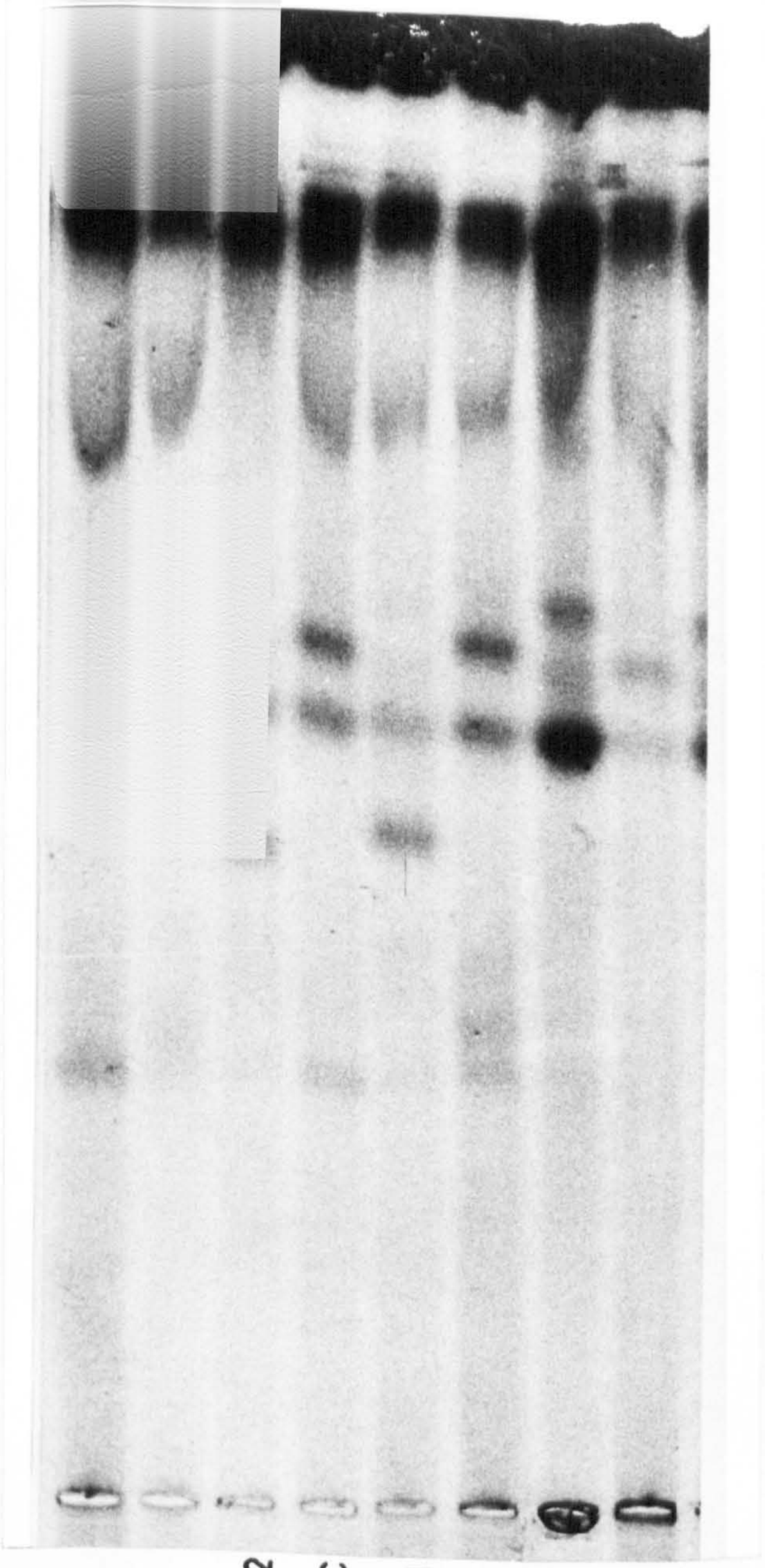



Fig. 2.2.2. a. Agarose gel electrophoretic patterns of the common transferrin C and some rare CB and CD phenotypes (Farhud, D.D. 1972).

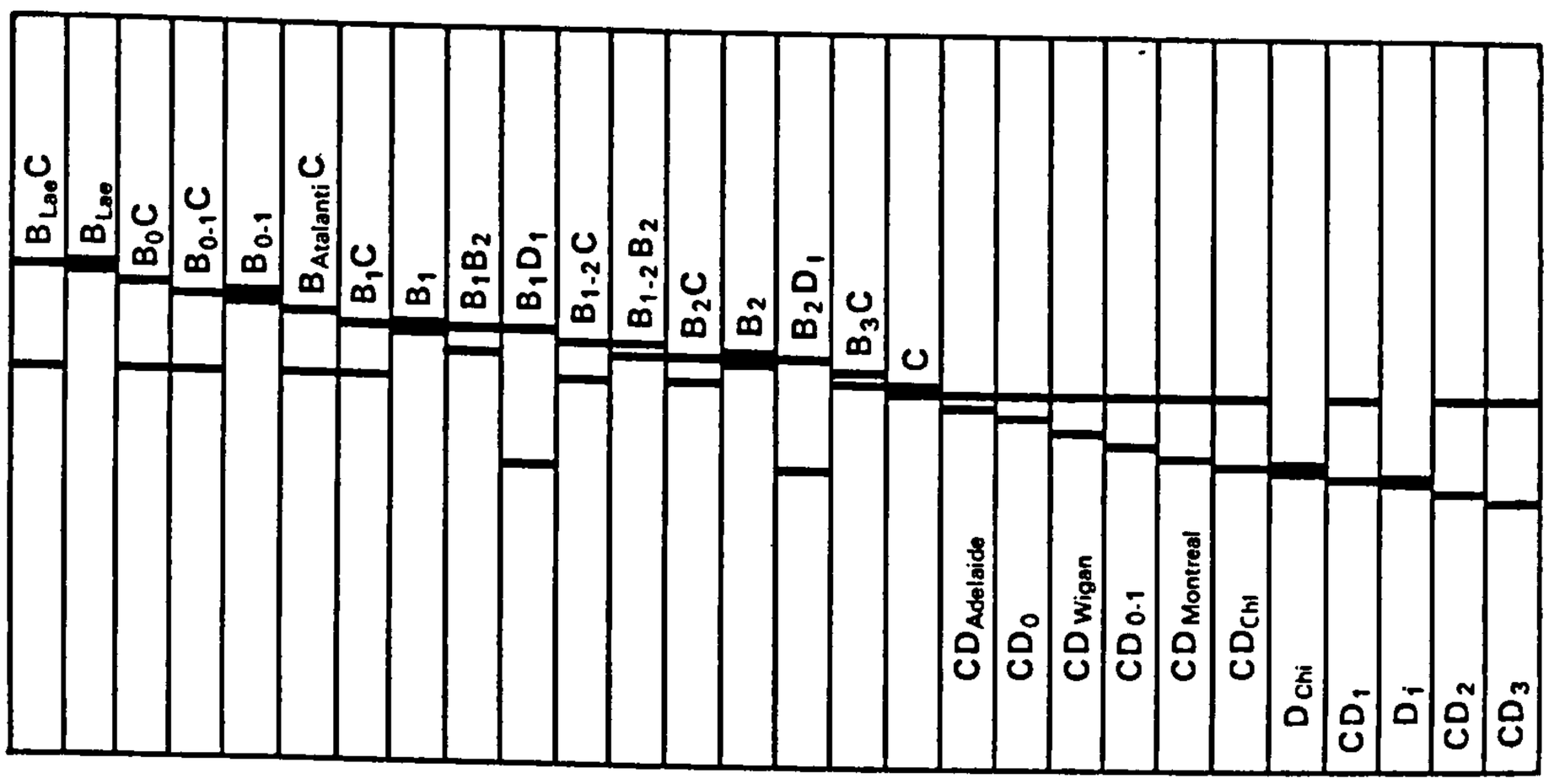



Fig.2.2.2.b. Diagram of the 28 reported transferrin phenotypes in human serum, representing TfC and 17 variants with slower or faster mobility ( Giblett, E.R. 1969).

geneity than was apparent by electrophoretic techniques. Kuhnl and Spielmann (1978), Thymann (1978), Kuhnl and Spielmann (1979), Kuhnl et al (1979), Altland et al (1980) and Constans et al (1981) have recently described two common subtypes of the C allele, $C_1$ and $C_2$, and three rare ones $C_3$,$C_4$, $C_5$ in some European populations, which revealed a clear distribution heterogeneity of this polymorphism within Europe. As far as we know, the only transferrin subtyping performed on population samples from other parts of the world is that of Walter et al (1981) on six Indian population samples.

No differences have been detected in iron-binding capacity of the common type and the different variants of the transferrin molecule. Whatever functional differences may exist, the rarity of the variants suggests that the common type has under most circumstances a selective advantage over all the others. This contrasts with the situation in cattle where several alleles are of comparable frequency and a complex system of selective fertility exists ( Ashton, 1965).
Because of the rarity of variants, and the even greater rarity of families including members with two different ones, there has been no comprehensive proof of the hypothesis that all variants are the result of mutations at a single locus. However, as pointed out by Giblett, this hypothesis is supported by the simple Mendelian behaviour of the individual variants, the absence of any record of individuals with three kinds of transferrin, the fact that the molecules appear to differ only in electrical charge and not appreciably in molecular weight, and in a few cases by the demonstration that the variants differ from the common type only in the substitution of a single amino-acid for another.

So far no specific association between an unusual Tf variant and any particular clinical disorder has been found. All transferrin variants, other than TfC, are rare, so that even if they showed disease associations or other selective effects, this would be difficult to prove. However, because of the occurrence in cattle of a well-defined system of balanced polymorphism of the transferrins ( Ashton, 1955), it is possible that such a situation could emerge in a human population having a high frequency of one of the variants. Walter and Bajatzadeh (1971) have suggested that the frequencies of the Tf alleles are not distributed equally within the human species and show marked racial differences with respect to the alleles, $Tf^C$ and $Tf^D$. Races living in tropical biotopes show high $Tf^D$ frequencies and the populations in non-tropical biotopes have low frequencies. They assumed that such geographical distribution is a result of selective adaptations to particular environmental conditions, so that the relatively high $Tf^D$ gene frequencies in tropical biotopes could indicate a better physiological functionning of these variants. Ashton (1965) reported a positive association between another slow variant, $Tf^E$, and tolerance to hotter climate in cattle. The assumption that Tf is associated with resistance to infectious diseases such as malaria appears to be unfounded (Curtain et al, 1965). Selective mechanisms which might be responsible for maintaining the four common 'aberrant' phenotypes in certain populations are still unknown.

2.2.3. The third component of human complement (C3) system

Complement is a complex mixture of blood proteins which are activated sequentially, many by conversion of a proteolytic zymogen into an active proteinase, and which play an

important role in the host defence against infection. They were recognized at the end of the last century by their ability to cause the lysis of bacterial cells that had been agglutinated by specific antibodies. Antibody-coated red blood cells soon replaced the bacterial cells as the test system in the assay of complement, as measurement of the haemoglobin released offered an easy method of estimating lysis. Complement is an important part of an animal's defence mechanism against pathogenic microorganisms, but subsequent work has suggested that some of the proteins in the system may have a direct role in the immune response itself by facilitating intercellular interactions. Most recently, four of the complement components have been found to be coded by structural genes located in the major histocompatibility complex. This again has suggested, by the analogy with other products of this genetic complex, that complement proteins may have a function in the cell surface in addition to their activity as an effector mechanism in the destruction and elimination of foreign antigenic material.

As many as 20 proteins in the blood may be part of the complement system, which has two pathways of activation as well as a variety of control mechanisms which include inhibitors and inactivators specific for different steps or components. Most of these proteins have now been isolated and have been characterized at least in part (Gotz and Muller-Eberhard, 1976; Lachman, 1973; Muller-Eberhard, 1975).

The major component of human complement ($C_3$ or $\beta_{1c}$ - globulin) was first isolated by Muller-Eberhard (1960). He

isolated a beta globulin from normal serum and showed that it was one of the proteins comprising the classic third component of complement. With the characterization of the remaining five proteins of this group, the term $C_3$ or third component has been reserved for the first protein of the group to be purified and characterized and the first of this group to be activated during complement fixation, formerly called $beta_{1C}$-globulin. The remaining proteins of the classic third component of complement have been numbered $C_5$ to $C_9$, in order of their participation in full complement activation (Muller-Eberhard et al, 1966; Bulletin of World Health Organization, 1968). The numbering of the first three complement components in the activation sequence, $C_1$, $C_4$ and $C_2$, persists with all the illogical force of conservative tradition.

In addition to being part of the classic third component of complement, $\beta_{1C}$- globulin ( Muller-Eberhard et al, 1960) has been shown to play a key role in the mechanism of immune adherence ( Nishioka and Linscott, 1963), conglutination, immune conglutination ( Lachmann and Coombs, 1965), and erythrophagocytosis in vitro ( Nelson, 1962) and in vivo (Mollison, 1965). Thus, $C_3$ has many important functions in the immune mechanisms (Muller-Eberhard et al, 1966).

$C_3$( $\beta_{1C}$ )is labile on storage in serum, and the main conversion product is a $\beta$-globulin called $\beta_{1A}$ globulin. The conversion of $\beta_{1C}$ to $\beta_{1A}$ globulin, whatever its underlying mechanism, is of immunological importance because it leads to complete loss of the serological activity associated with the intact $\beta_{1C}$- globulin ( Muller-Eberhard and Nilsson, 1960). Thus, $C_3$ converts during electrophoresis or when aged into $\beta_{1A}$- globulin ( West et al, 1965; Laurell and Laurell,

1967) which has a faster electrophoretic mobility than $\beta_{1C}$. After electrophoresis the phenotypes are characterized by proteins appearing into regions. The slower group ( $\beta_{1C}$)migrates behind $\alpha$2-macroglobulin, the faster one( $\beta_{1A}$)behind transferrin. When sera were stored for 24 hrs at room-temperature or at -20° C for more than 2 months, proteins in the region behind $\alpha$2-macroglobulin were found to disappear. The pattern of proteins in the region behind transferrin remains stable. It is advisable to use fresh serum samples. The interpretation of the bands in the faster regions is uncertain, because the protein bands of one type may overlap the bands of another type( Goedde et al, 1970).

Study of the genetically controlled molecular variation in human $C_3$ has been greatly facilitated by the circumstance that this protein occurs in normal serum in concentrations allowing it to be visualized easily in almost any zone electrophoresis of whole serum. The visualization is further improved if calcium is added to the electrophoresis buffers so that $C_3$ is slowed relative to transferrin and beta lipoprotein, an observation first made by Laurell, Laurell and Skoog (1956).

A polymorphism of the third component of complement($C_3$) in serum was described by Ropartz et al in 1965,using methods of hemagglutination inhibition of $C_3$-coated red cells.No family studies were reported.With the usual methods of agar or starch-gel electrophoresis $C_3$( $\beta_{1C}$-globulin)is seen as a single band in the slow $\beta$ -globulin region.Variants of this protein were suspected when Wieme(1965)noted that a few rare sera had an extra band in the $C_3$region of either slower or faster electrophoretic mobility than the usual $C_3$on agar electrophoresis

and that the extra bands disappeared with storage of the sera. By the use of high voltage electrophoresis on agar Wieme and Demeulenaere and Wieme and Seyers in 1967 and 1968,respectively, demonstrated duplication of $C_3$ bands in certain families from a Flemish and a Bantu population. Family studies indicated simple autosomal codominant inheritance of variant allele.

Genetically determined polymorphism of the third component of human complement ($C_3$) was demonstrated simultaneously by two research groups. Alper and Propp (1968) used a prolonged high voltage agarose electrophoresis and Azen and Smithies (1968), high voltage starch-gel electrophoresis. Both groups made observations consistent with the theory that the phenotype of $C_3$ is governed by autosomal co-dominant inheritance and presented evidence for the existence of two relatively frequently occurring genes as well as of rarer genes controlling this polymorphism. The discoverers of the $C_3$ polymorphism use different nomenclatures. Alper and Propp propose the symbols F and S for the common allele products, where F is the fastest (anodal) and S the slowest (cathodal ) migrating band on electrophoresis. The F and S bands correspond to the bands 1 and 2 in the nomenclature of Azen and Smithies. The two groups exchanged sera with different $C_3$ phenotypes and found this correspondence of $C_3$ components: 1=F, 2=S. The most frequenty occurring phenotypes thus are SS(or 2-2), FS (or 2-1), and FF ( or 1-1).

As mentioned above, $C_3$ proteins can be seen as distinct bands in the slow $\beta$ -globulin region ( $\beta_{1C}$- globulin). It has been postulated that each allele in the system controls a pair of protein bands, a heavy-staining band and, somewhat anodal to this, a lighter-staining band. $C_3$ types always re-

fer to the pattern of the cathodal heavy-staining bands.Normally, the $C_3$ fast band stains slightly more intensely than $C_3$slow bands in $C_3$ heterozygous individuals( i.e.C3FS)(McLean and Hoefnagel, 1980).

At the moment, our present assemblage consists of more than 20 structural electrophoretically distinct variants of $C_3$ in human sera. Two genes $C_3^S$ and $C_3^F$ and three phenotypes $C_3$SS, $C_3$FS and $C_3$FF are found in more than 99% of the populations. (Figure 2.2.3.a). All the other variants due to mutated alleles at the $C_3$ locus are very rare. The rare variants of $C_3$ have been described by different authors (Rittner and Rittner, 1974; Alper and Rosen, 1976). Figure 2.2.3.b. shows the twenty structural electrophoretically distinct forms of $C_3$ in human sera.

If the original fast variant is called $C_3F_1$ and the original slow variant is $S_1$, then any new variant may be assigned as subscript which expresses its migration relative to $C_3$S and either one of these variants. Thus, a variant migrating 60% of the distance between $C_3$S and $C_3S_1$ is called $S_{0.6}$. Another variant migrating a head of $F_1$ is $C_3F_{1.1}$ and so on.

The variant protein bands reported so far have been named $F_{1.2}$' $F_{1.1}$' $F_1$' $F_{0.85}$' $F_{0.8}$' $F_{0.5}$' $S_{0.4}$' $S_{0.6(or\ 3)}$'$S_{0.8}$'$S_{0.9}$ and $S_{1(or\ 4)}$in order of descreasing electrophoretic mobility (Alper, 1969; Azen and Smithies, 1968; Teisberg, 1971; Farhud, 1972).

In addition, the existence of a 'silent' or non-expressed allele of the system has been suggested.

Genetically controlled deficiencies of almost all complement components in humans have been described (Donaldson and Evans,1963; Klemperer et al, 1966;Abramson et al,1971; Leddy

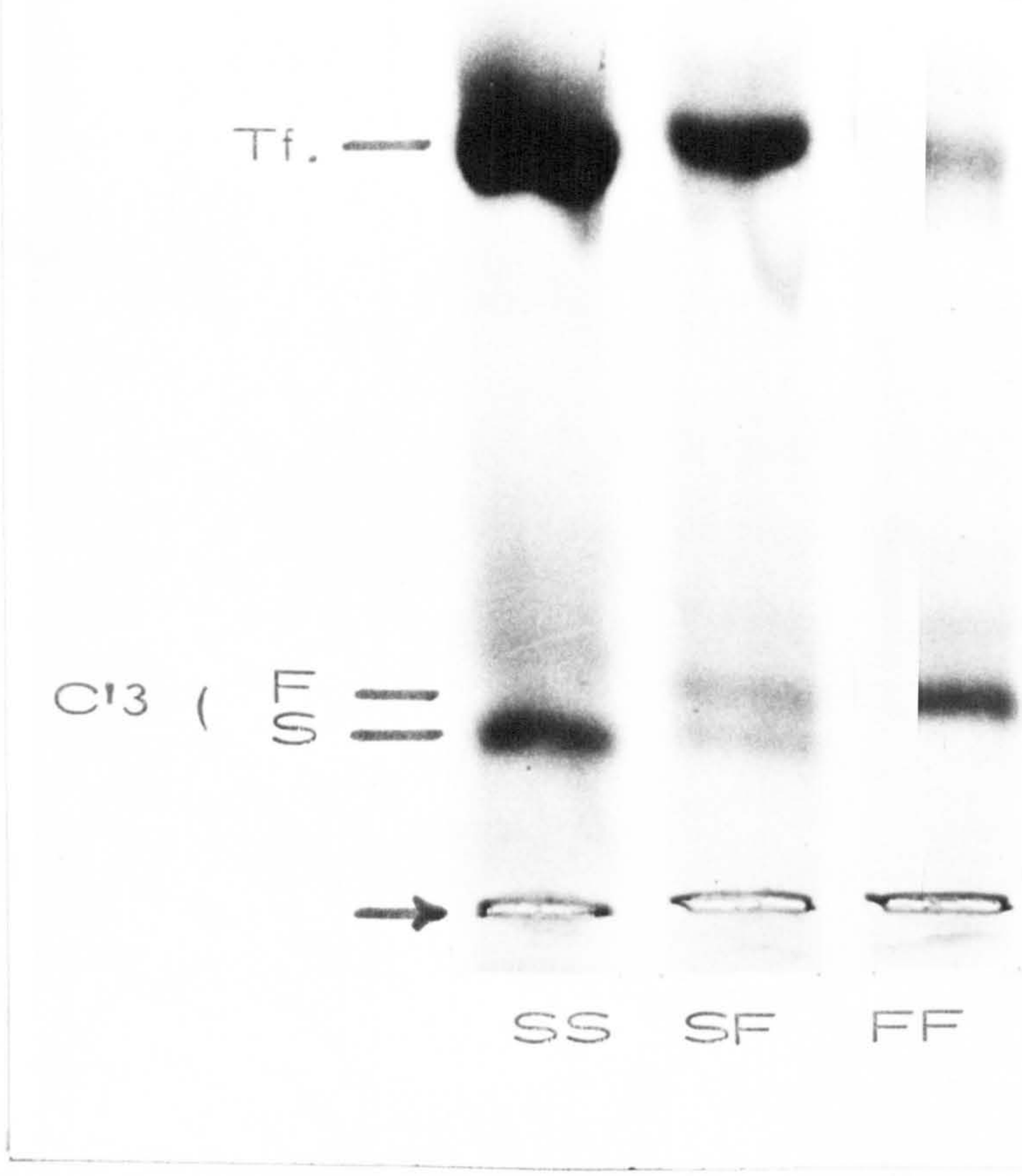



Fig.2.2.3. a. Agarose gel electrophoretic patterns of the three common C3 phenotypes, C3 SS, SF and FF (Farhud,D.D. 1972).

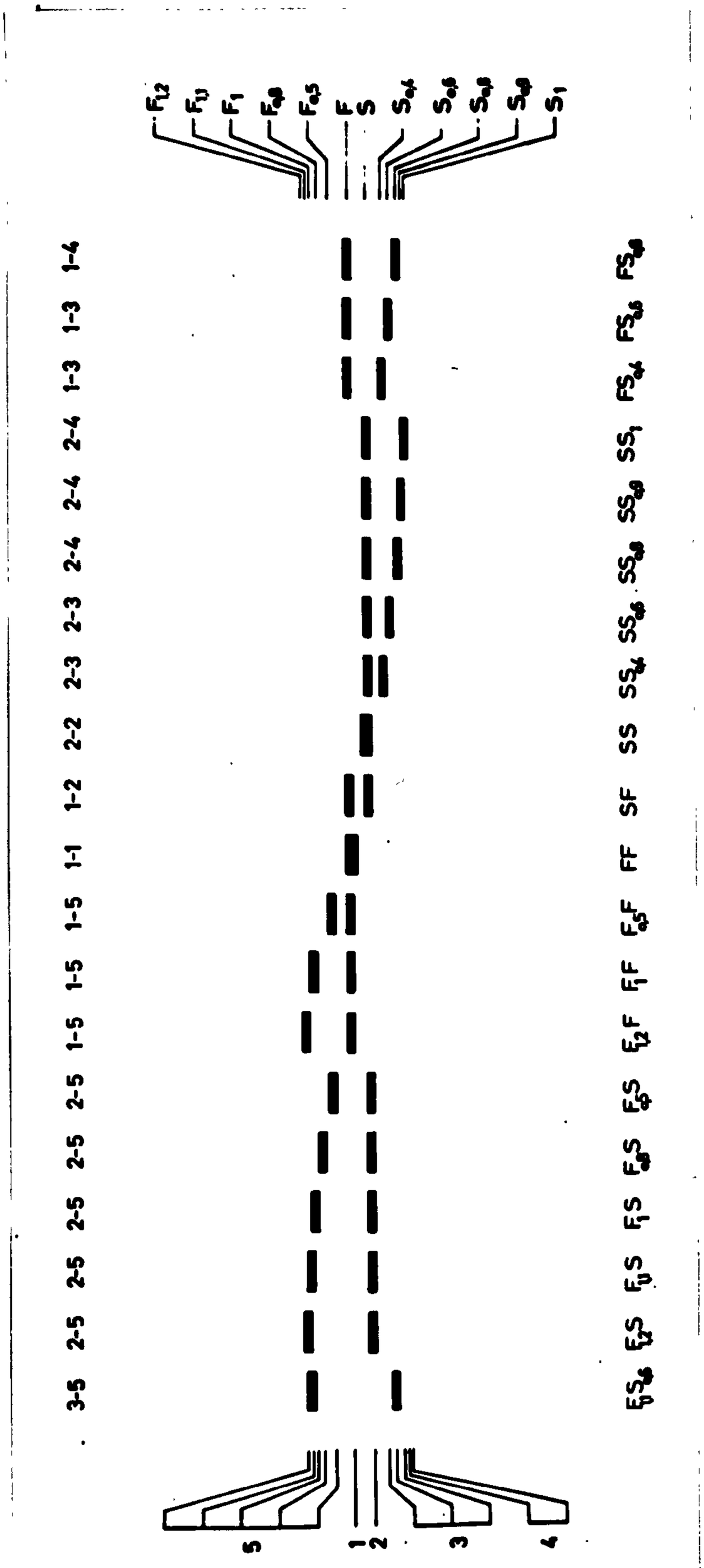



Fig.2.2.3.b. Diagram showing the twenty structural electrophoretically distinct variants of C3 in human sera. The nomenclature of Azen and Smithies (1968) is shown on the left and that of Alper and Propp (1968) on the right ( Farhud, D.D. 1972).

et al, 1974; Boyer et al, 1975; Peterson et al, 1975; Rosenfeld et al, 1976). Most of these cases were discovered more or less incidentally due to their increased susceptibility to infections or during other clinical investigations.A case of inherited partial deficiency of $C_3$was first discovered by Alper et al in 1969.The family survey revealed a $C_3^-$ gene in 7 family members.Other cases of inherited partial deficiency of $C_3$ were reported by Alper et al(1972),Alper and Rosen(1976). $C_3$deficiency regarded as a rare homozygote has been reported by Alper et al (1976)and by Papiha et al (1979). The association of $C_3$ phenotypes and various diseases were first reported by Farland and Raeder(1972).The $C_3S$ phenotype has been found to be associated with higher titre of immune antibodies than the $C_3FS$ and $C_3F$ phenotypes in normal persons(Bronnestam and Cedergren,1973).The observation that there is a significantly lower $C_3^F$gene frequency in pyelonephritis could support the assumption that the $C_3S$ phenotype is in some way less efficient than $C_3F$ phenotype in combating infections(Stoffersen and Jorgensen, 1980).

All $C_3$ phenotypes have the same haemolytic activity (Alper and Propp,1968)and are found in the same quantity in human blood (Agarwal et al, 1972).Arvilommi(1973)has shown that there is a difference between the common phenotypes ($C_3F$, $C_3FS$, $C_3S$) in their ability to bind mononuclear cells. The $C_3F$ has a greater binding capacity than the $C_3S$ type. The different phenotypes of the $C_3$polymorphism seem to have different biological properties, as shown by their association to various diseases.

A high incidence of $C_3F$ phenotype has been found in patients with Rheumatiod arthritis (Farhud and Walter, 1972; Bronnestam, 1973), atherosclerosis (Disssing et al, 1972),hepatitis

(Farhud et al, 1972), renal diseases (Stoffersen and J rgensen, 1980), cystic fibrosis (Schi tz et al, 1978).

Family studies have so far supported the theory of autosomal codominant inheritance of $C_3^{F1}$, $C_3^{F}$, $C_3^{S}$, $C_3^{S}0.6$, and $C_3^{S1}$.

It was of interest to determine whether the $C_3$ variants all functioned normally as the third component of complement. Total haemolytic complement was normal in all variant-containing sera. This, of course, is a rather gross measure and even if a variant were totaly inactive, haemolytic complement might be normal or only slightly reduced since, except for $C_3F$ and $C_3S$ all the variants have been found only in heterozygotes and $C_3$ concentration must be less than 50% of normal to effect a lowering of the $CH_{50}$ ( or total haemolytic capacity).$C_3$functional activity paralleled immunochemically determined concentration in $C_3$ heterozygous sera containing each of the variants (Alper, 1970).

There seem to be racial differences in $C_3$ allele frequencies which may become valuable in anthropological studies (Teisberg,1971). It is clear that the gene frequencies differ strikingly among the races of man. The allele frequencies of human $C_3$ polymorphism found in some population groups indicate that the system may become a valuable tool in practical work in the field of human genetics.

The S gene is common in the three major races of man. The F gene is relatively common in Caucasians,less common in American Negroes,and relatively uncommon in Orientals.The $F_1$,$S_1$and $F_{0.8}$ genes are probably rare in all populations. In most Caucasians, the $C_3S$ allele has an approximate gene frequency of 0.80, while the F gene has a frequency of about 0.20 (Seth

and Seth, 1976). The other allotypes, $F_1$, $F_{0.8}$, $F_{0.5}$, $S_{0.6}$ and $S_1$, are relatively rare. However, the SS type is the common in all populations studied thus far ( Alper and Propp, 1968; Azen and Smithies, 1968; Berg et al, 1970; Bronnestman, 1971). As mentioned above, the F gene appears to be rare in Negroes since it may be that the low incidence of $C_3^F$ in American Negroes represents a mixture of Caucasian genes (Alper, 1970). At present, nothing can be said about the possible selective effects of climate and living conditions on the allele frequencies. The allele frequencies of $C_3$ in Caucasian population groups (Alper and Propp, 1968; Azen et al, 1969; Teisberg, 1970; Farhud and Walter, 1973) indicate that the system may become useful in cases of disputed paternity. Much reference material is a prerequisite to the use of this polymorphic system in forensic serology. It appears that the rare $C_3$ proteins described up to now can be used as genetic markers in forensic serology. It was feared, however, that the lability of $C_3$would prove to be a major obstacle to its forensic use as these blood samples may be exposed to unfavourable conditions for several days during shipment.

Since $C_3$ is present in serum in a concentration higher than that of the other known complement components and is vitally important in immune mechanism (Muller-Eberhard, 1968), investigations of the $C_3$ polymorphism is of interest for both geneticists and immunologists.

The results obtained so far on $C_3$ polymorphism suggest that the system should be a valuable marker in population studies. The instability of the complement component $C_3$ may, however, cause some practical problems in the field of population genetics, since a certain fraction of serum samples may be diffi-

cult to type with certainty, due to storage alteration.

2.2.4. The group specific component (Gc) system

The Gc group of the plasma is the expression of genetically controlled variants of one of the ∝2-globulins. It was not recognized as a distinct protein until 1959 when, Hirschfeld, by means of immunoelectrophoresis of human serum, showed the precipitation pattern of this ∝2-globulin besides haptoglobin as showing systematic variation in appearance and migration rate. The three phenotypic patterns consist of a fast, a slow, and an intermediate (bimodal) arc. Mixture of sera containing the slow and fast types resulted in a pattern indistinguishable from the intermediate type. This variable globulin was named "Group specific component" or Gc. Family studies by Hirschfeld et al (1960) showed that the two main Gc proteins are the product of a pair of autosomal codominant alleles, called $Gc^1$ and $Gc^2$, giving rise to the expected three phenotypes, Gc1-1, Gc2-1, and Gc2-2, both genes being expressed in the heterozygote to give a mixture of the two proteins. Thus, individuals homozygous for $Gc^1$ have the fast moving component; those homozygous for $Gc^2$ have the slow-moving component; and heterozygotes have the intermediate arc containing both components. By means of immunoelectrophoresis each homozygous serum appears to contain a single Gc protein, and the heterozygote a mixture of the two. Starch-gel electrophoresis shows two bands for each homozygote, and three for the heterozygote, but the two bands for a given gene product may represent a single substance with different electrical charges. On starch-gel electrophoresis this protein migrates between transferrin and albumin ( Figure 2.2.4.a ).Recently, a new technique of Gc typing based on isoelectric focusing, polyacrylamide gel

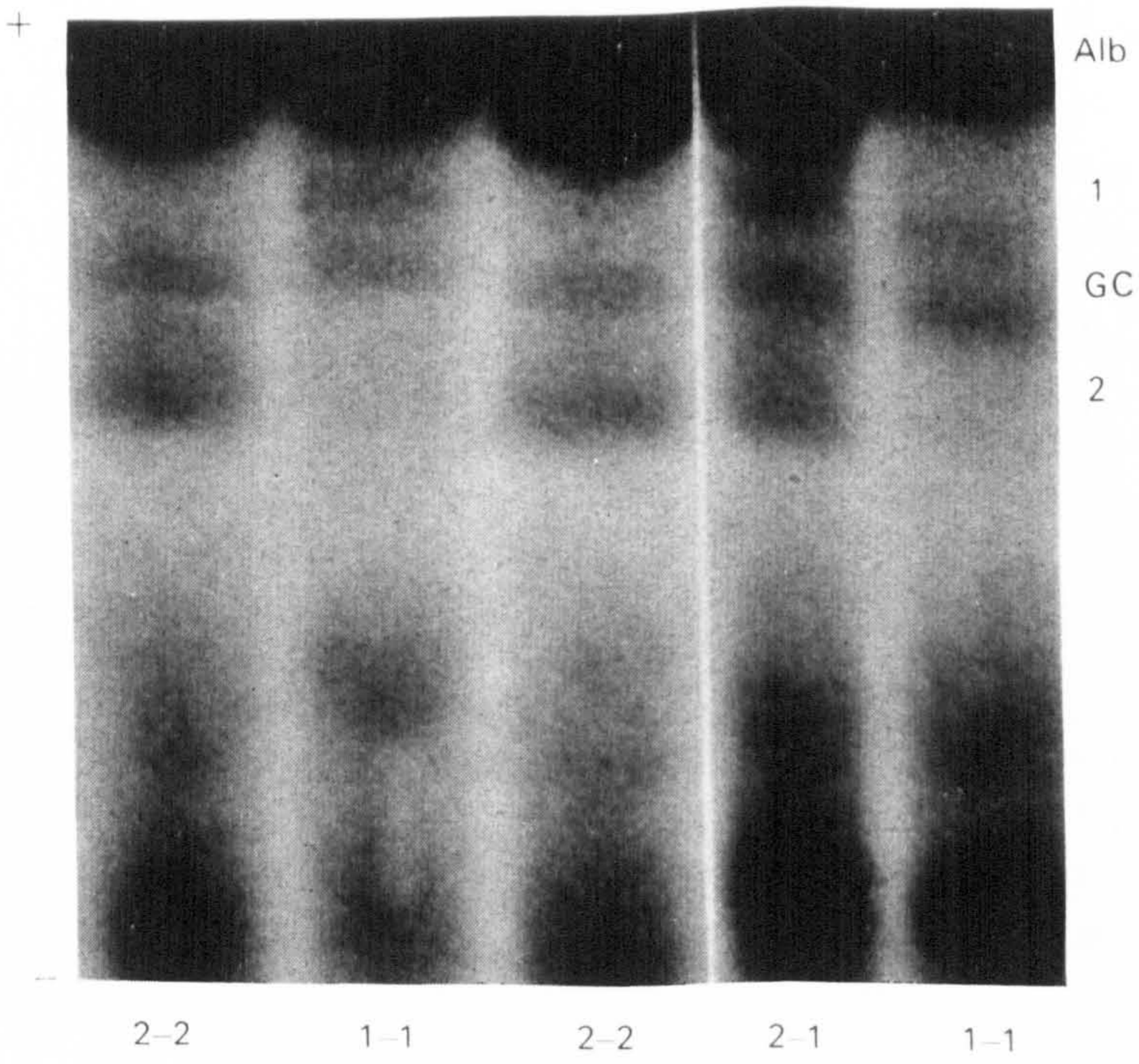



Fig. 2.2.4.a. Starch gel electrophoretic patterns of the three common Gc phenotypes Gc 1-1, 2-1 and 2-2(Bearn,A.G.,et al.1964).

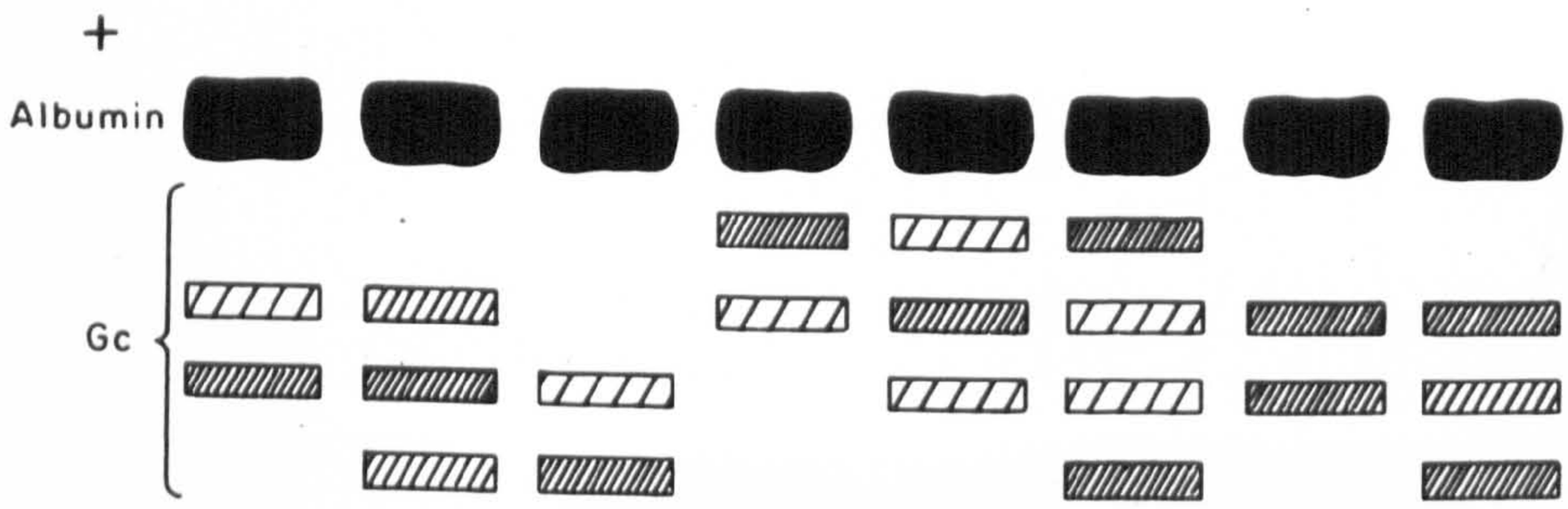



Fig.2.2.4.b. Diagram of the three common Gc 1-1, 2-1 and 2-2 and some rare Gc phenotypes (Kitchin, F.D., and Bearn,A.G. 1966).

isoelectrofocusing (PAGIF) with immunofixation was elaborated by Constans and Viau ( 1977), Constans et al (1978). Thereby, it was possible to demonstrate subtypes of the $Gc^1$ genes and several additional rare Gc variants. This method revealed heterogeneity in the $Gc^1$ band zone, which is explained by the existence of two $Gc^1$ suballeles, $Gc^{1F}$ and $Gc^{1S}$; the resultant phenotypes are Gc1F-1F, Gc1F-1S, Gc1S-1S,Gc2-1F, Gc2-1S and Gc2-2. This six Gc phenotypes can be explained by the three common alleles, $Gc^{1F}$, $Gc^{1S}$ and $Gc^2$. In addition to the common alleles, a considerable number of alleles are known, all of which are everywhere rare. In fact, of the aberrant Gc genes, only $Gc^{chip}$ and $Gc^{Ab}$ occur with a frequency of one percent or more in any known population.( Figure 2.2. 4.b). At an international workshop on the Gc system held in July 1978 ( Constans et al, 1979) the Gc phenotypes determined by a total of 30 different alleles were demonstrated. Of these Gc variants, 21 are presumably mutants of the $Gc^1$ allele, and 9 are presumably derived from the allele $Gc^2$. The existence of a rare $Gc^O$ ' silent' allele was suggested by Henningsen (1966) from studies of a family with anomalous inheritance. In nearly all previous population studies, the $Gc^2$gene has a lower frequency than $Gc^1$:

The importance of the Gc groups, other than as genetical markers, has recently been realized as a result of the discovery by Schanfield et al (1975) that the Gc proteins are the vitamin D carriers of the plasma. Subsequently Daiger et al (1975; 1977) showed that this protein is capable of binding vitamin D, a feature confirmed by many other experiments ( Van Baelen et al, 1978; Haddad and Walgate, 1976 ).

The metabolic role of the protein and its polymorphism contribute to its importance in clinical and biochemical research. In addition, the fact that it is observed at different frequencies of the Gc genes in different population studies, makes a knowledge of the genetic composition of a population imperative in studies of many disease states. Wendt et al (1968) and Cleve (1973) have summarized work on associations of diseases with Gc types. There are indications that Gc1-1 may be associated with psoriasis ( Jorgensen and Hopfer,1967) and less probably with carcinoma of the uterus, neurodermatitis, diabetes mellitus, and rheumatoid arthritis. Kitchin et al (1972) suggest an association of the $Gc^{Ab}$ variant with Kuru in New Guinea. As shown by Mourant et al (1976) Gc has in general, though with several exceptions, a high frequency in sunny climates and $Gc^{2}$ a relatively high one in dull ones, suggesting that the Gc types are affected by natural selection related to the availability of vitamin D. It is therefore desirable that Gc distribution should be investigated in patients with rickets, which results from vitamin D deficiency, as well as if possible, in the very rare condition of hyper-vitaminosis-D.

Kirk et al (1963), Walter and Steegmuller (1969), Mourant et al (1976), and later Daiger (1979) have demonstrated that the frequency of the gene $Gc^{2}$ follows a cline, diminishing as the mean intensity of solar radiation increases. The data recovered in the investigations show the presence of a gradient in the frequency of the gene $Gc^{1F}$ which increases from Europe to equatorial Africa. This gradient may be superposed on that of skin pigmentation (Loomis, 1967) which has always been considered to be linked to genetic and adaptative

conditions ( Hiernaux, 1977). The lowest frequencies of the gene $Gc^2$ correspond to regions where sunlight is stronger. There is also a north-south gradient in the $Gc^{1F}$ gene frequency. This seems to parallel the gradient seen in skin pigmentation. Group specific component polymorphism, as studied by modern electrophoretic techniques, represents an extremely instructive marker system for anthropological research. The frequencies of the Gc alleles and their presence or absence in the populations studied help us to evaluate the population dynamics. Every human population studied to date is polymorphic for the Gc protein. Even in a population completely lacking the $Gc^2$ allele, a polymorphism is present, because the Gc system is multiallelic. The distribution of the Gc alleles seems to correlate with geographical clines, notably the incidence of solar radiation as might be expected for the protein which transports vitamin D.

2.2.5. The ceruloplasmin (Cp) system

Ceruloplasmin is an $\alpha$ 2-globulin, which contains more than 90 percent of the plasma copper. It is a glycoprotein with a 7 percent carbohydrate content. Each molecule contains eight copper atoms ( Holmberg and Laurell, 1951). It was discovered in 1948 by Laurell and Holmberg. The protein is known to have oxidase activity in vitro ( Holmberg and Laurell, 1951; Walaas et al, 1967).

Though its biological function is not yet quite clear, it seems to play a role in copper metabolism, such as being involved in the enzymatic transfer of copper to copper containing enzymes like cytochromoxidase ( Bearn, 1966; Schultz and Heremans, 1966; Schreffler et al, 1967, 1968, Shokeir and Schreffler 1969; Marceau et al, 1970).

Genetically determined structural variants of ceruloplasmin was detected by McAlister et al ( 1961); and Martin et al (1961). They described a fast moving electrophoretic variant of this protein, which was found to be controlled genetically. However, no further evidence of inherited variants of this protein was obtained until the studies of Schreffler et al (1967, 1968), demonstrated a genetically controlled polymorphism of the ceruloplasmin . It has long been known that different plasmas tend to show different band patterns of ceruloplasmin on electrophoresis, but as the molecule is somewhat unstable it proved difficult to distinguish between gene controlled variants and the effect of molecular dissociation. As Schreffler et al (1967) showed, however, under suitable conditions clean band patterns can be obtained, each of five alleles giving rise to a characteristic band, and each heterzygote showing the bands of the two constituent genes. On the basis of family studies Schreffler et al assumed the existance of three allelic genes: $Cp^{A}$, $Cp^{B}$ and $Cp^{C}$ at the autosomal Cp-locus, determining six phenotypes: CpA, CpB, CpC, CpAB, CpAC and CpBC. Two further phenotypes: CpB-NH and CpA-NH produced by an additional allele $Cp^{NH}$ (NH. Stands for new haven) have been demonstrated in 1967 by Shokeir et al ( Figures 2.2.5.a, b and c). The electrophoretic migration rates of the product of the alleles in the starch gel decrease in the order: $Cp^{A}$, $Cp^{B}$, $Cp^{NH}$, $Cp^{C}$. Finally, Shokeir et al (1968) found a further ceruloplasmin variant in Tailand, controlled by an allele $Cp^{Th}$, the product of which migrates with a mobility similar but not identical to that of $Cp^{A}$.

The common phenotype, CpB, represent homozygosity for the gene, $Cp^{B}$, which has a frequency of about 94 percent in Negroes and



Fig.2.2.5.a. Ceruloplasmin patterns of (from left to right) CPB, CPAB, CPB, CPAB, CPAC, and CPBC sera after electrophoresis on starch gel and staining with O-dianizidine. The anode is at the top(Shokeir, M.H.K., and Shreffler,D.C. 1973).

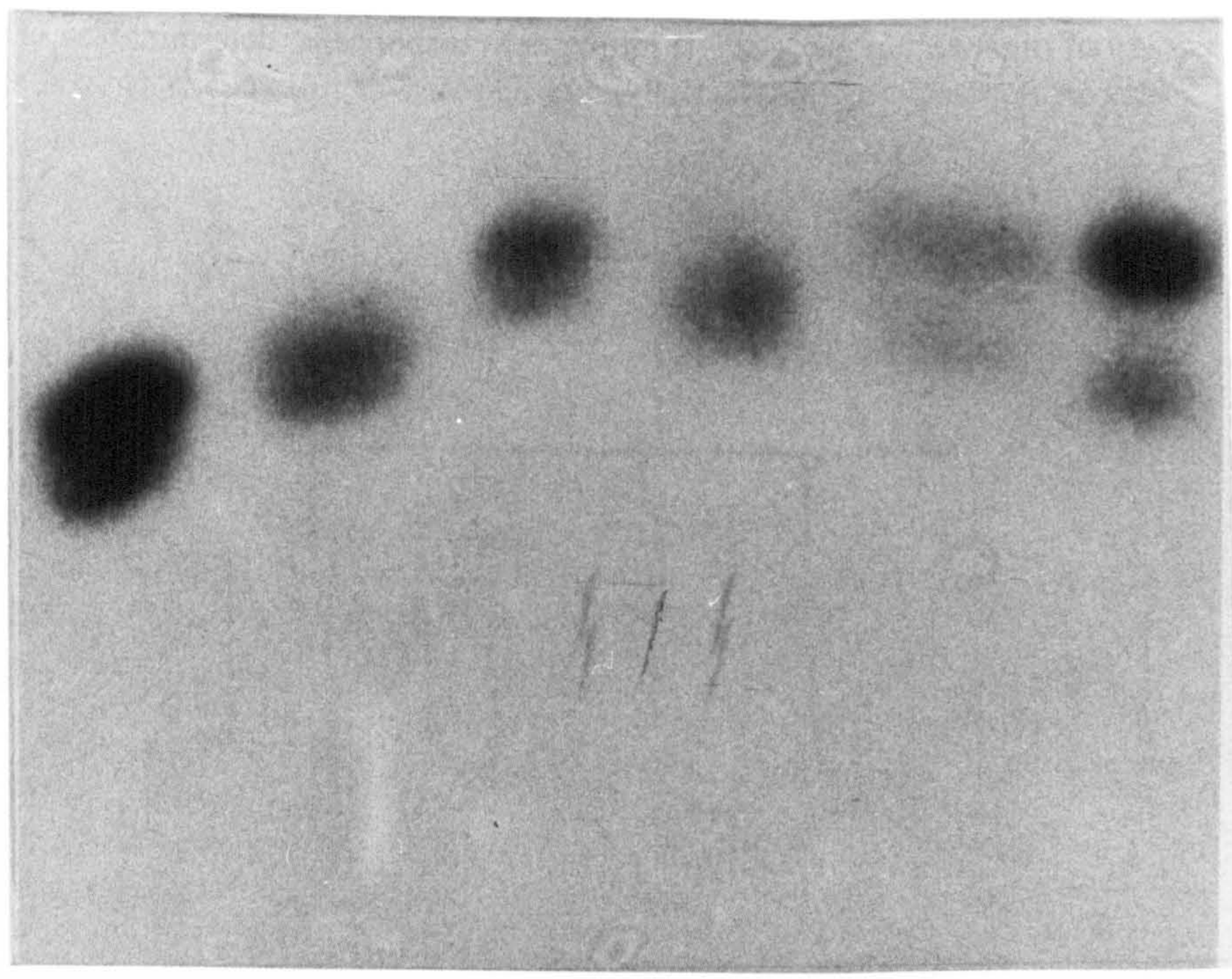


Fig.2.2.5.b. Ceruloplasmin patterns of (from left to right) CPBC, CPBNH, CPAB, CPBNH, CPANH, and CPAC sera after electrophoresis on starch gel and staining with O-dianizidine.The anode is at the top (Shokeir,M.H.K., and Shreffler, D.C. 1973).

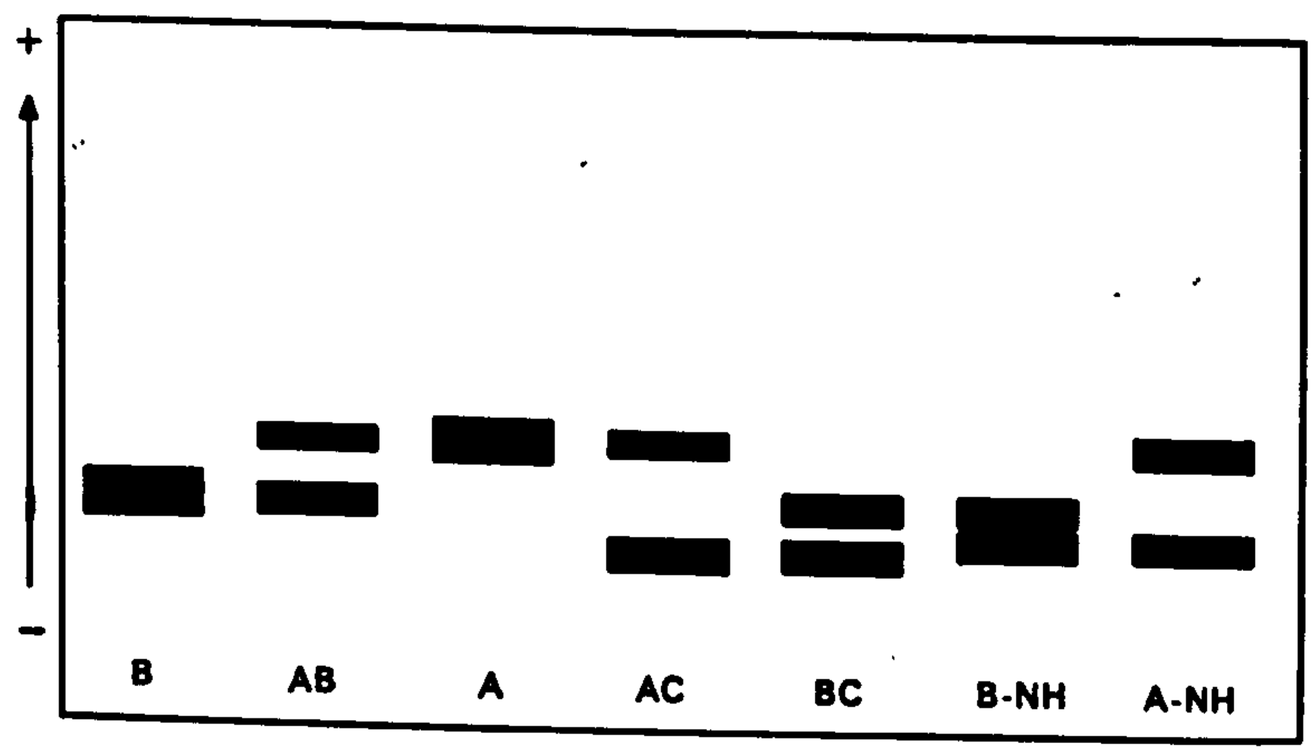



Fig.2.2.5.C. Diagram of the starch gel electrophoretic patterns of the seven ceruloplasmin phenotypes described by Shreffler et al. (1967) and Shokeir et al.(1967).The rare CP phenotype B-NH is very difficult to differentiate from the common CPB phenotype(Giblett, E.R.1969).

over 99 percent in non-Negroes tested. The $Cp^{A}$ gene, with a frequency of about 5 percent in Negroes, was also found in the homozygous state, as designated by the phenotype CpA.The genes $Cp^{C}$ and $Cp^{NH}$ have very low frequencies, estimated as 0.4 percent and their gene products have been seen only in combination with $Cp^{B}$ and $Cp^{A}$.

In all populations hitherto tested $Cp^{B}$ is of universal distribution and very high frequencies, whereas the alleles $Cp^{A}$, $Cp^{C}$, $Cp^{NH}$ and $Cp^{Th}$ show much lower frequencies. An exception seems to be $Cp^{A}$, because its frequency ranges between 0.04 and 0.15 in Negroes, it was therefore suggested that it can be used as a genetic marker indicating Negroid influence(Giblett, 1969). However, some remarkable racial differences seems to exist in this system. Though the ceruloplasmin polymorphism has been well known for several years, there are, in contrast to other recently detected polymorphism, surprisingly few publications on its allele distribution in different ethnic groups. The frequency of variants in all hitherto tested populations except the Negroes is very low.

The ceruloplasmins are probably of considerable importance in mineral metabolism, and it may be that certain alleles are selectively favoured in conditions of copper deficiency in the environment. However, it seems that it is too early to make general statements about its importance or non-importance for population genetical studies.

### 2.2.6 The $\alpha_1$-antitrypsin ( $\alpha_1$AT=pi) system

The term 'protease inhibitor' is applied to a group of serum proteins which have the property of inhibiting the activity of proteolytic enzymes such as trypsin, plasmin and thrombin. The protease inhibitors which have been most intensively

investigated (Ganrot, 1967) are in the $\alpha_1$ and $\alpha_2$ fractions. Human plasma contains several protease inhibitors both in the $\alpha_1$- globulin and the $\alpha_2$-globulin fractions, such as $\alpha_1$AT, $\alpha_2$ macroglobulin, $\alpha$ -anti-chymotrypsin and inter- $\alpha$ -tripsin inhibitor.

Only the $\alpha_1$ variety has been found to exhibit electrophoretic polymorphism, and it is the main trypsin inhibitor present in plasma and serum (Schultze et al, 1962). The protease inhibitor effect of $\alpha_1$-antitrypsin is due to the formation of an inactive complex with the proteolytic enzyme (Rimon et al, 1966.).

The history of investigations into its genetics has been summarized by Giblett (1969). Among the protease inhibitors present in human serum, $\alpha_1$AT is of special interest as it exists in high concentration, has approximately 90 percent of the serum's trypsin inhibitory capacity, and is active against other proteolytic enzymes, including chymotrypsin, elastase, collagenase, plasmin, thrombin and protease from human leukocytes. This protein shows a high degree of genetically determined heterogeneity; at least 25 alleles already have been identified. Nearly one half of the individuals in European and United States population samples are heterozygous for this system (Kueppers and Christopherson, 1978). There are, however, substantial differences in gene frequencies among human populations. When only the types detected by starch gel electrophoresis were considered, Asian populations were apparently more homozygous or almost monomorphic in the pi system(Ashley et al, 1980). With the availability of 'subtyping' by isoelectric focusing it is of interest to examine these groups for heterogeneity.

Genetic variants with severe deficiency can predispose to disease, as to pulmonary emphysema (Laurell and Eriksson, 1963), or be associated with Cirrhosis (Sharp, 1970). This deficiency was found to be hereditary and to form only part of a complex polymorphism at the chromosome locus which has been called pi. Famuly studies showed that the deficient state represents homozygosity for a mutant gene, and that heterozygotes have about half normal activity (Eriksson,1964, 1965; Kueppers et al, 1964; Lopez et al, 1964; Talamo et al, 1966; Ganrot et al, 1967).

The existence of other $\alpha_1$-antitrypsin variants in serum was first reported by Eriksson and Laurrell (1963). They described a slow moving variant subsequently shown to be inherited as an autosomal codominant, and its gene was found to be allelic to the gene determining the deficient variant (Axelsson and Laurell, 1965). Fagerhol and Braend (1965) reported that when human serum is subjected to starch gel electrophoresis, several bands with variable patterns in the prealbumin region, appear. These prealbumin zones were recognized $\alpha_1$-antitrypsin by Kueppears and Bearn (1966),Fagerhol and Braend (1966) and Fagerhol and Laurell (1967). The first three phenotypes described are the common type, called MM, a rare type, FF, and the ZZ type, which designates $\alpha_1$-antitrypsin deficiency. These three types represent homozygosity for the common gene, $Pi^M$ , and the rare genes $Pi^F$ and $Pi^Z$, respectively. The product of $Pi^F$ gene has faster mobility and that of $Pi^Z$ gene slower mobility than that of the common $Pi^M$ gene. (Figure 2.2.6.). Among twenty five alleles now known, all of them but one $Pi^M$, are rare in nearly all populations. The commonest allele, $Pi^M$ has a frequency of at

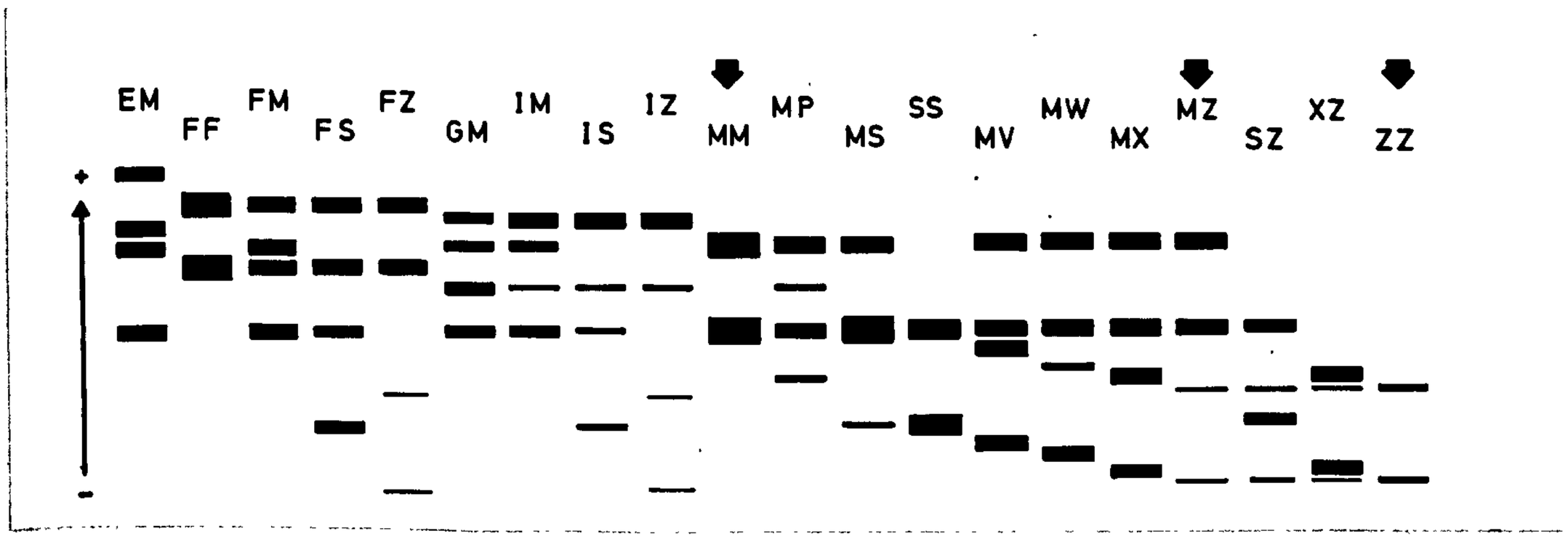



Fig. 2.2.6. Diagram of twenty Pi phenotypes, migrating ahead of albumin on starch gel electrophoresis at acid PH.(Fagerhol,M.K.1972).

least 85 percent in all populations tested.

The product of the gene $Pi^S$ has somewhat reduced activity, but two alleles, $Pi^Z$ and the very rare $Pi^-$ (or $Pi^0$) are responsible, in the homozygous state, for most of the severe deficiencies causing pulmonary disease. The $Pi^Z$ gene product has a small but measurable protease inhibitor activity, about 16 percent of that of $Pi^M$ (Cook, 1975), and the protein itself gives the normal precipitin reaction of the $\alpha_1$ protease inhibitors. The plasma of $Pi^-$ homozygotes gives no detectable protease inhibitor activity and no precipitin reaction, so that it may represent a gene deletion. Most of the electrophoretically abnormal variants are present in normal concentration in serum and show unimpaired inhibitory capacity. $\alpha_1$AT levels can be depressed, however, in case of the $Pi^Z$, $Pi^S$, $Pi^{WI}$, $Pi^P$, and $Pi^{null}$ alleles( Fagerhol and Tenfjord, 1968; Martin et al, 1975). Recently Fineman et al (1976) found a significantly increased frequency of Pi heterozygotes in Down syndrome patients whose mothers were more than 35 years old at the time of the patient's birth; whereas the frequency in patients whose mothers were less than 35 years old was not significantly different from that in the general population. These data suggest that $Pi^-$ phenotypes which comport a decreased $\alpha_1$AT activity may in some way predispose to chromosomal errors.

In view of the known pathological association of one of the alleles and the marked variations in frequency of the electrophoretically detectable alleles, this system merits further application both anthropologically and in disease surveys.

## 2.2.7. The Pseudocholinesterase system

Red cells contain a cholinesterase ( in official notation, acetylcholine acetylhydrolase, EC 3.1.1.7 ) which has the essential function of breaking down the acetylcholine produced at nerve endings of various kinds, especially those controlling voluntary muscles. It thus prevents the muscles from going into permanent spasm. Another cholinesterase, now commonly called pseudocholinesterase, and officially as acylcholine acylhydrolase, EC 3.1.1.8, is present in the plasma; its normal function is uncertain, but the existence of genetically determined variations in its activity came to light as a result of using another acylated choline compound, succinylcholine, as a muscle relaxant in surgical anaesthesia. This substance blocks the action of acetylcholine on voluntary muscles and so produces relaxation. Its effect is usually moderated by, and after some minutes most of it is broken down by,the action of pseudocholinesterase,so that the tone of muscles,including those of respiration, returns to normal. From time to time, however, individuals have been found who remain relaxed and without spontaneous respiration for prolonged periods.This has been found to be due to a deficiency of plasmatic pseudocholinesterase activity.Sometimes it could be explained as a secondary effect of the disease from which the patient was suffering,but it was liable also to occur in persons whose general health was excellent.In 1953,Forbat et al found one patient with a deficiency of the enzyme who had a healthy brother

with a similar deficiency.This group of workers then, and several others at about the same time (Harris, H., and Whittaker, M. 1962 and Kalow, W. 1959) began to look for further examples of inherited variants of the enzyme, and several came to light differentiated by their normal activity and its percentage inhibition in vitro under specified conditions by fluoride ions and by dibucaine. As a result of quantitative tests for enzyme activity with and without these inhibitors, and a further inhibitor which is a neostigmine analogue with the code name Ro2-0863, together with starch-gel electrophoresis and staining for enzyme activity, it has been possible to account for the various phenotypes in terms of the products of four allelic genes, the normal $E_1^u$, $E_1^a$ which gives prdoducts with a reduced activity but relatively resistant to dibucaine inhibition, $E_1^f$ whose products are relatively resistant to inhibition by fluoride, and $E_1^s$, the homozygote of which is almost totally inactive. $E_1^s$ is, however, very much rarer than $E_1^a$, and it is the $E_1^a$ homozygote which is the usual cause of trouble following relaxation anaesthesia.

Despite the rarity of variants, their clinical importance and biochemical interest have led to the performance of numerous population surveys. Some of these, depending upon inhibition with dibucaine, have distinguished only the genotypes containing $E_1^a$, and the very rare homozygote $E_1^s$ $E_1^s$ if present. Others, using also inhibition with fluoride have distinguished types containing $E_1^f$ as well. The genetics and serology have been fully reviewed by Giblett (1969).

Besides the immediate products of the genes at the $E_1$ locus, some sera contain another similar esterase which is partly independent genetically, known as C5. This has been

described in a series of papers by Harris et al (1963). It is most easily demonstrated by electrophoresis in a rather acid gel, when all the bands directly due to $E_1$ alleles come together, and that due to C5 moves slightly more slowly towards the positive pole.

To a first approximation this enzyme behaves as the product of a gene at a locus independent of $E_1$, and known as $E_2$, with dominant expression. However, it appears not always to be expressed in the heterozygote, and the expression is also affected by the $E_1$ genotype. This is, therefore, not a very useful genetic marker.

## 2.3. Red cell enzymes

Enzymes have long been known as proteins which catalyse biochemical reactions, the name 'enzyme' having been proposed by Kuhne in 1878.

The concept of specific enzymes as products of single genes is inherent in Garrod's paper in 1902 on alkaptonuria as well as in his later classical work, Inborn Errors of Metabolism(1909). The concept was, however, given much more concrete expression by Beadle and Tatum (1941) in their 'One gene, one enzyme ' hypothesis of 1941.

Human genetics, however, had until recently concerned itself mainly with hereditary diseases and abnormalities, and the work of Krebs and others on the enzymatic control of normal metabolism was not at first seen as having any important relation to genetics.

Interest in variants of the normal proteins of the body was greately stimulated by Pauling's discovery in 1949 of sickle-cell haemoglobin, Smithies's discovery of genetic variants of haptoglobin (1962), and Lehmann and Ryan's demonstration of

genetic variants of pseudocholinesterase (1959); but the genetic study of variants of normal enzymes owes most to the work of Professor Harry Harris and his colleagues. Some enzymes showing polymorphism are present in the plasma, but a much greater variety of them is to be found inside the red cell.

Many enzymes of which genetic variants (isozymes) have been studied are involved in various cycles of normal glucose metabolism. In order to understand the methods used in testing for the enzyme variants it is necessary to have some understanding of these normal cycles and of the function of the enzymes in them. The isozymes have hitherto been treated mainly as formal genetic markers in family studies involving such phenomena as linkage, in which their precise functions are of secondary importance.

However, the wide differences in frequency of certain isozymes in different populations is almost certainly due largely to natural selection.

A knowledge of quantitative and qualitative functional differences between the genetic variants of a given enzyme is thus an essential part of the study, as yet scarcely begun, of the evolutionary mechanisms involved in the interaction between human populations and their physiological and microbiologicalenvironment.

Inside the boundary membrane of the red cell is a solution, possibly with an elaborate microscopic structure, containing haemoglobin and a variety of other substances, especially enzymes. Haemoglobin and a great many of the enzymes show genetical polymorphism. The enzymes have, of course, precisely known functions and in some cases the products of al-

lelic genes differ quantitatively in functional activity. For each offa considerable number of the enzymes there is at least one allele conferring low or absent activity, and homozygotes for such alleles may suffer from distinct congenital diseases.

The physical anthropologist is not directly interested in the quantitative substrate activity of particular enzymes, but only with their multiple molecular forms. These multiple molecular enzymes were termed isoenzymes by Markert and Moller (1959). The application of standard histochemical staining techniques to starch gels has enabled a large number of enzymes to be visualised, the resulting pattern being known as a zymogram.

### 2.3.1. The acid phosphatase (AcP ) system

Human erythrocyte acid phosphatase (orthophosphate monoester phosphohydrolase, EC 2.1.3.2) was first shown to be genetically polymorphic by Hopkinson et al (1963), by means of starch-gel electrophoresis and appropriate staining. It catalyses the reaction involving the transfer of the phosphate ion from its ester to the inorganic state or to suitable acceptor substances. The precise function of this enzyme in the red cell is unknown. Five distinct electrophoretic patterns, designated A, BA, B, CA and CB, were described by Hopkinson, Spencer and Harris (1963 and 1964). Family studies indicate that the different types are controlled by three co-dominant alleles, $p^a$, $p^b$ and $p^c$, at an autosomal locus. Phenotypes A, B and C represent homozygous genotypes $p^a p^a$, $p^b p^b$ , $p^c p^c$ respectively, and phenotypes BA, CA and CB represent heterozygous genotypes $p^a p^b$ , $p^a p^c$ and $p^b p^c$ . They have shown that the five different types were controlled by

three autosomal allelic genes $p^a$, $p^b$, and $p^c$. According to the three allele hypothesis, the BA, CA and CB types represent the heterozygous combinations, and A and B are homozygotes. This hypothesis predicted the occurrence of a sixth phenotype C, which should be relatively rare. Lai et al (1964) presented confirmatory family data and the finding of the C phenotype. At about the same time Harris and his colleagues also found the C type.

Two alleles, $p^a$ and $p^b$, have been found to be polymorphic in all populations studied so far. The third allele, $p^c$, is polymorphic in some populations while totally absent in others. The relatively high frequency of $p^c$ in European populations led to it being regarded as a 'Caucasian' gene by Scott et al (1966) and its occurrence in other populations is thought to be due to Caucasoid admixture (Tashian et al, 1967).

Each homozygous phenotype consists of two electrophoretically distinct isozymes. The $AcP_1A$ phenotype has unique electrophoretic mobility, while $AcP_1$ types B and C are electromorphs, distinguished by differences in the relative intensity of the two isozymes. The erythrocyte enzyme is genetically and functionally distinct from the lysosomal $AcP_1$s of other tissues, although these tissues also contain the erythrocyte enzyme.

Figure 2.3.1.a shows that each type consists of two isozymes. The isozyme of type A exhibits two bands with about equal intensity and migrates faster than the other homozygous types. Type B has also two bands, but the faster band is more intense than the slow band. The mobility of the B band is slightly faster than the A band. The C type has two bands corresponding to the ones found in the B and CB types but the fast band is

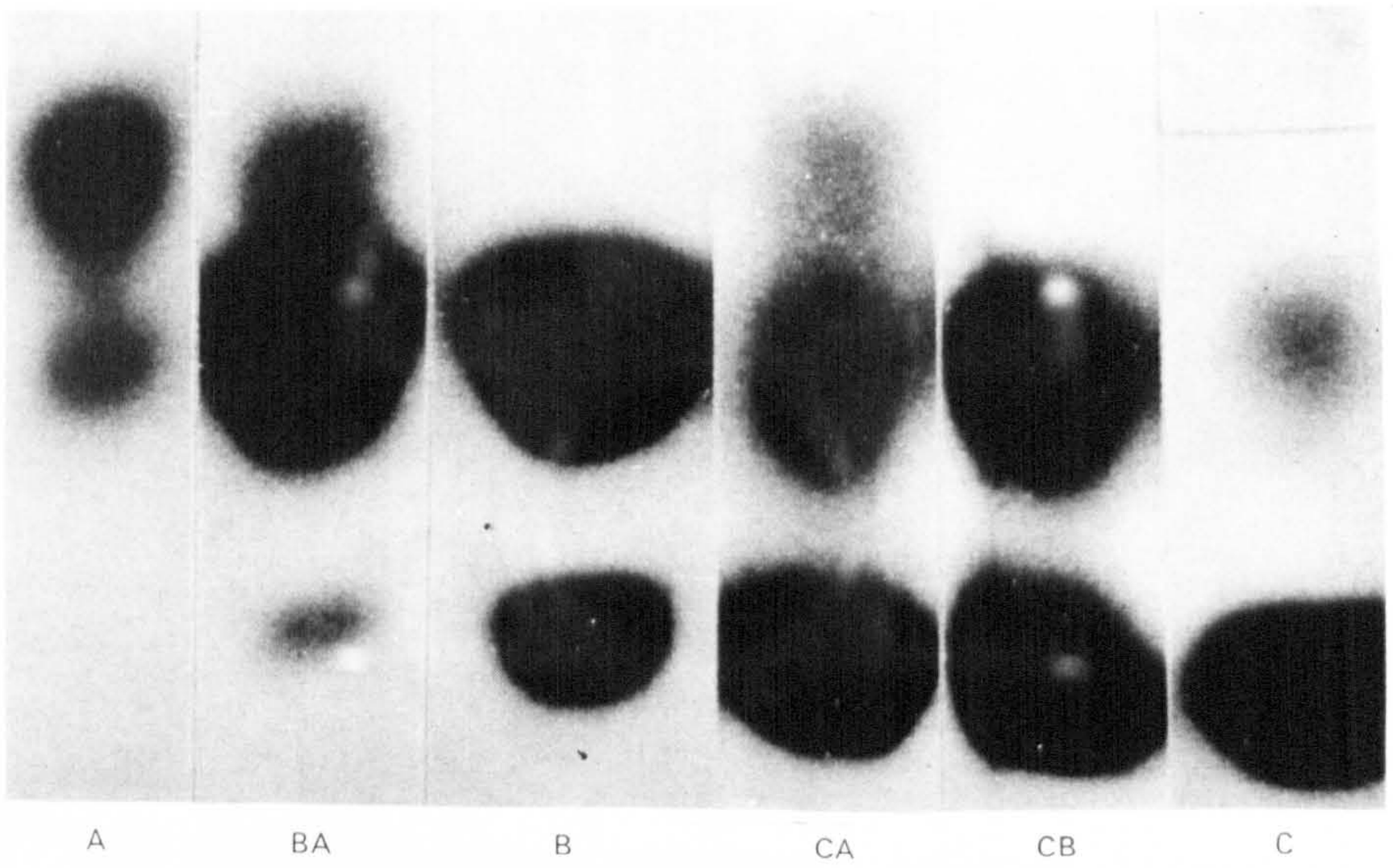



Fig.2.3.1.a. Electrophoretic patterns of red cell haemolysates with the six phenotypes representing homozygosity and heterozygosity for the three genes at the acid phosphatase locus(Giblett,E. R. 1969).

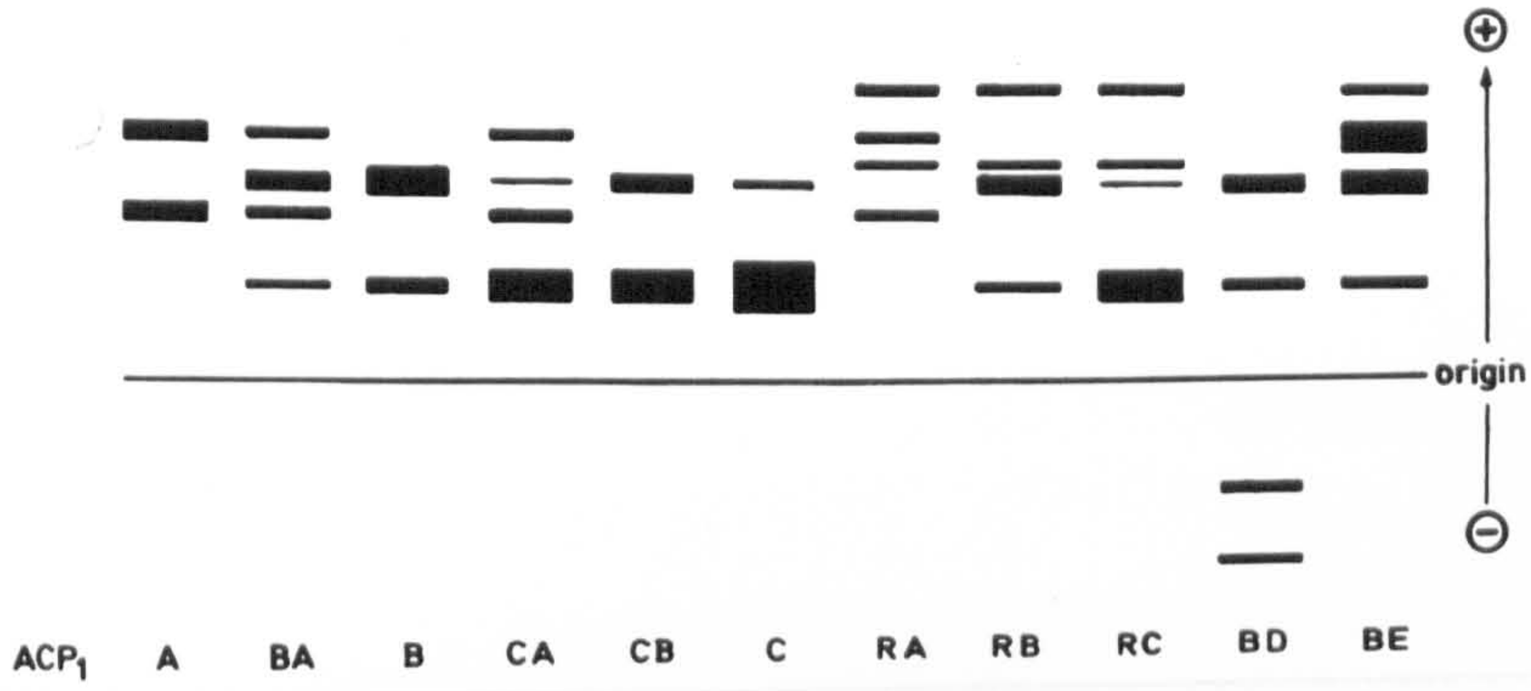



Fig.2.3.1.b. Diagram showing AcP isozymes in red cells from different phenotypes.Note that each of the alleles appears to determine two isozymes (Hopkinson,D.A., and Harris,H. 1969).

very faint and the slow band has a very high activity. The pattern in type BA, CA and CB, appears to be a mixture of the isozymes of their corresponding homozygous types. Type BA shows a complex pattern that is a combination of the components seen in the A and B types. The BA type can be reproduced by the electrophoresis of a mixture of haemolysates of type A and B in equal amounts. The part of the BA pattern that corresponds to the fast A components does not separate clearly, however, but appears as a smear. Both type CA and CB show a very intense slow band. Type CA has, in addition, two fast bands similar to the ones seen in type A. In type CB the intense slow band is associated with a faster band similar to the main band of the B type. In addition to the common types, two rare phenotypes, RA and RB, were reported by Giblett and Scott (1965) and Giblett(1967). The isozyme of type RA is characterized by a pair of fast moving components with either A or B or C zones. Type RB consists of two bands which migrate faster towards the anode.$P^r$is therefore suggested as a fourth allele and was confirmed by Karp and Sutton (1967).It determines approximately the same amount of AcP as does the $P^a$allele,and is allelic to $P^a$ and $P^b$( Jenkins and Corfield, 1972 ). The detection of another rare phenotype BD pointed to the existence of a fifth allele, $P^d$. Type BD has the two most cathodal bands which are not observed in other types. Lamm( 1970) described DA and DB variants in a Danish family and found the $P^d$ allele segregating at the AcP locus ( Figure 2.3.1.b). The occurrence of a rare $AcP_1^0$ allele has been described in two Australian ( Herbich et al, 1970; Herbich and Meinhart, 1972) and several German families (Brinkmann, 1974), and in one Danish family (Dissing and Svensmark, 1976) and one fa-

tribution of AcP alleles indicated that some environmentally induced selective pressures are at work.The earliest observations on the possible adaptive value of the AcP polymorphism are attributed to Ananthakrishnan and Walter(1972),who discovered that the $P^a$allele frequencies are negatively and significantly correlated with temperature;they thus reached the conclusion that the $P^b$allele would be advantageous for populations living in hot climates.They found a marked gradient in the world distribution of the AcP alleles.The frequency of the $P^b$allele rises with the increase in mean annual temperature of the various biotopes whereas $P^a$ shows a decrease.Even in a relatively small area,West Germany,they found a significant negative correlation between mean annual temperature and the frequency of the $P^a$ allele.

Jenkins and Corfield (1972) speculated that selective forces could be acting against the $P^c$ allele which carried the highest activity thus accounting for the current low $P^c$ frequencies or, alternatively, that the $P^c$ allele could be a relatively recent mutation which is advantageous and so is gradually increasing in frequency.

As regards the $P^c$ allele distribution, Bottini et al (1972)and Palmarino et al (1975) hypothesised that this allele has some adaptive relevance concerning malaria, since its frequency is correlated positively with altitude and negatively with malaria morbidity in Sardinia in the past. There is some evidence from New Guinea of a negative association between the AB phenotype and the presence of malarial parasites (Harrison et al, 1976). The hypothesis formulated by Jenkins and Corfield(1972) postulated that in sub-Saharan Africa there could be a connection between the distribution of the $P^r$allele and distribution of various cultures,since this allele is more frequent

among the herder and hunter-gatherer peoples than among settled farming populations.

Observing that this latter distribution is closely associated with the geographical environment, Spedini et al (1978) surmised that the $p^r$ allele could be negatively correlated with malaria, as appears to be the case with the $p^c$ allele in the Mediterranean peoples subject to malaria. They have therefore attempted to ascertain whether the $p^r$ allele, known as "Negro allele" is correlated with certain climatic factors more directly responsible for the incidence of malarial morbidity. The wide range of gene frequencies, and the quantitatively different activities of the gene products, make this system a most promissing one for studies of ecology and possible natural selection.

2.3.2. The adenylate kinase (AK) system

The enzyme adenylate kinase (AK) or ATp: AMP phospho-transferase (EC 2.7.4.3.) reversibly catalyses the reaction: Adenosine diphosphate $\rightleftharpoons$ Adenosine triphosphate + Adenosine monophosphate ($2ADP \rightleftharpoons ATP + AMP$)
within the red cell and in muscle and other tissues. Its molecule is extremely stable, which renders it highly suitable for investigation in far-travelled blood specimens.

The polymorphism of the human red cell adenylate kinase was first described by Filds and Harris (1966), by means of starch-gel electrophoresis. Population and family studies indicate that the variants o AK are genetically determined by two co-dominant autosomal alleles, called $AK^1$ and $AK^2$ . Individuals homozygous for the alleles are phenotypes AK1-1 and AK2-2 respectively, and heterozygous persons possessing bothe $AK^1$ and $AK^2$ genes have the AK2-1 phenotype. Three dif-

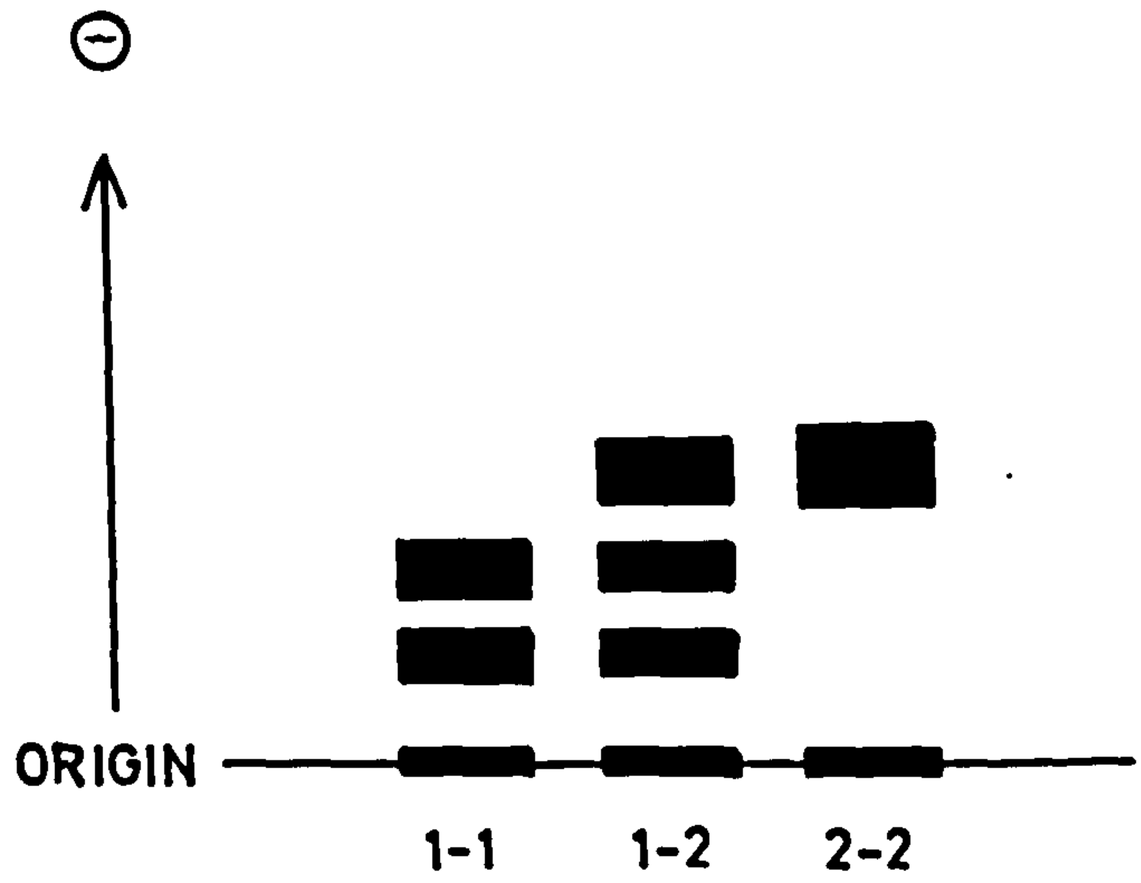



Fig. 2.3.2.a. Diagram of the three common AK phenotypes AK1-1, AK2-1 and AK2-2. Electrophoresis at PH 5.0.

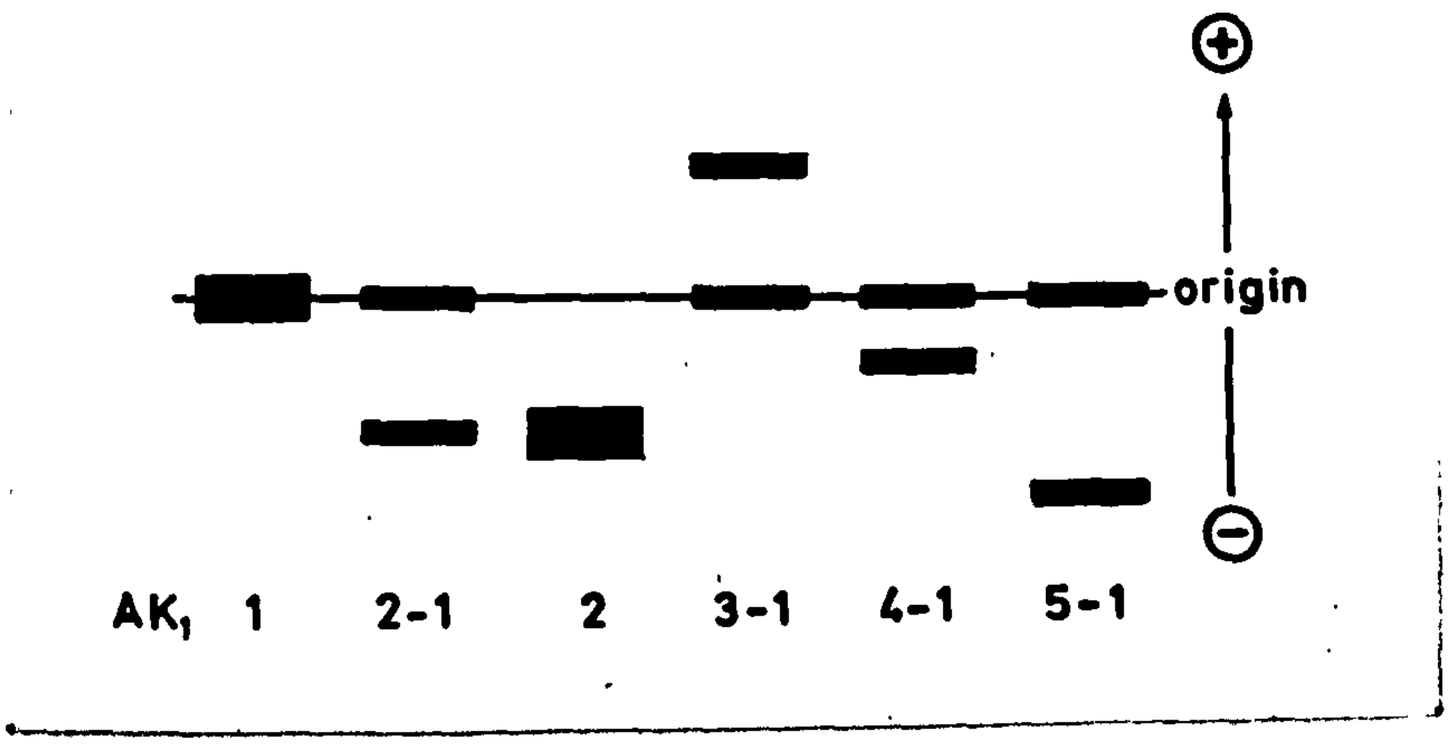



Fig. 2.3.2.b. Diagram of various AK phenotypes. Only the main isozymes are shown. Electrophoresis at PH 7.0(Harris,H., and Hopkinson,D.A. 1976).

ferent isozyme patterns referred to as AK1-1, AK2-1 and AK2-2 are shown in Figure 2.3.2. a. The most common phenotype, AK1-1 consists of two major components, of which the most intense remains to the fastest, and the less active zone migrates slower, a third slower-moving component stains poorly and tends to be diffuse. The AK2-1 pattern consists of the same three components,but with relatively less activity, and in addition a fourth zone which moves faster. The pattern seen in AK2-1 represents a mixture of isozymes present in AK1-1 and AK2-2. The AK2-2 pattern consists of one fast component which stains strongly. In addition to the common types, further variants due to the very rare genes, $AK^3$, $AK^4$ and $AK^5$ have been described (Bowman et al 1967, Rapley et al 1967, Benercetti et al 1972). Individuals with such aberrant phenotypes are heterozygotes for the usual allele $AK^1$ and the rare allele $AK^3$, $AK^4$ and $AK^5$ ( Figure 2.3.2.b.).

The only alleles of any substantial importance in population studies are $AK^1$ and $AK^2$, the $AK^2$ gene being by far the rarer of the two with a frequency of only about 5 percent. With the large differences in adenylate kinase frequencies found in different parts of Asia, the system may prove of considerable value in tracing population relationship in this continent.

## 2.3.3. The phosphoglucomutase (PGM) system

The enzyme phosphoglucomutase $PGM_1$ ( $\alpha$-D-glucose 1.6. diphosphate: $\alpha$-D-glucose-1-phosphate phosphotransferase, EC 2.7.5.1) reversibly catalyses the transfer of a phosphate group between the 1 and 6 positions of glucose. It occurs in the red cells and several other human tissues (Hopkinson & Harris, 1965, 1966; Renninger, 1969; McAlpine et al, 1970 ) .

The existence of multiple isozymes of human PGM detectable by starch-gel electrophoersis, and inherited variation in this enzyme were demonstrated by Spencer et al (1964). Since then, three independent loci, $PGM_1$, $PGM_2$ and $PGM_3$ have been indentified ( Spencer et al, 1964; Hopkinson and Harris, 1965, 1966, 1968), and assigned to chromosomes 1, 4 and 6 respectively (Nguyen et al, 1971; Douglas et al, 1973; McAlpine et al, 1975; Jongsma et al, 1973). In most tissues the isozymes of $PGM_1$ predominate, exhibiting about 90 percent of the total PGM activity; the residual activity is yielded mainly by $PGM_2$ and to a slight extent by $PGM_3$ (Harris and Hopkinson, 1976). In the red cells, however, only $PGM_1$ and $PGM_2$ isozymes are expressed, and they display nearly equal activities. This means that erythrocytes have a smaller amount of $PGM_1$, but a correspondingly higher content of $PGM_2$, approximately 50 percent (McAlpine et al, 1970). The $PGM_3$ contribution is relatively small in all tissues and apparently not detectable in erythrocytes at all (Harris and Hopkinson, 1976; Quick et al,1974). Bissbort et al (1975), however, were able to demonstrate the polymorphism of $PGM_3$ in erythrocyte haemolysates by means of horizontal starch-gel electrophoresis. The three common phenotypes in whites, detectable on PGM-stained starch gels are expressions of two common alleles at $PGM_1$ ($PGM_1^1$ and $PGM_1^2$) and analogously at $PGM_3$ ( $PGM_3^1$ and $PGM_3^2$ ).

The $PGM_2$ isozymes can be characterized by a remarkable phosphoribomutase (PRM) activity (Quick et al, 1972), whereas $PGM_1$ and especially $PGM_3$ are not efficient in this reaction(Quick et al,1974). Due to low $PGM_3$ activities in all tissues, Harris(1975) suspected that its isozymes have another main catalytic activity, which is so far unrecognized.

The different loci of phosphoglucomutase probably arose from gene duplications during early vertebrate evolution (Engle and Schmidtke, 1975). Spencer et al (1964) demonstrated that when red cell haemolysates are subjected to starch -gel electrophoresis, seven different zones of PGM can be observed,labelled a, b, c, d, e, f and g from the cathodal end,which appear to be due to alleles at two distinct and not closely linked autosomal loci, $PGM_1$ and $PGM_2$. The $PGM_1$ locus determines the a, b, c and d bands while the $PGM_2$ locus determines the e, f and g bands. There are two common alleles at the $PGM_1$ locus, $PGM^1$ and $PGM^2$. Three common phenotypes are referred to as PGM1-1 PGM2-1 and PGM2-2. $PGM_1^1$ produces the a and c bands and its homozygote is called PGM1-1, while $PGM_1^2$ , gives rise to the bands b and d and its homozygote is type PGM2-2. The heterozygote PGM2-1 has all four bands; a, b, c and d which has the appearence of a mixture of PGM1 and PGM2. The three fast moving bands; e, f and g are found in all three phenotypes although the g band is usually weak or absent (Figure 2.3.3.a.) Family studies showed that the results could be explained by the segregation of two autosomal allelic genes, $PGM_1^1$ and $PGM_1^2$, at the $PGM_1$ locus. Thus, the phenotypes PGM1-1 and PGM 2-2 correspond to the homozygotes $PGM^1$ $PGM^1$ and $PGM^2$ $PGM^2$ respectively and phenotype PGM2-1 represents the heterozygote $PGM^1$ $PGM^2$. The isozymes a and c are controlled by the $PGM^1$ gene while b and d isozymes are products of the allelic gene $PGM^2$. Studies by Hopkinson and Harris (1965, 1966) indicate that in addition to the common alleles, $PGM_1^1$ and $PGM_1^2$ , there are at least six rare alleles at this locus. (Figures 2.3.3. b & c.). They are determined by six alleles designated as $PGM_1^3$ , $PGM_1^4$, $PGM_1^5$, $PGM_1^6$ , $PGM_1^7$, and $PGM_1^8$ ( Harris et al,1973; Fiedler

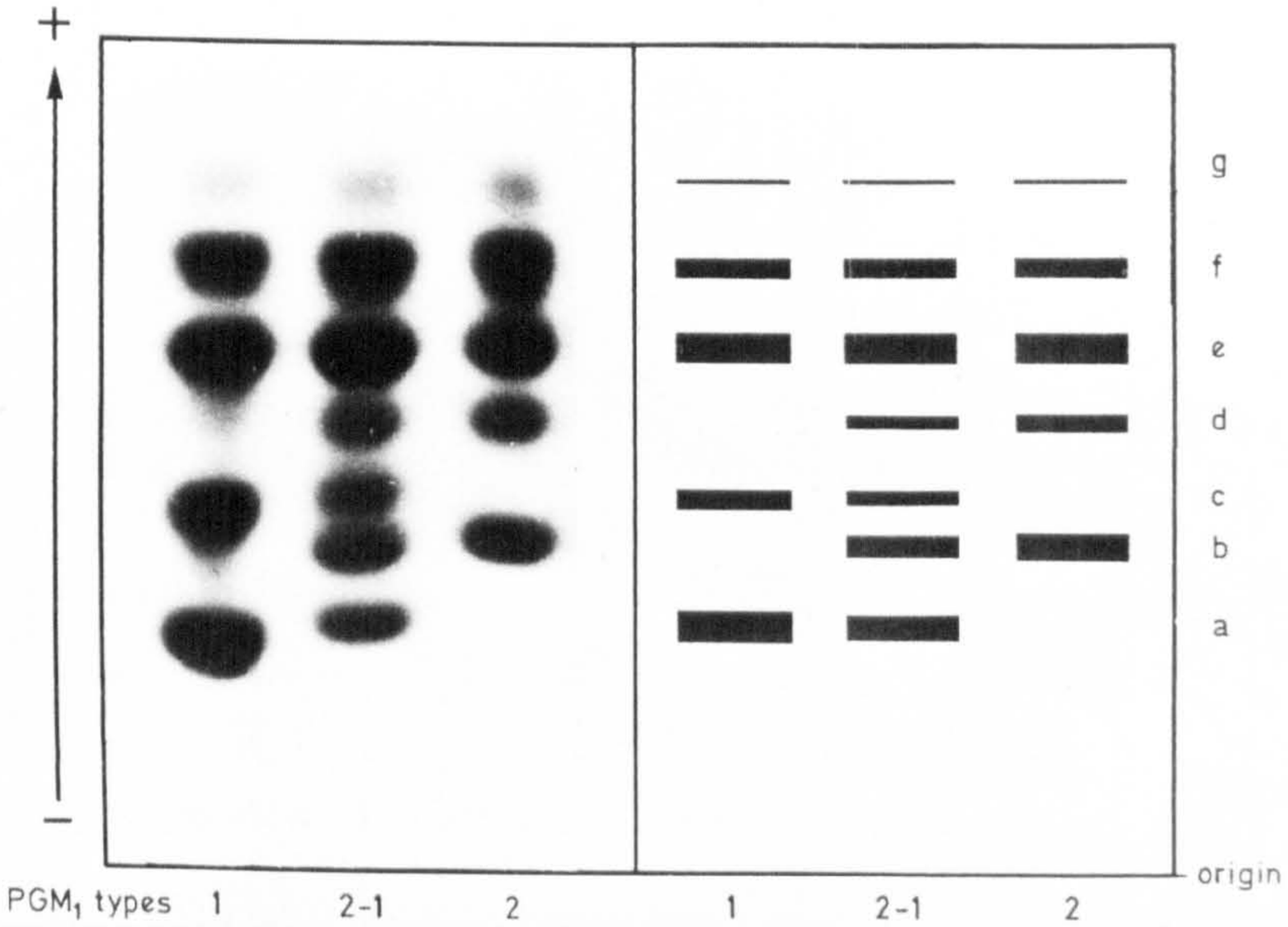



Fig.2.3.3.a. Red cell phosphoglucomutase isozymes in the three common phenotypes, $PGM_1$ 1, 2-1 and 2. Isozymes c and d are thought to be secondary isozymes derived from a and b respectively. Isozymes f and g are thought to be secondary isozymes derived from e ( Harris, H., and Hopkinson,D.A. 1976).

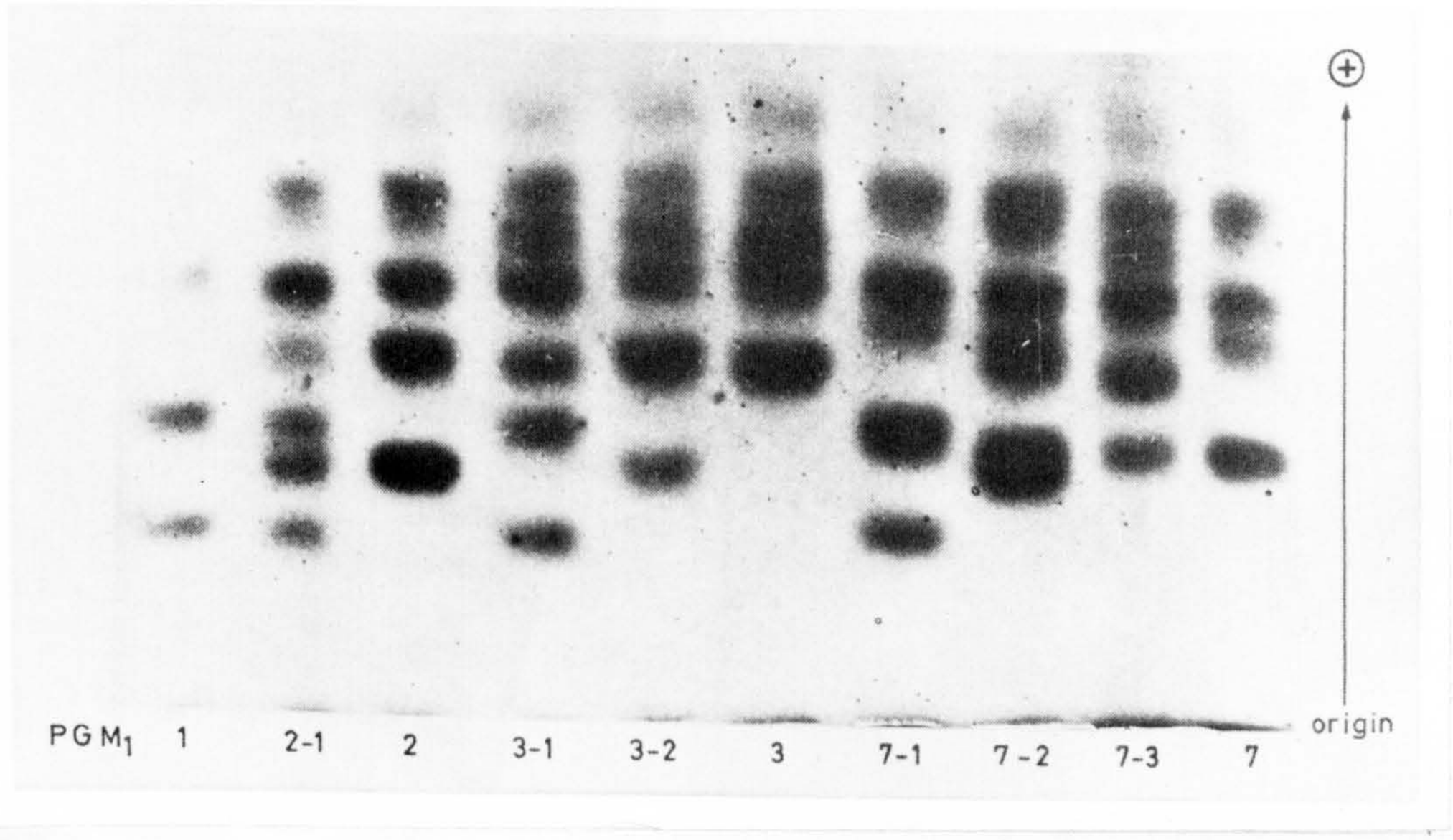
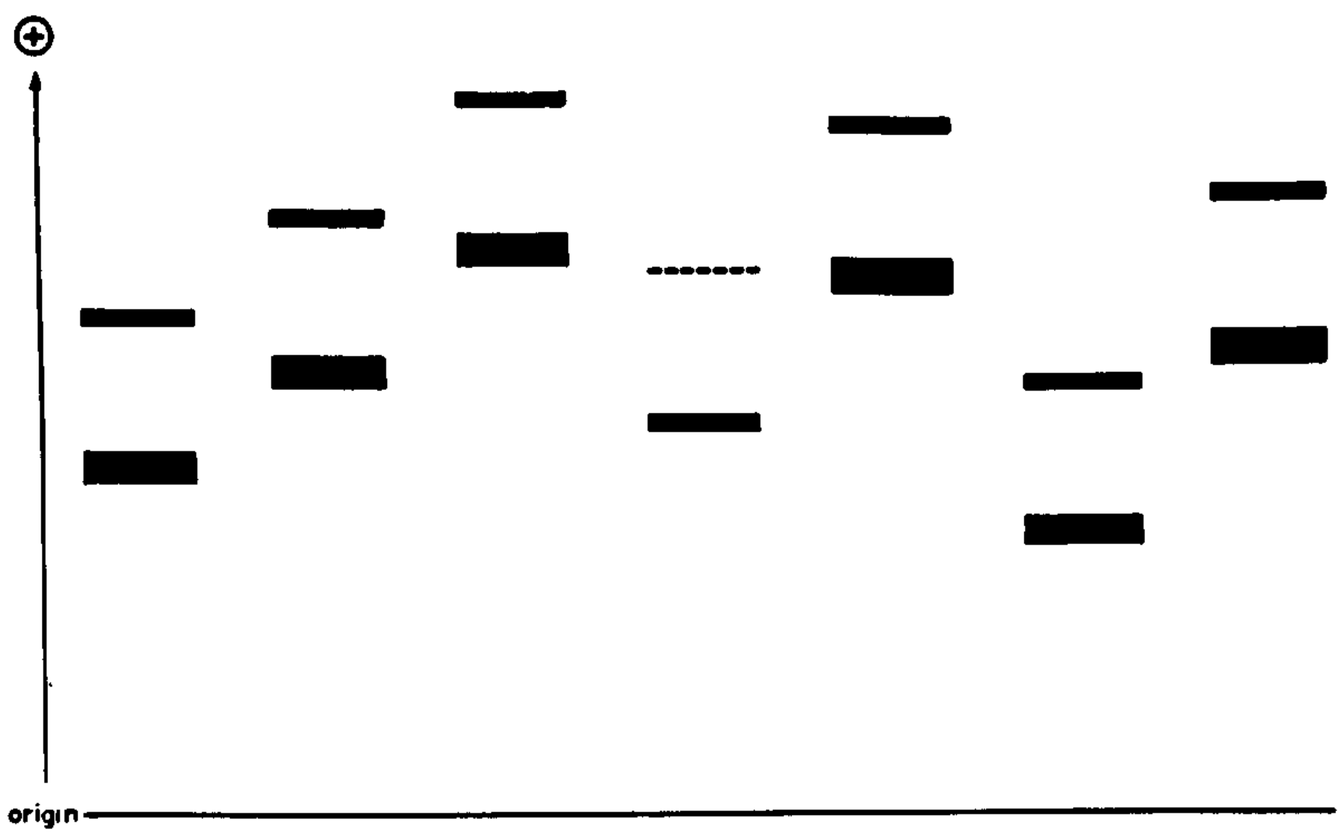



Fig.2.3.3.b. Red cell phosphoglucomutase isozymes in three common phenotypes $PGM_1$ 1, 2-1 and 2 and several rare $PGM_1$ phenotypes (Blake, N.M., and Omoto, K. 1975).




| Allele | $PGM_1^1$ | $PGM_1^2$ | $PGM_1^3$ | $PGM_1^4$ | $PGM_1^5$ | $PGM_1^6$ | $PGM_1^7$ |
|---|---|---|---|---|---|---|---|
| Number detected | 15,649 | 5,005 | 3 | 1 | 1 | 3 | 4 |
| Frequency | 0·757 | 0·242 | 0·00014 | 0·00005 | 0·00005 | 0·00014 | 0·00019 |

Fig.2.3.3.C. Diagram of the isozymes determined by seven different alleles at the $PGM_1$ locus. The isozyme patterns seen in heterozygotes appear to be simple mixtures of the products of the two alleles. The incidence of the different alleles found in a survey of 10333 unrelated individuals(20666 alleles)is given (Harris,H., and Hopkinson,D.A. 1976).

and Pettenkofer, 1969).

Most individuals are homozygous for the commonly occuring allele at the $PGM_2$ locus, $PGM_2^1$, which determines a set of bands, e, f and g, well separated from $PGM_1$ components. Variation in the $PGM_2$ locus have been found only in Negroes. So far nothing is known of the factors which maintain the genetic polymorphism at the $PGM_1$ locus. There does not seem to be any apparent differential superiority of one phenotype over the other with regard to enzyme activity.

In population studies the most useful system is $PGM_1$ with two common genes $PGM_1^1$ and $PGM_1^2$ varying widely in frequency in different populations, and at least six rare genes.
With a very few known exceptions, the frequency of $PGM_1^1$ always exceeds 50 percent. It is therefore convenient to describe populations in terms of the frequencies of the genes $PGM_1^2$, and of the rarer genes where they occur.

2.3.4. The adenosine deaminase (ADA) system

The enzyme adenosine deaminase (ADA; adenosine aminohydrolase E.C. 3.5.4.4.) acts as an aminohydrolase catalysing the deamination of adenosine into inosine. It was found in different tissues and in red blood cells. The enzyme is know to exist in multiple molecular forms.

Genetically determined variants of this enzyme were first demonstrated by means of starch-gel electrophoresis of red-cell haemolysates, by Spencer et al (1968). These were shown to be the products of a pair of allelic genes $ADA^1$ and $ADA^2$ at an outosomal locus.
They observed three different patterns. (Figure 2.3.4 a), ADA 1-1, ADA2-1 and ADA2-2, and family studies indicated the patterns to be genetically controlled by two co-dominant alleles at an autosomal locus, termed ADA1 and $ADA^2$. The phenotypes

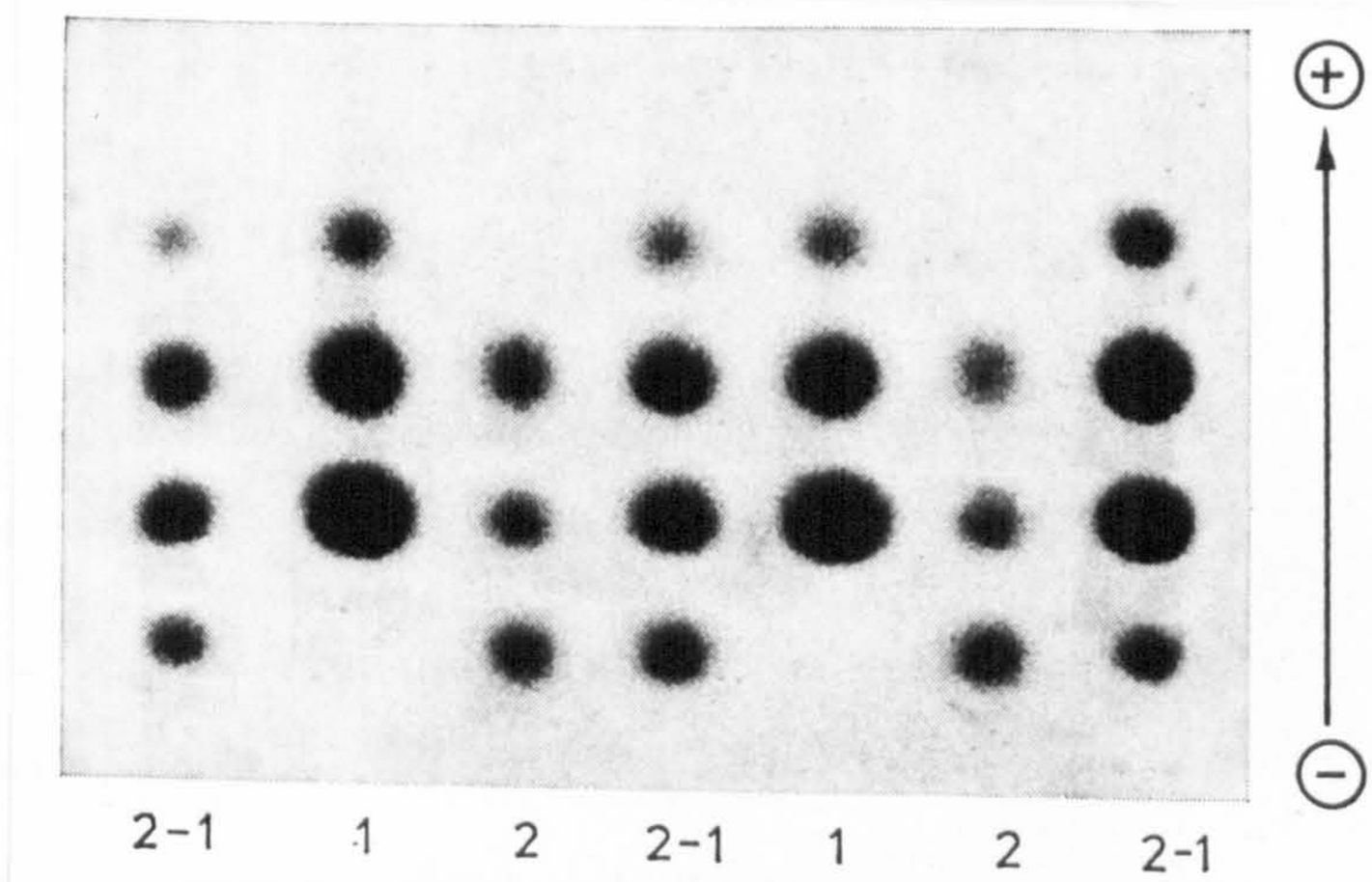



Fig. 2.3.4.a. ADA isozymes in red cells of the three common phenotypes ADA 1, 2-1 and 2.( Walter, H., and Ananthakrishnan,R.1975).

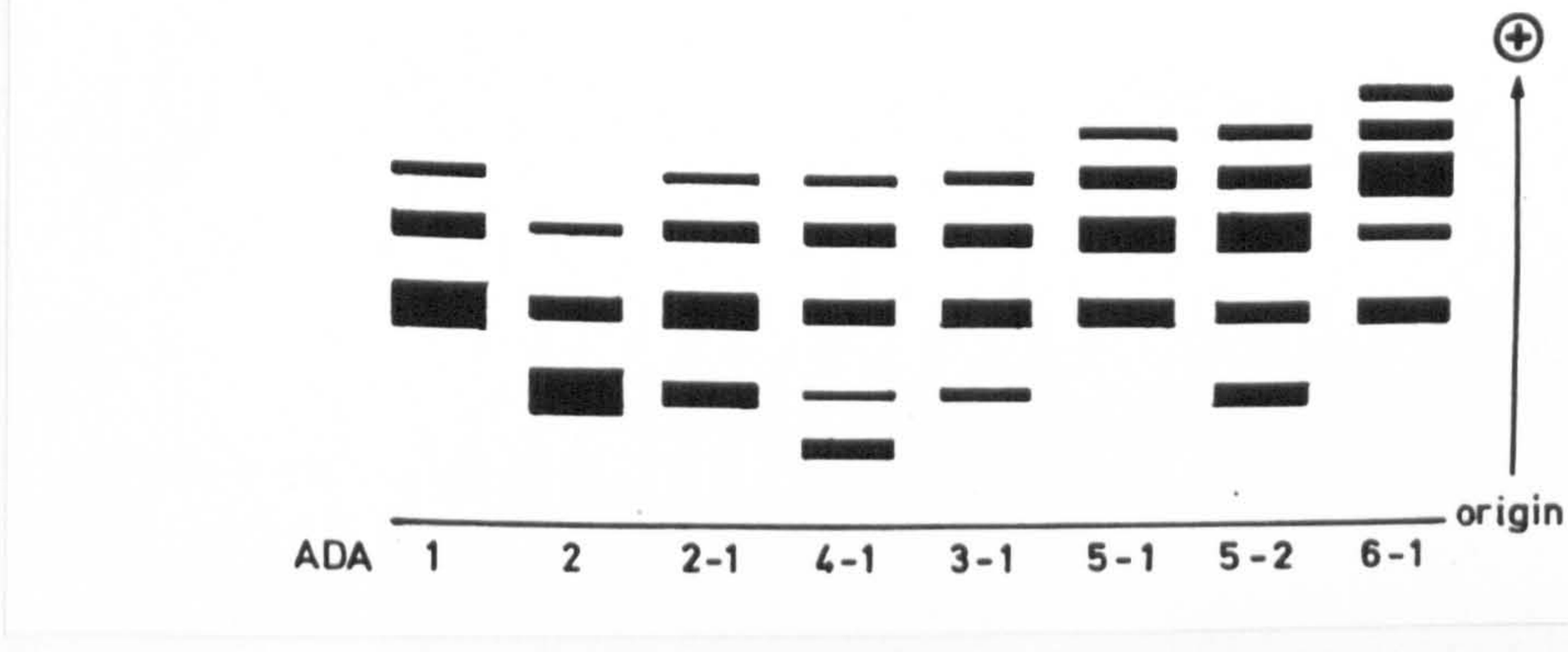



Fig.2.3.4.b. Diagram of the red cell ADA isozymes in various phenotypes (Radam, R., et al. 1974).

ADA1-1, ADA2-1 and ADA2-2 according to this hypothesis represent the genotypes $ADA^1/ADA^1$, $ADA^1/ADA^2$ and $ADA^2/ADA^2$ respectively. In a further paper Hopkinson, Cook and Harris (1969) described another allele $ADA^3$ at the same locus which, however, appears to be very rare, if present at all, in all populations. Since then several additional rare phenotypes have been observed (Figure 2.3.4.b.), which were attributed to the alleles $ADA^3$, $ADA^4$, $ADA^5$, $ADA^6$, and $ADA^8$( Dissing and Knudsen, 1970, and Detter et al, 1969). In addition, a 'silent' allele $ADA^0$ has been described (Chen et al, 1974; Giblett et al,1972) which appears to be associated with combined immunodeficiency. Adenosine deaminase is an importan enzyme in purine metabolism as evidenced by the occurrence of severe combined immunodeficiency disease in invidviduals deficient in leucocyte ADA activity( Giblett et al, 1972; Meuwissen, Pollara and Pickering,1975) and in one case elevated red cell ADA activity appears to be a cause of haemolytic anaemia (Valentine et al,1977).Since thediscovery ofgeneticallydétermined variants of red cell adenosine deaminase,it has been screened in numerous populations. The gene frequencies of the common alleles $ADA^1$ and $ADA^2$ have been established in many areas and several rare variant phenotypes, attributable to uncommon alleles at the ADA locus have been identified (Harris and Hopkinson, 1976).

The wide range of frequencies and the well-marked geographical differentiation suggest that his system will prove of considerable value in population studies. In Asiatic populations and to a less extent in Europeans, the frequency of the rarer of the two common genes is sufficient to make the system of value in family investigations of such features as linkage. Other ADA genes $ADA^3$, $ADA^4$, $ADA^5$, $ADA^6$ and $ADA^8$, are so rare as to be of very little interest in populations so far studied,

and even $ADA^2$ is not known to exceed 20 percent in frequency in any population.

2.3.5. The esterase D(EsD) system

The enzyme esterase D(EC 3.1.1.1.) is a ubiquitous tissue esterase of unknown biological function. This enzyme,which is present in red cells and most other tissues, was shown to be biochemically and genetically distinct from the previously recognized red cell esterases A, B and C(Tashian,1961,1969). So it represents one of the four ester isozymes described in human erythrocytes. It is distinguished from other tissue esterases by its electrophoretic mobility and by its narrow specificity for methylumbelliferyl esters as substrates.

Genetically determined variants of human red cell EsD was first described by Hopkinson et al(1973),by means of starch-gel electrophoresis,using an ester of fluorescent compounds (4-methylumbelliferyl acetate) as substrate. The appearance of fluorescent bands is due to the release of umbelliferon at the site of enzyme activity. Three distinct common phenotypes can be distinguished designated according to their electrophoretic mobilities ESD1-1, EsD2-1 and EsD2-2.(Figures 2.3.5.a and b). Family studies confirmed that these three phenotypes are determined by the co-dominant expression of two alleles,$EsD^1$ and $EsD^2$, segregating at an autosomal locus.Formal genetic researches have concluded to assignment of EsD locus on chromosome 13. The homozygous phenotype EsD 1-1 and EsD2-2 patterns each consist of three isozymes and EsD2-1 heterozygous phenotype, of five isozymes. However, the fastest moving band of the EsD2-2 and EsD2-1 cannot be clearly seen as they superimpose the slowest of the Esterase $A_2$ bands. The EsD1-1 and EsD 2-2 bands show decreasing anodal intensities with the slowest band of EsD2-2 having the same mobility as that of the fastest

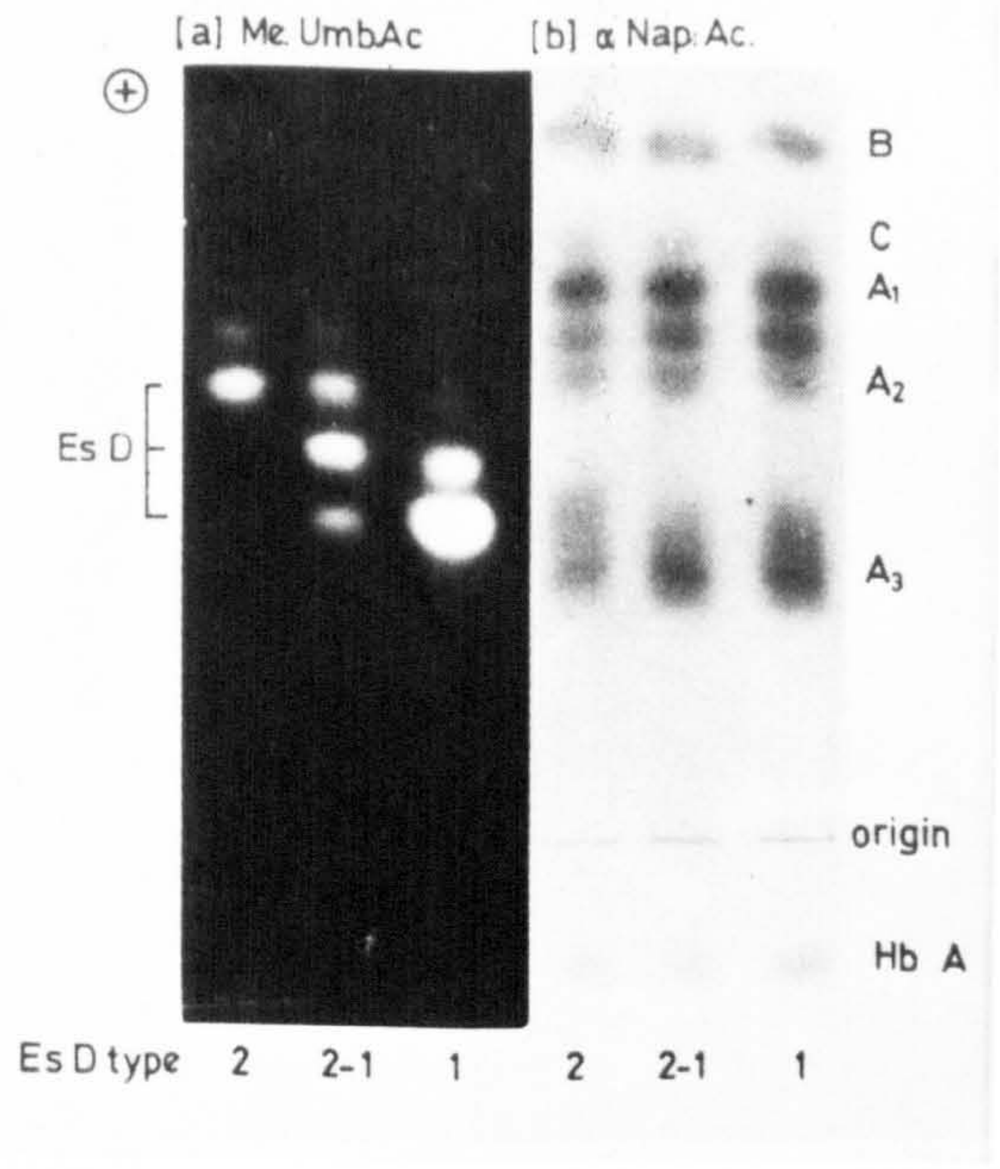



Fig.2.3.5.a. Red cell esterase isozymes stained with 4-methylumbelliferyl and ∝ -naphtyl acetate (Harris, H., and Hopkinson,D.A.1976).

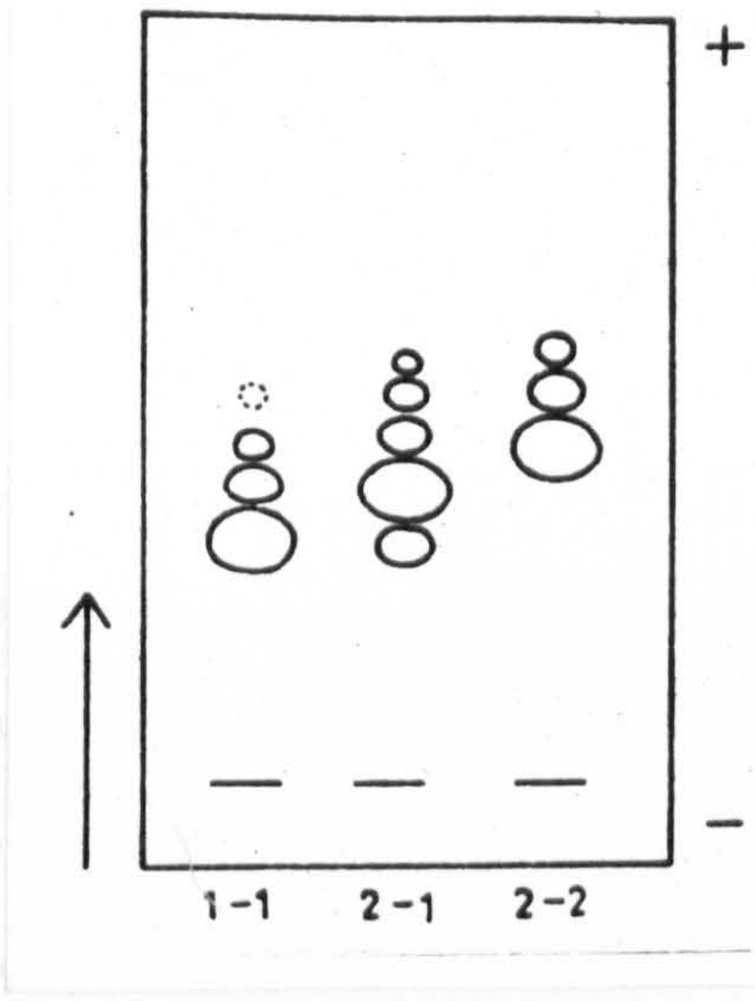



Fig.2.3.5.b. Starch gel electrophoretic patterns of the three common EsD phenotypes (Benkmann,H.G., and Goedde, H.W. 1974).

band of the EsD1-1 pattern. EsD2-2 can be easily distinguished due to its relatively greater mobility. Care must be taken in distinguishing EsD1-1 and EsD2-1. In the electrophoretic pattern of EsD1-1 phenotype, band 1 gradually loses its intensity until bands 1 and 2 are about the same intensity, with band 3 much weaker. In EsD2-1, the strongest band, band 2, loses intensity until bands 1, 2 and 3 are of the same intensity. Thus, when interpreting the EsD types,it has been found that the comparison of bands 1 and 3 in each type gives the most consistent interpretation. In EsD1-1,band 1 is stronger than band 3 and in EsD2-1,bands 1 and 3 are approximately of the same intensity.

Since the discovery of EsD polymorphism by Hopkinson et al (1973), several reports have been published of its distribution in European and Asian samples(Welch and Lee,1974; Koster et al, 1975; Cartwright et al 1976; Olaisen et al,1976). Of the two alleles,the $Es D^{1}$ gene is the more frequent in all populations so far studied, but the $Es D^{2}$ gene is commoner in Asia than in most European populations(Hopkinson et al, 1973; Benkmann and Goedde, 1974).

To date, two rare alleles for EsD has been described. Bender and Frank(1974) have reported the slow electrophoretic variant EsD3-1 in an individual from South West Germany, and Rittner and Muller(1975)have reported an example of EsD3-1 inna blood donor from Bonn and Bargagna et al(1975)have found the variant in Tuscany, Italy. This very rare phenotype indicates the existence of a third allele $Es D^{3}$; at the EsD locus. More recently, Berg et al(1976) have described even slower-electrophoretic variant EsD4-1, attributed to a fourth allele $Es D^{4}$. One instance of an individual apparently homozygous for a null allele, has also been reported. This was detected in an elec-

trophoretic study and there was no further family analysis, nor was there any quantitative characterization of esterase D activity (marks et al, 1977).

This system has already proved to be of considerable interest for genetic delineation of human populations.

2.3.6 The 6-phosphogluconate dehydrogenase (6PGD) system

The enzyme 6-phosphogluconate dehydrogenase (D-glucose-1-phosphate phosphotransferase, EC 1.1.1.4.1.)takes part in the hexose monophosphate shunt, that is to say, a relatively minor series of reactions branching off from the main anaerobic ( Embden-Meyerhof )glucose metabolic cycle, and converting hexoses into pentoses needed for making nucleic acids. It catalyses the next step in the chain of reactions after that catalysed by glucose-6-phosphate dehydrogenase, causing the conversion of 6-phosphogluconate to ribulose-5-phosphate, the co-enzyme, nicotinamide adenine dinucleotide phosphate (NADP) being simultaneously reduced to NADPH.

Though the enzyme occurs in many tissues, studies have been performed mainly on red cells in the form of haemolysates.

The existence of genetically determined variation of this enzyme in man was first demonstrated by Fildes and Parr(1963) by means of starch-gel electrophoresis of human red cell haemolysates. They found three electrophoretic patterns and showed that these isozymes represented the two homozygotes and the heterozygote of a pair of allelic genes, $PGD^A$ and $PGD^C$ at an autosomal locus. The commonest phenotype, AA, consists of a single anodal band in the 'a' position due to the homozygous presence of the $6PGD^A$ allele. The second most frequent.phenotype is the 'common' variant, CA,which is heterozygous for the $PGD^A$& $PGD^C$ genes.It exhibits an anodal band(a) and a cathodal band(b).Bands a

and b have equal intensity. Individuals homozygous for the $PGD^C$ gene exhibit the third variant designated the 'canning variant' CC. It consists of bands a, b and c, with the anodal band as a minor component.(Figure 2.3, 6.a.), Family studies showed that the inheritance of these variants is controlled by two alleles, $PGD^A$ and $PGD^C$ at an autosomal locus.

It is assumed that the enzyme 6PGD exists as a dimer, so that in the two homozygous types, the gene product is a subunit, (i.e. $S^A$ or $S^C$), which dimerizes to form either the a band ($S^AS^A$) or the c ban $S^CS^C$ ). In the heterozygote, both bands a and c are present, as well as the b band, which is thought to be a mixed dimer, $S^AS^C$( Parr, 1966; Parr and Fitch, 1967).

Apart from common variants, futhter variants due to alleles at the same locus have been found and examined by Fildes and Parr (1964), Parr and Fitch (1964), Parr and Parr (1965), Parr (1966), and Parr and Fitch (1967). These variants include two, recognizable electrophoretically, due to the alleles $PGD^H$ and $PGD^R$, as well as alleles producing unstable enzymes, and one which, in the homozygous condition, gives no detectable enzyme activity. The rare phenotype, due to the allele $PGD^R$, called the 'Richmond variant' was detected by Parr (1966) and in an American family by Davidson (1967). The corresponding phenottype appears to be $PGD^A$ $PGD^R$. The phenotypic pattern consists of three bands, a,d and e. The most cathodal band corresponds to the usual a band, while the intermediate band is intensively stained and predominates. Another rare variant due to the allele $PGD^H$, called the 'Hackney variant' was reported by Parr (1966). This phenotype apparently represents heterozygosity for $PGD^A$ and $PGD^H$. It differs from others in showing slow mobility of all the bands. Davidson (1967) has described

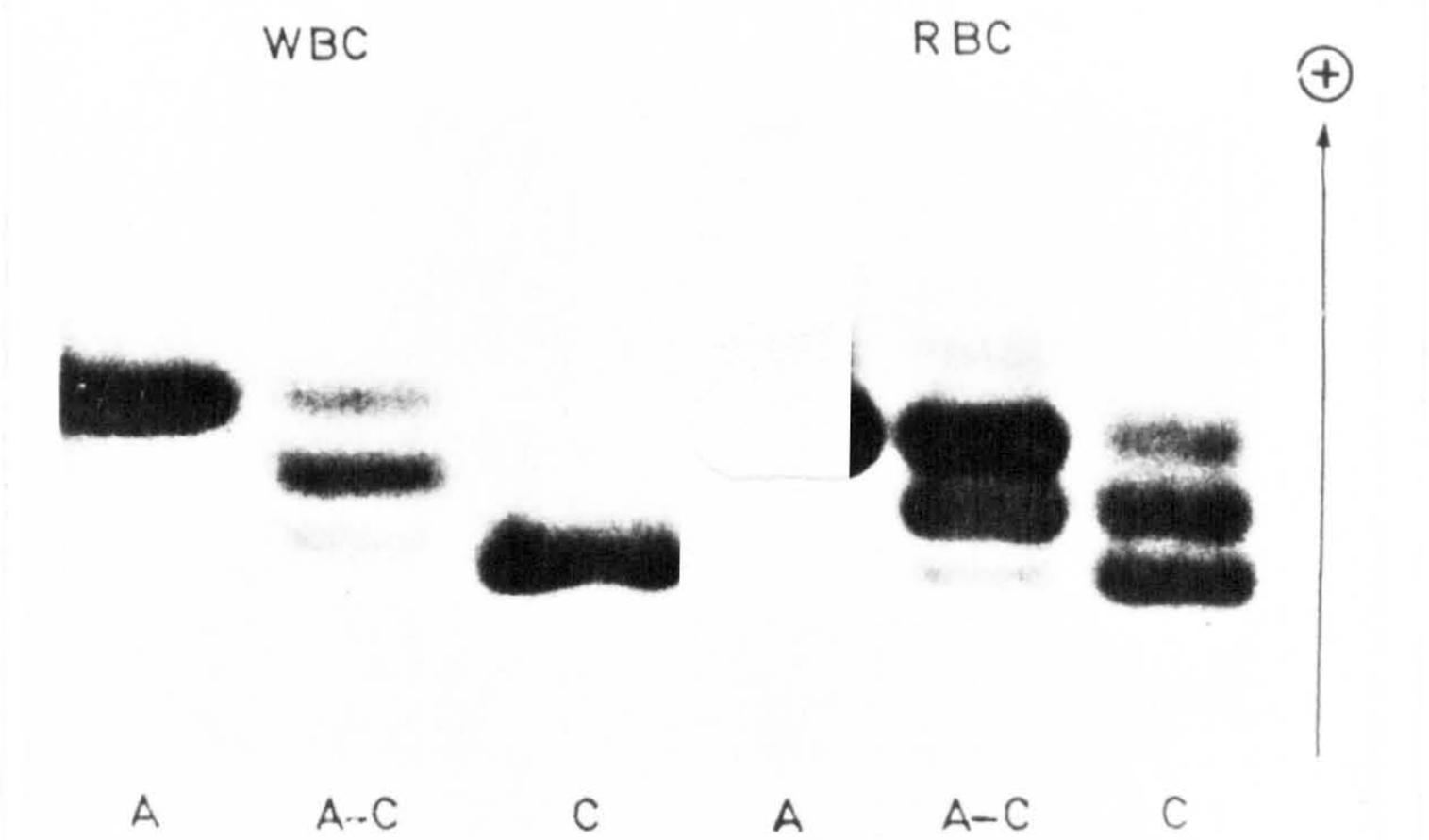



Fig. 2.3.6.a. Isozyme patterns in the three phenotypes PGDA, PGDA-C,PGDC in red cells and white cells. Note the more anodal secondary isozymes seen in the red cell samples(Giblett,E.R. 1969).

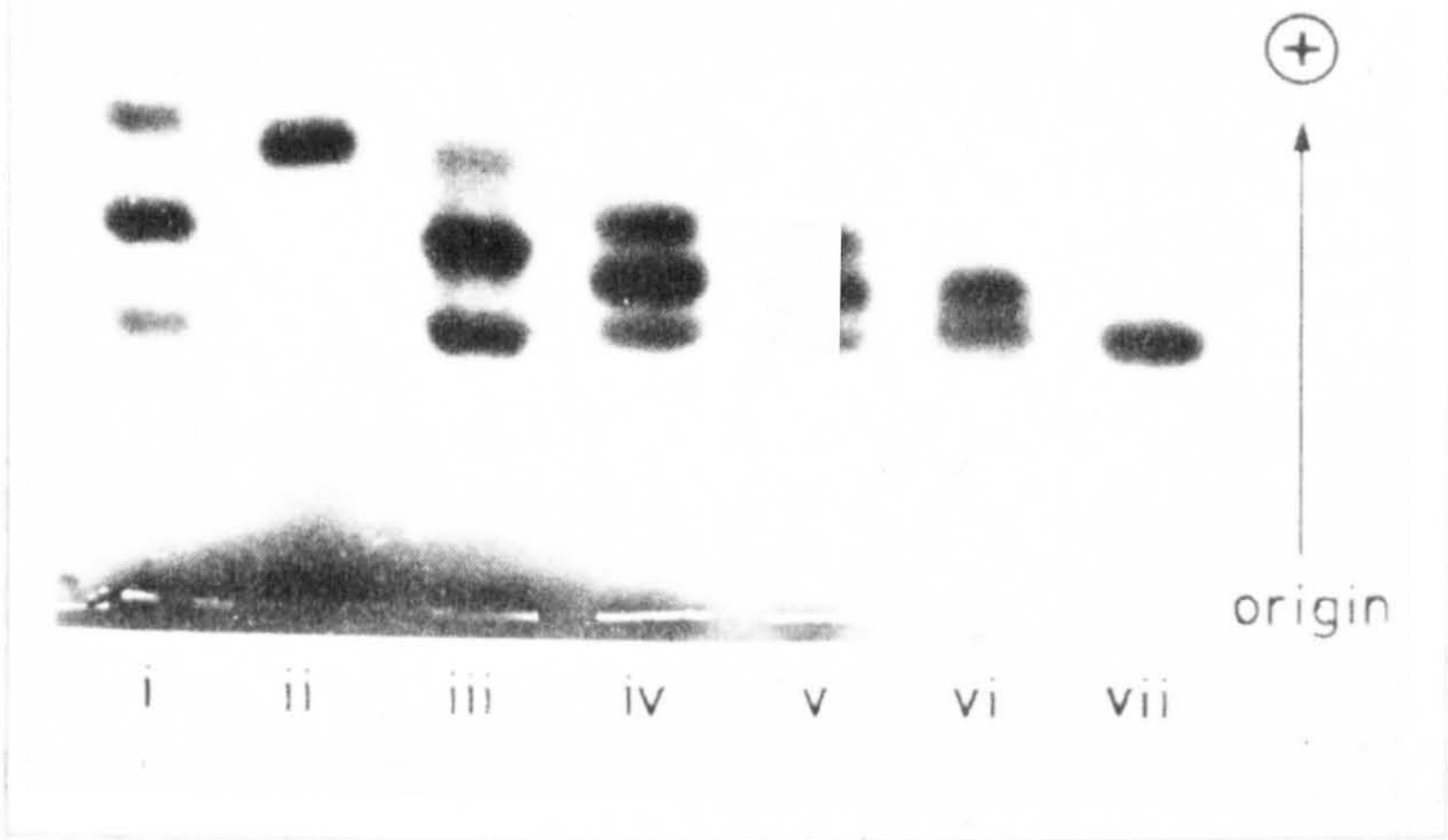



Fig.2.3.6.b. Some rare PGD phenotypes:(i)PGDA-Elcho;(ii)PGD Kadar (Presumed homozygote);(iii)PGDA-Kadar;(iv)PGDA-Caspian;(v)PGDA-Richard;(vi)PGDA-Bombay;(vii)PGDA(Blake,N.M.,et al. 1974).

a rare variant due to an allele $PGD^F$, called the 'Friendship variant'. Its electrophoretic pattern exhibits bands a, d and e. The most anodal band, e,is weakly stained, and the other two cathodal bands are of equal intensity. All these variants are controlled by a group of alleles at a single autosomal locus and are found normally in the heterozygous state. The whole subject of the variants and their relationships has been reviewed by Carter et al (1968) and by Giblett (1969), but Blake and Kirk (1969) have since described a variant of considerable anthropological interest. This is the 'Elcho' variant, so far found only in Australian aborigines( Figure 2.3. 6.b.).

In nearly all populations $PGD^A$ has a frequency exceeding 90 percent and $PGD^C$ comes next in frequency. All other variants are rare and sporadic.

Parr (1966) has shown that both the heterozygote $PGD^A$/ $PGD^C$ and the homozoygote $PGD^C/PGD^C$ have slightly less enzyme activity than the common $PGD^A/PGD^A$ homozygote. The heterozygotes of $PGD^A$ with $PGD^R$ and $PGD^H$ which have been found in some random population surveys, as well as that with $PGD^F$, appear to have normal activity but some of the very rare variants give rise to considerably reduced enzyme activity. It would not be surprising to find that such variations were associated with selective advantages or disadvantages.

The common genetic phenotypes consituting this genetic polymorphism, i.e., AA, AC and CC, as determined by the codominant alleles $PGD^A$ and $PGD^C$, are also characterized by variations in specific enzyme activity (Parr, 1966; Gordon et al, 1969; cf. Giblett, 1969). However, unlike G-6-PD, gross enzyme deficiency appears not to be of significance with regard to the mani-

festation and geographic distribution of this genetic polymorphism ( Parr, 1966; Parr and Fitch, 1967; cf. Giblett, 1969) . Thus, while contributing to the intraerythrocyte level of NADPH, like G-6-PD, the genetic variation underlying the 6-PGD polymorphism has not been implicated in the occurrence of endemic falciparum malaria. In fact, the significance of the anthropogenetic variation of this polymorphism in terms of adaptation, anthropogeography, etc, is still poorly understood (Tills et al, 1970, 1971; cf. Giblett, 1969; Ananthakrishnan, 1975).

2.3.7 The glucose-6-phosphate dehydrogenase (G-6-PD) system

The enzyme Glucose-6-phosphate dehydrogenase (G6PD: D-glucose-6-phosphate : $NADP^+$1-oxido-rductase, EC 1.1.1.49) is an essential catalyst in one of the body's method of oxidizing glucose, known as the pentose phosphate pathway, since it involves a breakdown from the 6-carbon molecule of glucose to a 5-carbon chain (G-6-phosphate to 6-phosphogluconate). Through the action of the enzyme the hydrogen so lost is transferred to nicotinamide adenine dinucleotide phosphate (NADP) to give rise to its reduced form NADPH. The latter is responsible through the action of the enzyme glutathione reductase, for maintaining an adequate level of reduced glutathion ( a tripeptide with an SH group ) necessary, in a manner not fully understood for the continuing integrity of the red cells. Thus in the absence of adequate G6PD activity red cells tend to haemolyse. The enzyme is present in red cells and also in other tissues.

Investigations of the enzyme in different human populations have shown that a wide variety of genetic variants exists. Two molecular forms, the B and A types, recognized by their electrophoretic mobility on starch gel, ar usual ( Figure 2.3.7.a ).

A very large number of variants of G6PD are now known (150

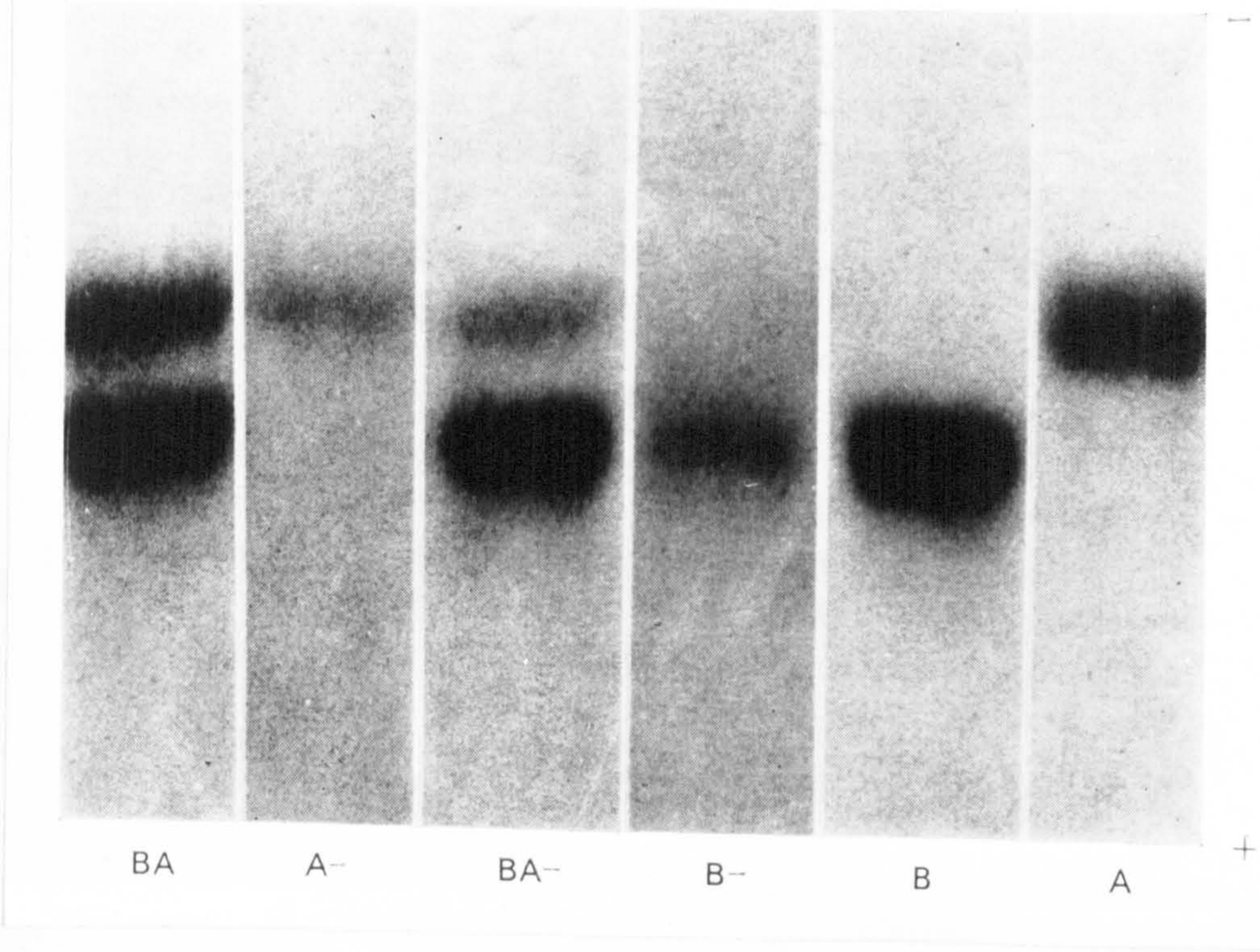



Fig.2.3.7. a. Starch gel electrophoretic patterns of six different G6PD phenotypes in red cell haemolysates (Giblett,E.R. 1969).

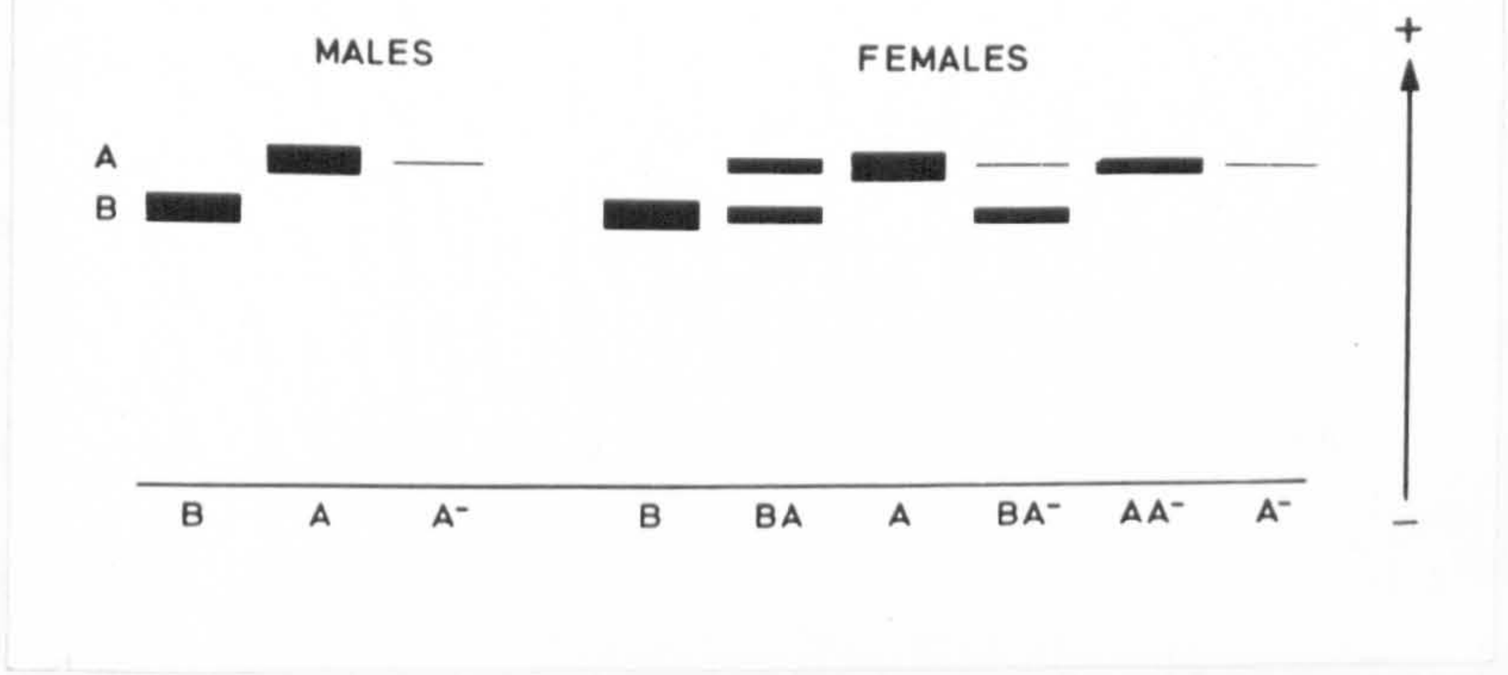



Fig.2.3.7.b. Diagram of red cell glucose-6-phosphate dehydrogenase isozymes in Blacks of different phenotypes (Harris,H., and Hopkinson, D.A. 1976).

G6PD variants tabulated by Yoshida and Beutler, 1971, 1978 ; and Beutler and Yoshida, 1973) but, in addition to the normal type, only three variants are commonly recognized as being of any importance in population studies. The common type in all populations is known as $B^+$, the B referring to its rate of migration on electrophoresis and the + to its normal enzymatic activity. The common abnormal type in the Mediterranean area is B-, migrating at the same speed as the normal but with an activity between 0 and 7 percent of normal. It is, of course, possible to determine the speed of migration by the standard electrophoretic method only if at least a trace of enzyme activity is present.

In African populations two abnormal types are known, A+ which has normal activity but which migrates faster than the normal, and A- which migrates at the same speed as A+ but has only 8 to 20 percent of normal activity.

The variants of G6PD are determined by a series of allelomorphic genes on the X chromosome. Thus a male with only one X chromosome can have only one type of enzyme, but a female, having a pair of X chromosomes, can be heterozygous and have genes for two different alleles ( Figure 2.3.7.b.).

Tests are of three main kinds, screening tests for the presence of enzyme deficiency, precise quantitative tests to measure the degree of deficiency, mainly of use in identifying rare variants, and tests by starch gel electrophoresis, which, provided that the blood specimens are fresh so that all the variants retain some enzyme activity, will in males,and to a large extent also in females, show both the relative electric charge and the activity of any variants present.

Deficiency of erythrocyte glucose-6-phosphate dehydroge-

nase is the commonest genetic enzymatic abnormality in human beings, probably affecting more than a hundred and fifty million males. It has long been known that some persons in many Mediterranean countries suffer from favism, a haemolytic anaemia wiht an appreciable mortality, precipitated by consumption of the common horse bean or broad bean, vicia fava. During World War II it was found that some American Negro soldiers suffered from haemolysis when treated with certain antimalarial drugs derived from quinoleine, such as primaquine. Investigations by numerous workers finally traced both these conditions, and a number of others such as jaundice among newborn Chinese infants in Singapore, to a deficiency in the red cells, of the enzyme, glucose-6-phosphate dehydrogenase, the complicated history of research on the genetics of this enzyme has been reviewed by many authors including Giblett (1969)who gives full references to previous works.

Over the past decade the genetic variability in man associated with the enzyme G-6-PD has rapidly developed into a subject of major concern in medical, genetic (Pharmacogenetic ), and anthropobiological research.

The central theme underlying this interest concerns the occurrence of two major types of genetically determined enzyme deficiencies, i.e., the Negro-type (10%-15% of normal enzyme activity in red cells ) and the Mediterranean type (8% of normal enzyme activity in red cells), both affecting millions of people, with all the clinical, public health and anthropogeographical implications associated with it. Genetically, the two types of enzyme deficiency are attributable to the manifestation of two alleles, $Gd^{A-}$ and $Gd^{B-}$, respectively, at the sex-lniked Gd locus. The alleles $Gd^{A}$ and $Gd^{B}$, on the other

hand, are characterized by the synthesis of enzyme molecules with 'normal' enzyme activity and electrophoretic mobilities indistinguishable from those of their defective counterpart molecules. Biochemically, the dimeric enzyme G-6-PD is responsible for the first and rate-controlling reaction in the hexose monophosphate shunt pathway in red cells. With this reaction NADPH is generated, an ample supply of which is essential for the stability and longevity of red cells.However, under certain dietary conditions, after treatment with a variety of specific drugs, or after infections by certain bacteria, the genetically determined enzyme defficiencies impair the normal metabolic function of the red cells, cause their premature destruction, and thus enhance the possibilities of overt manifestation of severe haemalytic anaemia, especially in children (allison, 1960, 1963; Motulsky, 1960, 1961, 1964, 1965; Motulsky and Campbell-Kraut, 1961; Motulsky et al, 1966; cf.Giblett, 1969; Livingstone, 1971; Allison, 1975; Flatz and Xirotiris, 1975). The postulated association between the worldwide distribution of these enzyme-deficient genetic markers and the endemicity of falciparum malaria represents a significant step in the understanding of the selective mechanism possibly under - lying the maintenance of this polymorphic genetic system and provides a basis for specifically directed population qenetic and anthropogeographical research. The malaria hypothesis is based mainly on the striking correlation between the geographic distribution of endemic falciparum malaria and that of the enzyme-deficient alleles $Gd^{A-}$ and $Gd^{B-}$, and also on supporting comparative parasitological studies. More specifically, it is postulated that whereas hemizygous males and homozygous females for normal enzyme activity are highly susceptible to mala-

ria infections, they are in no undue danger when exposed to certain dietary or pharmacological haematolytic agents. Conversely, whereas enzyme-deficient hemizygous males and homozygous females are likely to develop severe haemolysis upon exposure to the conditions and agents mentioned( leading to elevated mortality, especially during childhood), they are at a relative selective advantage in malarious environments, in that they enjoy a measure of natural resistance against malaria parasites.Follwoing this reasoning, heterozygous females are regarded as optimally equipped genetically against malaria infections, without undue risk of suffering severe bouts of haemolytic anaemia when exposed to especific drugs, diets, or certain bacterial infections. It follows that this genetic polymorphism appears to be maintained in the population by a balance of selective forces in heterozygotes analogous to the sicklecell polymorphism. A significant difference, however, is the fact that whereas the latter is autosome-linked, the Gd locus is sex-linked, entailing its subjection to lyonization and a more complex selective meachanism and approach to genetic equilibrium. The crucial factor in this type of balanced polymorphism obviously is the selective advantage and greater genetic fitness enjoyed by the heterozygous female (Allision, 1960, 1963; Motulsky, 1960, 1961, 1964, 1965; Motulsky and Campbell-Kraut, 1961; Motulsky et al, 1966; Luzatto et al, 1969; Bienzle et al, 1972; cf. Giblett, 1969; Livingstone, 1971; Allison, 1975; Flatz and Xirotiris, 1975).

2.3.8. The lactate dehydrogenase (LDH) system

Lactate dehydrogenase (L-lactate: NAD oxidoreductase, EC 1.1.1.27) catalyses the reversible reduction of pyruvate to lactate. This enzyme is present in many tissues and has been

the subject of much investigation. Work done on it up to 1965 has been very fully reviewed by Vessel(1965).

The molecules of this enzyme are tetramers of two types of polypeptide chain, A and B. Each tetramer molecule can contain any number from 0-4 of any type of chain. Thus, in the normal case, where only the single common homozygote of each chain type is present, five molecular types are present, A4, A3B, A2B2, AB3, and B4, giving rise to five electrophoretically distinct bands. (Figure 2.3.8). The two chain types are the products of two sets of alleles at different autosomal loci which are not closely linked. The relative strength of the various electrophoretic bands differ according to the tissue or red cell type from which the enzyme is derived. The isozymes are numbered from LDH1 ( the fastest moving towards the positive pole) for B4 to LDH5 ( which migrates slowly towards the negative pole) for A4. The structure of each isozyme is as follows:

LDH-1 = BBBB (B4)

LDH-2 = BBBA (B3A)

LDH-3 = BBAA (B2A2)

LDH-4 = BAAA (BA3)

LDH-5 = AAAA (A4)

Only bands 1, 2 and 3 are well developed on electrophoretic patterns of red cell haemolysates, variants at either locus are all of very low frequency but a considerable number of rare variants only in the form of heterozygotes with the normal allele, are known (Vesell, 1965), some apparently affecting the A chain and some the B chain. Because of the lack of constraint as to the manner in which tetramers can be built up, a heterozygote at one locus should be able to give rise to fifteen bands, and a bouble heterozygote (a type not yet identified ) to thirty five.

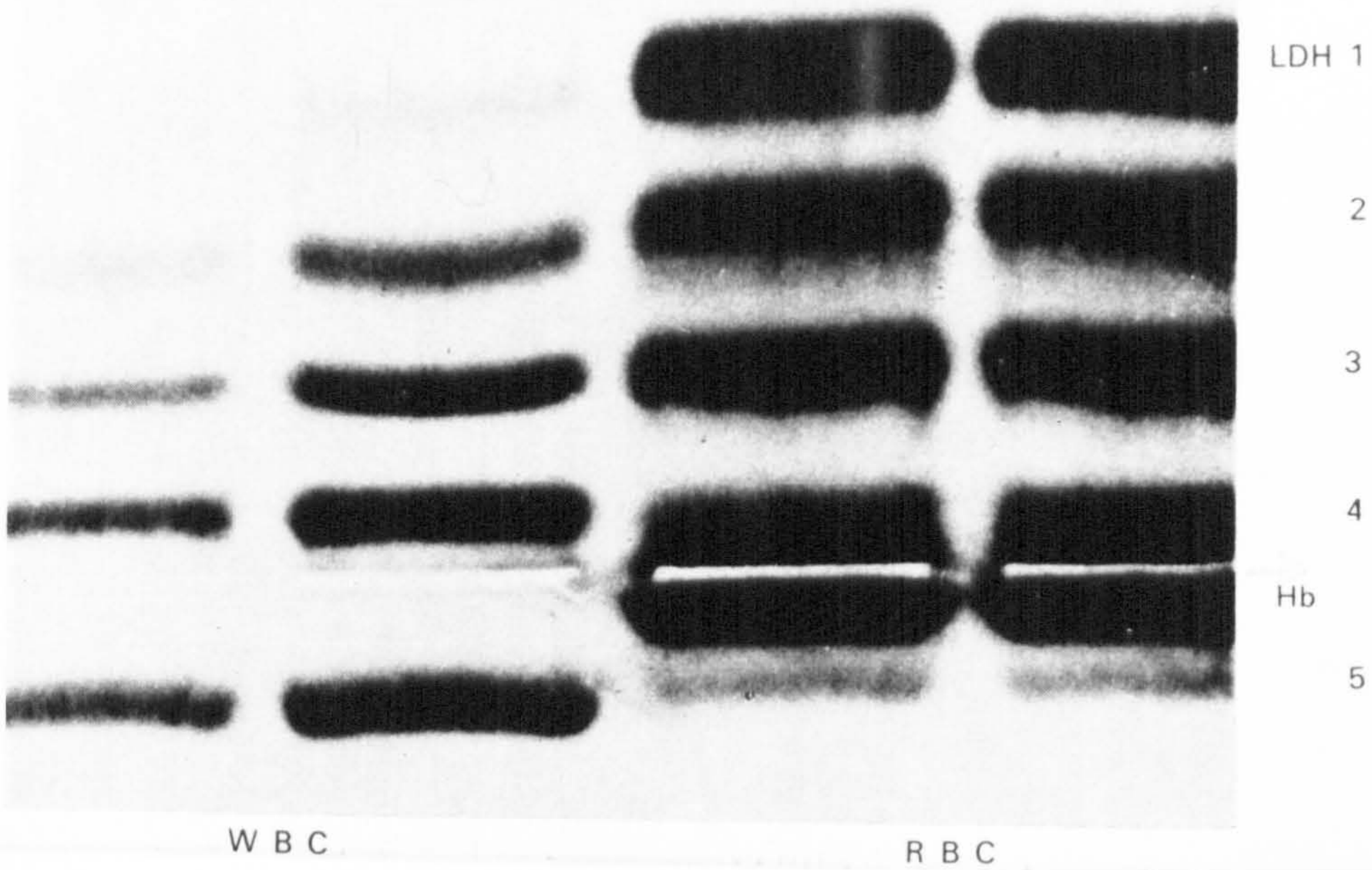



Fig.2.3.8. Electrophoretic patterns of LDH isozymes in lysates of white cells (left) and red cells (right). From Giblett,E.R.1969.

The patterns actually observed involve a splitting of the main bands LDH 1-5 into spectra of fine lines. The complete analysis of a given variant so as to interpret it in terms of an allele at the A or B locus is a very complicated process. Thereafter, newly observed variants can with more or less confidence be identified by running them alongside a known variant on the same gel.

Variants are in general very rare, occurring with a frequency of the order of 1 in 1000 in most populations.

The most widespread variant appears to be memphis -4 of Kraus and Neely (1964) which is a slow A variant. This (or a type or types indistinquishable from it) is the main variant in European populations. Type Calcutta 1, heterozygous for a fast A variant, has a frequency of somewhat over one percent in various Indian populations. A number of other variants are known, all with very low frequencies indeed.

2.3.9 The malate dehydrogenase (MDH) system

The enzyme malate dehydrogenase (l-malate: NAD reductase, EC 1.1.1.37) catalyses the reversible oxidation of malate to oxaloacetate. Two electrophoretically distinct forms of the enzyme are foundrin man, one present in solution in cytoplasma, including red cells and one localised within the mitochondria ( Christie and Judah, 1953; Shrago, 1965). These are the products of genes at separate autosomal loci, not closely linked. Nearly all populations are uniformly homozygous for one common gene, but variants are known in a few populations.

Genetically controlled polymorphism of the cytoplasmic enzyme was first described by Davidson and Cortner (1967) in a survey of 1,440 North American whites and 1,470 North American Negroes. The common MDH phenotype, MDH1-1, which is homozygote

for the usual allele $MDH^1$, exhibits on electrophoresis, when fresh, one strong band migrating towards the anode, with some faint and variable faster bands which disappear in older specimens. All the whites and all but one of the Negroes showed the common phenotype, but one Negro exhibited a variant pattern, MDH2-1 heterozygote phenotype, migrating more slowly than the normal, and consisting of three bands, the fastest of which had the same mobility as the single major band of the common pattern and the other two with slower mobility. From a family sutdy of the propositus, they were able to show that the variant and the usual types behave as if controlled by a pair of co-dominant alleles at an autosomal locus.

Blake et al (1970) described a fast variant, termed MDH3-1 in a number of persons from New Guinea. This heterozygote phenotype exhibits two moderately strong bands, together with a stronger intermediate band, and a fainter and more transient band in front of each.

Leakey et al (1972) reported the homozygote phenotype MDH3-3 among some people of New Guinea. It exhibits one strong band, faster than that for the common type, together with one fainter faster band which is absent in older specimens( Figures 2.3.9. a and b.).

The heterozygote patterns are presumably due to each molecule being a dimer, with the heterozygote giving two pure dimers and one mixed one.

The slow variant has been found only in the heterozygous 2-1 form. The fast variant, mostly as the 3-1 heterozygote, but including a few 3-3 homozygotes, has up to now been found only in Papua, New Guinea.

Many thousands of persons from different parts of the world

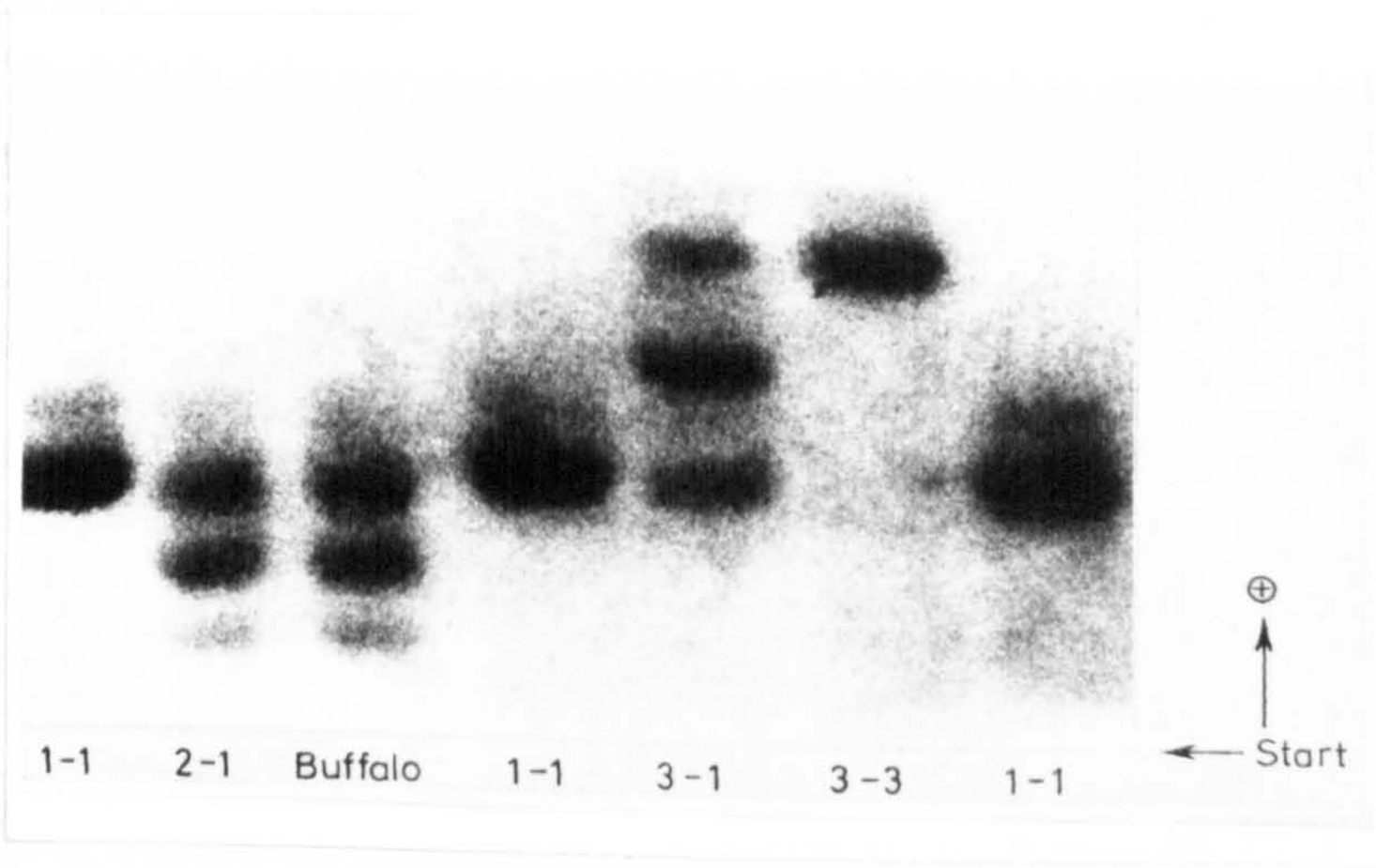



Fig.2.3.9.a. Electrophoretic patterns of the usual (MDH 1-1) and some variant phenotypes of soluble malate dehydrogenase in red cells (Leakey, T.E.B., et al. 1972).

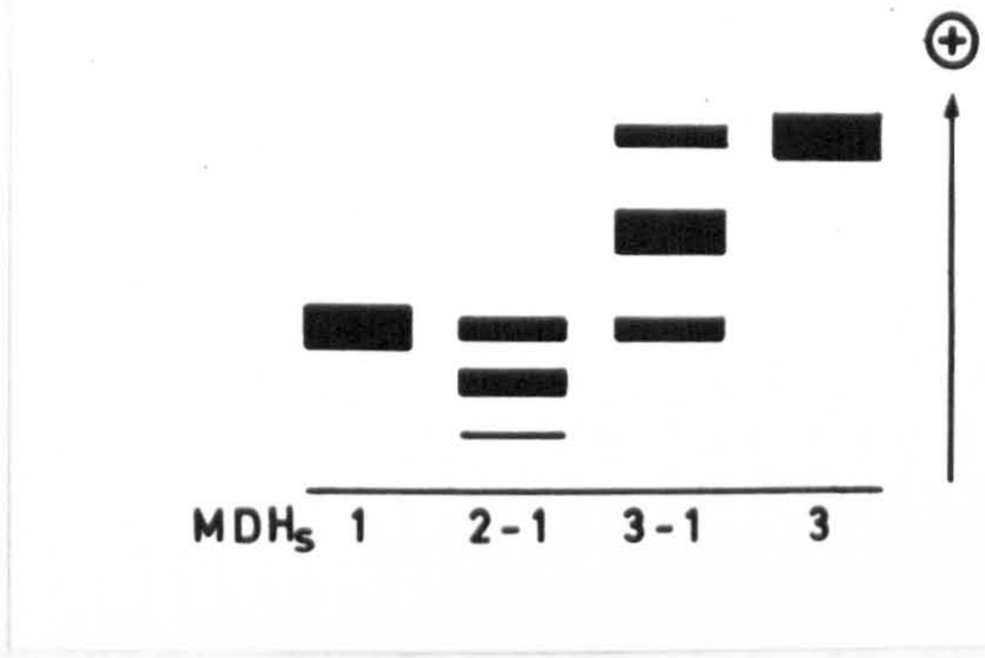



Fig. 2.3.9.b. Diagram showing the usual (MDH$_S$ 1) and some variant phenotypes of soluble malate dehydrogenase in red cells. Only the primary isozymes are shown (Leakey,T.E.B., et al. 1972).

tested for MDH system have shown only the common 1-1 homozygote type.

2.3.10 The phospho hexose isomerase (PHI) system

The enzyme phosphohexose isomerase, also known as phosphoglucose isomerase, glucose phosphate isomerase, or D-glucose-6-phosphate Ketol isomerase (EC 5.3.1.9) catalyses the reversible conversion of glucose-6-phosphate to fructose-6-phosphate.

Genetically determined variants of this enzyme were first demonstrated by Detter et al (1968). They found, in addition to the usual phenotype, PHI1-1, ten distinct variant phenotypes called PHI2-1, 3-1, 4-1, 5-1, 6-1, 7-1, 8-1, 9-1, and 10-1. Family studies showed an autosomal codominant mode of inheritance for those variants that have been subjected to it, and PHI1-1 individuals are homozygotes for the common allele at the PHI locus. The variant phenotypes are found in individuals who are heterozygous for the common allele $PHI^1$ and one of the rare alleles at this locus. Nothing is known about the inheritance of phenotypes 2-1, 4-1, 7-1, and 8-1.

The usual type PHI1-1 exhibits three bands, one major and two minor. The major band is cathodal. All the vairant phenotypes, except PHI9-10, consist of three major bands, one of which corresponds to the major cathodal band of the usual type. (Figure 2.3.10). The triplet pattern of the variant phenotypes suggests a dimeric structure. It is assumed that the heterozigosity at the PHI locus produces two subunits, one usual and one variant. Phenotypes such as 2-1, 3-1, 4-1 and 6-1 have a negatively charged variant subunit, whereas in types like 7-1, 8-1, and 9-1 the variant subunit is positively charged. Phenotype PHI7-1 represents a hybrid dimer. All the variant isozyme patterns are rare in the populations reported upon to date.

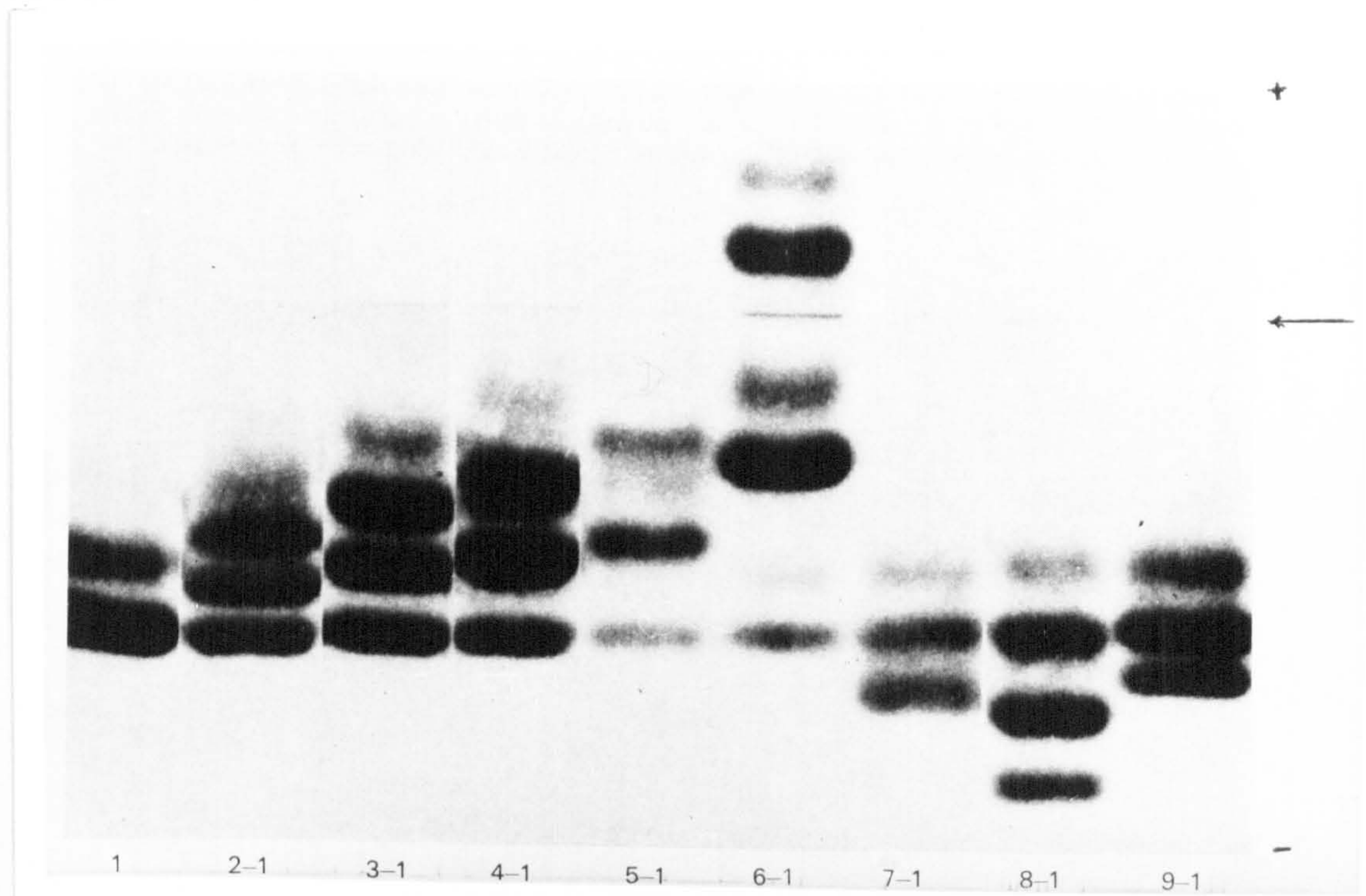



Fig.2.3.10. Electrophoretic patterns of nine phosphohexose isomerase(PHI) phenotypes. The usual phenotype, PHI 1, is on the left. The arrow points the origin ( Giblett, E.R. 1969).

## 2.3.11 The glutamic-pyruvic trasaminase (GpT) system

The enzyme glutamic-pyruvic trasaminase, synonym alanine aminotransferase (GpT; EC 2.6.1.2), catalyses the reversible interconversion of L-alanine and $\alpha$-ketoglutarate to L-glutamate and pyruvate. In rat liver, the enzyme occurs in two distinct molecular forms: a cytoplasmic or soluble form and a mitochondiral or granular form ( Swick et al, 1965). It is probable that a similar state prevails in human liver and other human tissues. In human red blood cells, a soluble form occurs which has been shown, by means of starch-gel electrophoresis, to exhibit genetic polymorphism. The polymorphism is determined by two alleles $GpT^1$ and $GpT^2$. An autosomal codominant mode of inheritance is known for this enzyme system. Three common phenotypes, (Figure 2.3.11.a), called GpT1-1, GpT2-1, and GpT2-2 are distinguishable ( Chen and Giblett, 1971). Apart from their different electrophoretic patterns, each of the phenotypes has a different mean level of enzyme activity. GpT1-1 individuals have, on the average, 2-3 times the red cell GpT activity of GpT2-2 individuals, while the GpT2-1 phenotype shows intermediate levels (Welch, 1972; Chen et al,1972; Olaisen, 1973; Kompf and Bissbort, 1974). Unfortunately, measurements of enzyme activity alone do not adequately distinguish members of the different phenotypes, as considerable overlaps in activity occur ( Welch, 1972; Chen et al, 1972). In addition to the common alleles $GpT^1$ and $GpT^2$, population studies have led to the discovery of several rare phenotypes (Figure 2.3.11 b), and it has been postulated that six other alleles, $GpT^3$, $GpT^4$, $GpT^5$, $GpT^6$, $GpT^7$, and $GpT^8$, exist (Chen et al, 1972;Olaisen, 1973; Santachiara Benerecetti et al, 1975; Gussmann and Schwarzfischer, 1972; Spielmann et al, 1973; McAlpine et al,

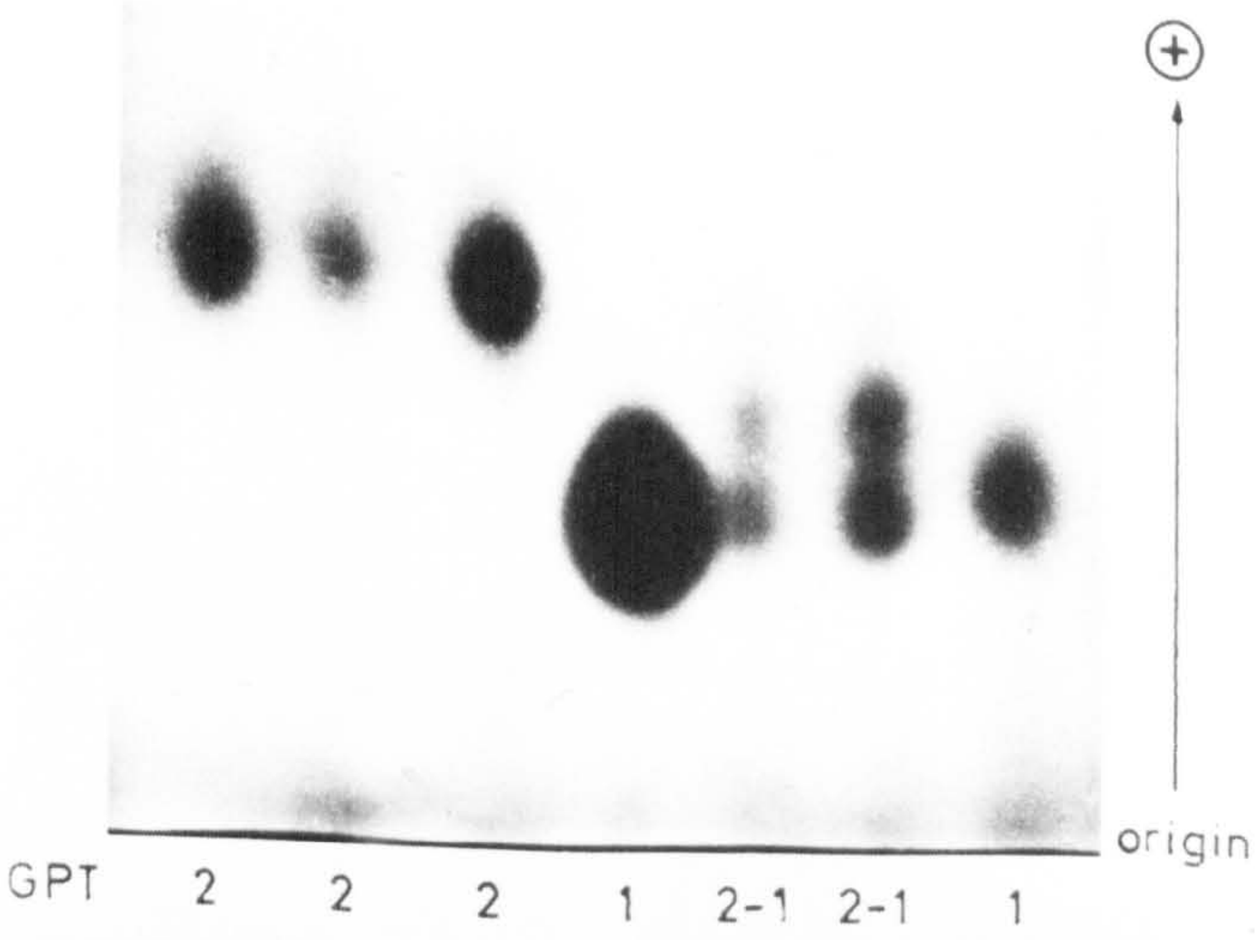



Fig.2.3.11.a. GPT in haemolysates from individuals of the three common phenotypes GPT 1, 2-1 and 2 (Harris,H., and Hopkinson,D.A. 1976).

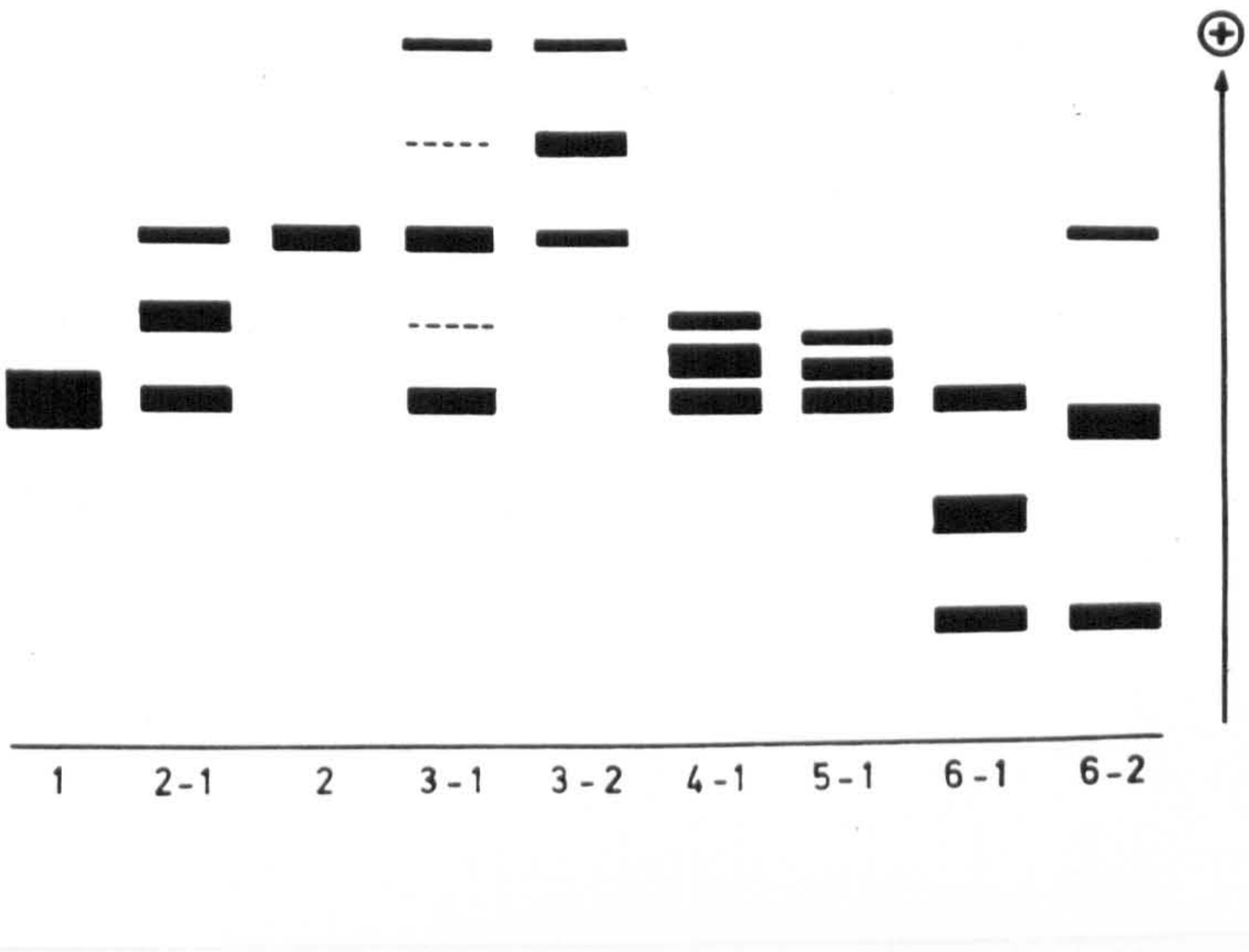



Fig. 2.3.11.b. Diagram of electrophoretic patterns of nine different GPT phenotypes( Chen, S.H., et al. 1972).

1974; Ishimoto and Kuwata, 1974; Olaisen, 1973; Brinkmann et al, 1972; Martin and Niebuhr, 1973; Wiebecke and Brackebush, 1974). Evidence for the existence of a GpT "silent" allele ($GpT^0$) has been reported by Olaisen (1973) in a Norwegian family, by Spielmann et al (1973) and very recently by Mithal et al (1980).

# CHAPTER 3. MATERIALS AND METHODS

## 3.1. Blood collection

A total of 1709 blood samples were collected from ten different population groups of Iran for the purpose of the present study.

The areas and the population groups belonging to them were as follows:

| Areas | Population groups |
|---|---|
| Tehran | The Moslems |
| | The Zoroastrians |
| Rezaieh | The Turks |
| | The Kurds |
| Luristan | The Lurs |
| Kerman | The Kermanis |
| Sistan and Baluchistan | The Zabolis |
| | The baluchis |
| Shirvan,Khorasan | The Turks |
| | The Kurds |

All the blood donors were healthy and unrelated. These samples were taken in several occasions during 1979, 1980, 1981 and 1982.

In the present investigation blood grouping was carried out for the following blood group systems:

ABO, MNSs, Rh, P, Kell, Duffy and Kidd, and electrophoresis was performed for the following serum protein and red cell enzyme systems:

Haptoglobin(HP), Transferrin (Tf), the third component of human complement (C3), Acid phosphatase (AcP), Adenylate Kinase(AK), Phosphoglucomutase Locus 1 ($PGM_1$), Adenosine deaminase (ADA),

Esterase D (EsD) and 6-phosphogluconate dehydrogenase(6-PGD). Two out of the seven blood group (ABO and Rh), all the three serum protein and four out of the six red cell enzyme systems (AcP, AK, $PGM_1$, and EsD) were done for all the above mentioned samples but the remaining blood group and red cell enzyme systems were done if not for all the samples but for most of them.

A total of 645 blood samples of Tehran and Kerman were tested for blood group and serum protein systems in Tehran( Unit of human genetics and anthropology, school of public health, University of Tehran) and the lysates were brought to Durham by air and at dried iCe temperature (-78$^{o}$C) for the red cell enzyme systems analysis.

Another total of 670 blood specimens of Rezaieh, the Zoroastrians and Shirvan were analysed for all the blood group, serum protein and red cell enzyme systems in Tehran.

The final collection of blood samples of Luristan and Sistan, Baluchistan( total = 394) were tested only for serum proteins in Tehran and the fresh red cell suspensions and the lysates were taken to Durham within 72 hours of their collection and were tested for blood groups and for the red cell enzyme systems.

## 3.2.Laboratory procedures

When the blood samples were received at the laboratory:

1)The plasma was separated from the red cells by centrifugation into tubes and stored at -20$^{o}$C until required for serum proteins analysis. A few drops of plasma were placed in another tube for ABO grouping procedures.

2)For blood grouping procedures a few drops of red cells (0.5-1.0 ml) were separated into tubes and washed three times in normal Saline (0.85 percent) and then diluted to a 4 per-

cent suspension.

3)Haemolysates were prepared by the Carbon tetrachloride method of Ager and Lehmann (1961); briefly described below: The remaining red cells were washed three times in normal Saline. An equal volume of distilled water was added to the washed red cells, a volume of carbon tetrachloride,at least equal to twice the volume of cells plus distilled water was added and all the contents throughly mixed The tubes containing the mixture were centrifuged at 3000 R.P.M. for 20 minutes. The supernatant was placed in tubes and stored at -20$^{o}$C untill required for red cell enzymes analysis.

## 3.3.Blood grouping techniques

Three main blood grouping techniques were used and these will be described briefly.

### 3.3.1.Tile technique

The tile technique involves the use of an equal volume (one drop) of antiserum and 4 percent saline suspension of red cells.The cells were mixed with the particular antiserum on a clean white tile and left for a fixed period of time at a certain temperature(these two points being dependent on the antiserum being used).The tile is then rocked gently and inspected for agglutination over a strong light.

The following antisera required this technique; anti-A, anti-B, anti-AB, anti-A1, anti-A2, and anti-P1.

### 3.3.2.Tube technique

The tube technique involves placing an equal volumes of 4 percent red cell suspension and the appropriate anti-serum into precipitin tube and leaving at a prescribed temperature for a specific period of time. After this time the mixture is examined for agglutination microscopically.

The following antisera required this method; anti-M, anti-N,

anti-D, anti-C, anti-c, anti-E, anti-e and anti-$Lu^a$.
In the case of incomplete antibodies, a layer of 30 percent bovine serum albumin was added as an overlay for the reason described above after one and one and a half hours, and left for a further thirty minutes. Only anti-$C^w$ serum required this latter technique. The presence of agglutination is investigated microscopically.

### 3.3.3. Indirect coombs technique

The remainder of the blood grouping required the Indirect coombs test. In this test one volume of the antiserum is incubated with one volume of 4 percent red cell suspension at 37°C in a precipitin tube for a specific period of time.
After-wards the cells, removed of antisera by washing four times in large volumes of saline, are placed on a clean tile with one drop of anti-human globulin reagent. The tile is then rocked for 5-10 minutes and the mixture is inspected for agglutination over a strong light.
This method was used with the following antisera; anti-S, anti-s, anti-$Fy^a$, anti-$Fy^b$, anti-$Jk^a$, anti-$Jk^b$, anti-K, anti-k (anti cellano), anti-$KP^a$, and anti-$KP^b$. Also all Rh(D) negative cells were tested by this method for the presence of the $D^u$ antigen.

All the controls were set up at the same time, under the same conditions and were read immediately before the tests.

### 3.4.Thick layer starch gel electrophoresis

During the last fifty years, and especially over the last twenty years, the discovery of new techniques has led to the detection of many genetically determined polymorphisms in man, and the frequency of the genes and phenotypes can be used in classifying and comparing populations. Genetic traits now com-

monly investigated in population surveys include the major blood group antigens and the serum protein and red blood cell isoenzyme polymorphisms. The single most important technique developed in recent years is starch-gel electrophoresis (Smithies 1955).

Demonstration of serum and red cell enzyme phenotypes by horizontal starch-gel electrophoresis depends upon separating the individual components on the basis of their molecular size as well as their electrical charge. The resolving power of this medium has a greater ability to separate complex serum proteins, enzymes, hormones, tissue extracts, etc. The mechanism for high resolving power is unknown but it is believed that molecular sieving plays an important role. Another advantage of the method lies in the relative ease of manipulation and the sharpness of the Zones obtained.

In the present investigation this method was only used for typing of the haptoglobin groups.

3.4.1.Apparatus used:

Electrophorsis on starch gel was carried out in the horizontal position and the equipment consisted of:

1)A power pack capable of providing a constant current up to 50 mA and a constant voltage up to 500 volts.

2) Plastic tanks for tank buffer solution.

3)Platinum electrodes.

4)PH meter.

5)Gel plates: plastic plates were used with formers. The formers were placed over the plates and held secure with elastic bands around each end.The internal dimensions of the plates were 20 cmx 15 cm x 7mm.The plates were greased with liquid paraffin, before pouring the gel, to stop the gel sticking.

6) A electrical stirrer.

7)A vaccum pump.

8)Dental forceps for inserting the specimens on the gels.

9)Shandon comb for making slits in the gels.

10) A slicer board fitted with a horizontal wire for cutting the gels.

11) Plastic boxes for staining the gels.

### 3.4.2. Preparation of gel

For the preparation of the gel, starch obtained from Sigma or merck company was used. For each gel, a suspension of 25 gms of starch in 250 mls of gel buffer was heated over a gas flame in a one litre pyrex flask. The contents were agitated by an electrical stirrer. Heating was continued until boilling. A vaccum was applied to the flask for a few seconds for expel the air bubbles. After disconnecting the vaccum, the starch solution was poured slowly into the plastic plates until they were full. The gels were left to set for one hour at room temperature.

### 3.4.3. Sample insertion

After the gels had set, a cut was made with a surgical blade across each gel about two inches from the cathodal end. The gel was pushed back gently to allow space for the insertion of samples. Care was taken to avoid breakage or distortion of the gels. A piece of whatman No.3.filter paper was held by forceps and immersed in the serum sample. The moist filter paper was inserted into the cut so that it adhered to the surface of the gel. The slot was closed by applying slight pressure on the gel so that it came back to its original position. A space of 1-2 mm was left between the adjacent inserts.

### 3.4.4. Slicing the gel

After electrophoresis the formers were removed and the

inserted sample strips of paper were removed too, with forceps and then the gels were cut just above the insert line and 3-4 inches above the insert line. This was discarded leaving the middle protein. The gels were sliced horizontally by drawing them slowly through the wire of slicer. The required height of the horizontal wire was fixed with the height adjuster. The plastic plates with the gel on them were removed gently towards the wire to cut them in half. The sliced gels were then transfered to plastic boxes for staining.

3.5.Thin layer starch gel electrophoresis

This method was used in the present investiation for typing of all the red cell enzyme systems.

3.5.1. Apparatus used:

Electrophorsis on starch gel was carried out in the horizontal position and the equipment consisted of:

1) A power pack capable of providing a constant current up to 50 m A and a constant voltage up to 500 volts.

2) Plastic tanks for tank buffer solution.

3) Platinum electrodes.

4) PH meter.

5) Gel plates: Glass plates were used with 1 mm high glass side pieces set at the edge with araldite to provide a former for the gel. The internal dimensions of the plates were 20 cm x 15 cm x 1 mm.

6) A electrical stirrer

7) A vaccum pump

8) Dental forceps for inserting the specimens on the gles.

9)Shandon comb for making slits in the gels.

10) Plastic staining formers: These are rectangles of plastic which hold the agar overlay in place. Two size were required

for the different electrophoretic runs, their external dimensions were 13½ cm x 8 cm and 13½ cm x 11½ cm; they were 3-4 mm deep.

3.5.2. Gel making

For the preparation of the gel, starch obtained from sima or Merck company was used. For each gel, a suspension of 3.5 gms of starch in 40 mls of gel buffer was heated over a gas flame in a 250ml pyrex flask. The contents were agitated by an electrical stirrer. Heating was continued until boilling. A vaccum was applied to the flask for a few seconds for expel the air bubbles. After disconnecting the vaccum, the starch solution was poured slowly into one end of the plates and immediately spread down the plates using a bevelled edged scraper which is as wide or wider than the gel plates. The excess starch solution was scraped away from the gels and the gels were allowed to set for half an hour in the cold room.

3.5.3. Insertion of the specimens

After the gels had set, slits were made with a Shandon comb across each gel about two inches from the cathodal end. The inserts were threads drawn from a well-washed sheet of cotton. The threads were held by forceps and soaked in the lysate samples. The moist threads were inserted into the slits.

3.5.4. Clelland's reagent

This reagent is required for old specimens only and is used to eliminate any storage effect so to produce clear electrophoretic patterns.

The clelland's reagent can be used to improve 6-PGD, ADA, ESD and AcP. This reagent should not be used for PGM and AK.

Preparation of the clelland's reagent:

75 mgs of Dithiothreitol was added to 10 mls of the app-

ropriate gel buffer,then was mixed and stored at 4°C. One drop of this solution was added to one drop of lysate in a disposable 10mmx64mm tube.This mixture was applied to the gel in the usual way.

3.6.Thin layer agarose gel electrophoresis

In the present investigation this method was used for typing of trnaferrin and the fhird component of human complement.

3.6.1. Apparatus used.

Electrophoresis on agarose gel was carried out in the horizontal position and the equipment consisted of:

1) A power pack capable of providing a constant current up to 50 mA and a constant voltage upto 500 volts.

2) A apparatus designed for thin layer electrophoresis(Behringwerke AG. Marburg.Lahn).This apparatus was chosen because of its effective cooling system consisting of a centrally placed,metal cooling block which is perfused with tapwater.The glass plates rest directly on this block.On each side of the cooling block are two buffer troughs.The lid of the apparatus has a circuit breaker which interrupts the current if the lid is opened during electrophoresis.

3)Platinum electrodes.

4)PH meter.

5)Gel plates: Glass plates with dimensions of 20 cmx20cmx2mm were used.

6)Plastic boxes for staining the gels.

3.6.2.Preparation of gel

For the preparation of the gel, agarose obtained from Behringwerke AG, Marburg Lahn,Germany(OP.No.1285)was used.For each gel,a suspension of 0.4 gm of agarose in 40 mls of gel buffer was heated over a boilling water bath in a 250 ml pyrex flask.Heating was continued until boiling.

The plates were covered with a 1.5 mm thick layer of the above mentioned 1% agarose gel. The gels were allowed to set for one hour in room temperature and then were stored in a airlight moist chamber in the cold room for 1-3 days before use.

3.6.3. Sample insertion

10-15 slots for application of serum were made by gently pressing the end of a 0.9 cm broad piece of whatman filter paper No.3 into the gels about 2 cm from the cathodal end of the plates. In each slot 5 microlitres of serum was applied.

3.7.Electrophoretic methods

The serum protein and red cell enzyme systems all require fairly strict control of electrophoretic method,PH,temperature, strength of buffer solution and purity of the ingredients used in the buffer and incubation mixtures.

Electrophoretic conditions should be designed to give optimum separation of isozymes without any loss of activity.

It is often necessary to put a sheet of glass over the gels to hold the wicks in place.It is adivisable also to lay a sheet of polythen over the gels for overnight runs to help prevent evaporation.

In the present investigation all electrophoretic runs were read by two persons.All discrepancies were re-run as a double check and any weakly reacting samples were also re-run using a thicker insert and if unreliable results were still obtained the sample was not included in the overall results.

3.7.1. Haptoglobin (HP)

The method used was that of smithies(1955),but using the modification of a discontinuous system of buffers as described by Poulik(1957).One drop of a 4 percent suspension of fresh haemoglobin is added to three drops of plasma and the resulting mixture so treated in inserted into the gel using a whatman No.3

filter paper insert. Horizontal thick layer starch gel elecro-phoresis was carried out at constant voltage(100 volts) and 20 mA for 16-17 hours(over night)at 4°c., or until the borate line has moved about 3 inches from the origin.The gels were run cathode to anode(-ve to +ve).

After electrophoresis the gels were sliced horizontally in half. Stain was applied(see details bellow)and the bands appeared within 2 to 3 minutes.

The buffer system was discontinuous and was made up as follow:

Gel buffer:

| | | |
|---|---|---|
| Tris | 9.196 | gms |
| Citric acid | 1.05 | gms |
| Distilled water | 1 | litre |

Adjusted to PH 8.65

Tank buffer:

| | | |
|---|---|---|
| NaOH | 8 | gms |
| Boric acid | 74.172 | gms |
| Distilled water | 4 | litres |

Adjusted to PH 8.5.

Staining:

Leuco-malachite green method:

| | | |
|---|---|---|
| Glacial acetic acid | 100 | mls |
| Distilled water | 150 | mls |
| Leuco-malachite green | 1 | gm |
| Zinc dust powdered | 1 | hand full |

The above ingredients were boiled in a one litre beaker until the green colour of the Leuco-malachite green disappeared. The mixture was then filtered to remove the solids and stored at 4°c. until required.The above mixture was poured over the sliced gels and left for 10 minutes; the excess stain was then poured off and 10 mls of 1/10 20 vols H202 was

poured on the gels. The bands then appeared after a few minutes.

3.7.2. Transferrin (Tf) and the third component of human complement (C3).

Tf and C3 typing was performed on the same gel because the electrophoretic conditons required are identical.

The method used was high voltage thin layer agarose gel electrophoresis described by Teisberg (1970).

5 microlitres of serum samples were applied in 10-15 slots on the gel plates.

Horizontal electrophoresis was carried out at constant voltage ( 20 v/cm) for 3-4 hours at room temperature. The voltage was measured by inserting two platinum wires connected to a voltmeter into the gel 15 cm appart.

A double layer of whatman filter paper No. 3 acted as connection between the buffer compartments on each side and betwe-en the two central buffer compartments on the gel.

The gels were run cathode to anode (-ve to +ve).

The buffers used were as follows:

Gel buffer: barbital/calcium lactate buffer

| | |
|---|---|
| Natrium barbital | 11.855 gms |
| Barbital acid | 1.704 gms |
| Calcium lactate | 0.693 gm |
| Distilled water | 2500 mls |

Adjusted to PH 8.6.

Tank buffer: barbital/calcium lactate buffer

| | |
|---|---|
| Natrium barbital | 31.443 gms |
| Barbital acid | 4.881 gms |
| Calcium lactate | 1.837 gms |
| Distilled water | 2500 mls |

Adjusted to PH 8.6

After electrophoresis, the gels were fixed in a 4%(v/v) solution of acetic acid, washed in distilled water and dried under filter paper at room temperature for 12-18 hours.

Staining:

Staining was performed with Amidoshwarz 10B obtained from E. Merck AG, Darmstadt, Germany.

A 1-2% solution of amidoshwarz was made up in methanol, acetic acid and water mixture. This was poured on the gels and left for 10 secs. Then rinsed off with tapwater and placed in methanol, acetic acid, water mixture.

This mixture was made up in the following ratios:

| | |
|---|---|
| Methanol | 6 parts |
| Acetic acid | 1 part |
| Water | 4; parts |

The gels were allowed to stand in this mixture until amidoshwarz was cleared and the transferrin and C3 bands were visible. C3 bands could be seen about 2-3 cm and those of Tf about8-10 cm anodal from the application slots.

3.7.3. Acid phosphatase (AcP)

The method used was that outlined by Hopkinson et al (1963) with slight modification.

All the lysates had clleland's reagent mixed with them, volume for volume, and the resulting mixture was soaked onto threads of cotton which acted as the inserts. Horizontal thin layer starch gel electrophoresis was performed at constant voltage (105 Volts) and low amperage (5mA) for 16-17 hours at $4^{o}$c. The gels were run cathode to anode(-ve to +ve). The acid phosphatase bands were then demonstrated by staining with a reac-

tion mixture containing 4- methylumbelliferyl phosphate soacted into No. 3 whatman filter paper which was placed on the gel surface for 15 minutes at 37°c. and the resulting bands could then be read under u.v. light.

The buffers used were as follows:

Tank buffer:

| | | |
|---|---|---|
| Natrium dehydrogen phosphate (Na H2 Po4, 2 H20) | 152.8 | gms |
| Tri-Natrium citrate (Na 3 C6H507, 2H20) | 128.4 | gms |
| Distilled water | 4 | litres |

Adjusted to PH 6.3 with NaoH.

Gel buffer:

The tank buffer was diluted 1 in 40 and adjusted to PH 6.3 with 0.2 M citric acid.

Incubation buffer:

| | | |
|---|---|---|
| Citric acid | 105.074 | gms |
| Distilled water | 1 | litre |

Adjusted to PH 6.0 with 0.05 M NaOH.

Staining (per gel):

| | | |
|---|---|---|
| 4-methylumbelliferyl phosphate | 8 | mgs |
| Incubation buffer | 10 | mls |

The stain was applied on the gels using whatman filter paper overlay. The gels were then incubated at 37°C. for approximately 15 minutes and then read under U.V light.

3.7.4. Adenylate Kinase(AK)

The method used was that described by Fildes and Harris (1966) with slight modification.

Horizontal thin layer starch gel electrophoresis was carried out at constant voltage (100 volts) and low amperage (5 mA)

for 16-17 hours (over night).

The gels were run anode to cathode(+ve to -ve). Clelland's reagent should not be added to the specimens for AK's.

Lysates were soaked onto threads of cotton which acted as the inserts.

The gels were stained using an agar overlay, and when the agar has set the gels were placed in an 37°c oven, electrophoretic bands appearing after about 30 minutes.

The buffers used were as follows:

Gel buffer:

| | | |
|---|---|---|
| Succinic acid | 1.89 | gms |
| Tris | 2.226 | gms |
| Distilled water | 1 | litre |
| Adjusted to PH 5.0 | | |

Tank buffer:

| | | |
|---|---|---|
| Citric acid | 114.88 | gms |
| NaoH | 42.7464 | gms |
| Distilled water | 4 | litres |
| Adjusted to PH 4.9 | | |

Incubation buffer:

| | | |
|---|---|---|
| Tris | 12.12 | gms |
| Distilled water | 1 | litre |
| Adjusted PH to 8.0 using concentrated Hcl. | | |

Staining (Mixture per gel):

| | | |
|---|---|---|
| 2% Agar solution (in H20) | 10 | mls |
| Incubation buffer | 10 | mls |
| Glucose | 18 | mgs |
| Mgcl2 | 40 | mgs |
| A.D.P. | 4.9 | mgs |
| N.A.D.P. | 3.1 | mgs |

| | | |
|---|---|---|
| M.T.T. | 2.5. | mgs |
| P.M.S. | 2.5 | mgs |
| G6PDH | 20 | microlitres |
| Hexokinase | 20 | microlitres |

The ingredients were mixed in the incubation buffer and placed in the 37$^{o}$ oven. The agar was boiled, and then cooled. When the agar solution was hand hot ( approximately 55-60$^{o}$c ) the incubation buffer was mixed in and this was poured in the previously placed former. When the agar was set, the gels were placed in the 37$^{o}$ c oven. Bands appeared after 30 minutes.

### 3.7.5. Phosphoglucomutase ($PGM_1$).

The method used was that described by Spencer et al (1964), with slight modification.

Horizontal thin layer starch-gel electrophoresis was carried out at constant voltage (110.V) and 5-8 mA for 16-17 hours at 4$^{o}$c.

The gels were run cathode to anode(-ve to +ve). Clleland's reagent should not be added to the specimens for PGM's.

The haemolysates were soaked onto threads of cotton which acted as the inserts.

The buffers used were as follows:

Tank buffer:

| | | |
|---|---|---|
| Tris | 48.44 | gms |
| Maleic acid | 46.48 | gms |
| EDTA( acid ) | 11.68 | gms |
| Mgcl2 | 8.12 | gms |
| Distilled water | 4 | litres |

Adjusted to PH 7.4 with 40% NaoH.

Gel buffer:

The tank buffer was diluted 1 in 15 with distilled water.

Incubation buffer

| | | |
|---|---|---|
| Tris | 7.28 | gms |
| Distilled water | 1 | litre |

Adjusted to PH 8.0 with concentrated Hcl.

Staining (Mixture per gel):

| | | |
|---|---|---|
| 2% Agar solution (in H20) | 10 | mls |
| Incubation buffer | 10 | mls |
| Mgcl2 | 20 | mgs |
| G-1-phosphate (containing G-1:6 diphosphate) | 30 | mgs |
| N.A.D.P. | 1.5 | mgs |
| M.T.T. | 2.5 | mgs |
| P.M.S. | 2.5 | mgs |
| G-6PDH | 20 | microlitres |

The ingredients were mixed in the incubation buffer and placed in the 37°c oven. The agar was boiled, and then cooled. When the agar solution was hand hot (55-60°c) the incubation buffer was mixed in and this was poured in the previously placed former. When the agar was set the gels were placed in the 37°c oven.

PGM, electrophoretic bands appeared after 30-60 minutes.

3.7.6. Adenosine deaminase (ADA)

The method used was that outlined by Spencer et al (1968) with slight modification.

All the lysates had cleland's reagent mixed with them, volume for volume, and the resulting mixture was soaked onto threads of cotton which acted as the inserts. Horizontal thin layer starch gel electrophoresis was performed at constant voltage (60volts) and low amperage (5mA) for 17 hours at 4°c.

The gels were run cathode to anode (-ve to +ve).

The buffers used were as follows:

Tank buffer:

NaH2P04 46.802 gms

Solution A:

Distilled water 3. litres

Na2HP04 19.874 gms

Solution B:

Distilled water 1.4 litres.

Solution A was added to solution B in order to be adjusted to PH 6.5.

Gel buffer:

The tank buffer was diluted 1 in 10 with distilled water.

Incubation buffer:

NaH2P04 0.195 gm

Solution A:

Distilled water 50 mls

Na2HP04 0.7098 gm

Solution B:

Distilled water 200 mls

Solution A was added to solution B in order to be adjusted to PH 7.5. This solution was then diluted 1 in 4 for use.

Staining ( Mixture per gel):

| | | |
|---|---|---|
| 2% Agar solution (in H20) | 10 | mls |
| Incubation buffer | 10 | mls |
| Adenosine | 15 | mgs |
| M.T.T. | 2 | mgs |
| P.M.S. | 2 | mgs |
| Xanthine oxidase | 10 | microlitres |
| Nucleoside phosphorylase | 10 | microlitres |

The ingredients were mixed in the incubation buffer and placed in the $37^{o}$c oven. The agar was boiled and then cooled.

When the agar solution was hand hot ( 55-60°c) the incubation buffer was mixed in and this was poured in the previously placed former. When the agar was set the gels were placed in the 37°c oven.

ADA electrophoretic bands appeared after 30 minutes.

### 3.7.7. Esterase D(EsD)

The method used was that outlined by Koster et al (1975), with slight modification.

All the lysates had clelland's reagent mixed with them, volume for volume, and the resulting mixture was soaked onto threads of cotton which acted as the inserts. Horizontal thin layer starch gel electrophoresis was performed at constant voltage (300 volts) and low amperage (5 mA), for 2 hours at 4°C. The gels were run negative to positive (-ve to +ve). Th esterase D bands were then demonstrated by staining with a reaction mixture containing 4 - methylumbelliferyl acetate( as described by Hopkinson et al, 1973) soacted into No.3 whatman filter paper which was placed on the gel surface for 5 minutes at 37°c. and the resulting bands could then be read under u.v. light.

The buffers used were as follows:

Gel buffer:

| | | |
|---|---|---|
| Tris | 1.636 | gms |
| Citric acid | 0.756 | gm |
| Boric acid | 0.272 | gm |
| Lithium hydroxide | 0.016 | gm |
| Distilled water | 1 | litre |

Adjusted to PH 7.4.

The gel buffer was diluted volume for volume with distilled water for use.

Tank buffer:

| | | |
|---|---|---|
| Boric acid | 108.84 | gms |
| Lithium hydroxide | 6.72 | gms |
| Distilled water | 4 | litres |

Adjusted to PH 7.2

Staining (per gel):

1- 41 mgs anhydrous sodium acetate was added to 10 mls distilled water and adjusted to PH 6.9.

2- 4 mgs 4-methylumbelliferyl acetate was mixed with a few drops of aceton.

3- Solutions 1 and 2 were mixed for incubation buffer and placed on 3 mm strips on each gel. The gels were incubated at 37°c. for 5 minutes and then were read under U.V. light.

3.7.8. 6- Phosphogluconate dehydrogenase (6-PGD)

The method used was that described by Hopkinson et al (1976), with slight modification.

All the lysates had clleland's reagent mixed with them volume for volume, and the resulting mixture was soaked onto threads of cotton which acted as the inserts. Horizontal thin layer starch gel electrophoresis was performed at constant voltage (100 volts ) and low amperage (5 mA) for 17 hours at 4°c. The gels were run cathode to anode (-ve to +ve).

The buffers used were as follows:

Tank buffer:

| | | |
|---|---|---|
| NaH2PO4 | 46.802 | gms |

Solution A:

| | | |
|---|---|---|
| Distilled water | 3 | litres |
| Na2HPO4 | 19.874 | gms |

Solution B:

| | | |
|---|---|---|
| Distilled water | 1.4 | litres |

Solution A was added to solution B in order to be adjusted to PH 7.0 and then 10 mgs NADP was added to each 500 mls of tank buffer.

Gel buffer:

The tank buffer was diluted 1 in 10 and then 4 mgs NADP was added to each 200 mls of gel buffer.

Incubation buffer:

| | | |
|---|---|---|
| Tris | 15.14 | gms |
| Distilled water | 250 | mls |

Adjusted to PH 8.0 with concentrated Hcl.

Staining ( Mixture per gel):

| | | |
|---|---|---|
| 2% Agar solution (in H20) | 10 | mls |
| Incubation buffer | 10 | mls |
| 6-Phosphogluconate | 10 | mgs |
| Mgcl2 | 0.2 | gm |
| NADP | 5 | mgs |
| M.T.T. | 5 | mgs |
| P.M.S. | 5 | mgs |

The ingredients were mixed in the incubation buffer and placed in the 37$^{o}$c oven. The agar was boiled and then cooled. When the agar solution was hand hot (55-60$^{o}$c) the incubation buffer was mixed in and this was poured into the previously placed formers. 6PGD, electrophoretic bands appeared after 30 minutes.

# CHAPTER 4. RESULTS

## 4.1. Blood group antigens

### 4.1.1. The ABO blood group system

The distribution of the ABO blood groups and respective gene frequencies in the ten Iranian samples, expressed in term of four phenotypes after testing with two antisera, is demonstrated in Table 4.1.1.a. There was good agreement between the observed and expected phenotypic values in all samples, thus confirming the assumption of Hardy-weinberg equilibrium and that they are random mating populations.

The frequency of the A gene ranges from 19.00 percent in the Baluchis of Sistan and Baluchistan to 27.46 percent in the Turks of Rezaieh; that of the B gene from 11.97 percent in the Kurds of Rezaieh to 22.99 percent in the Kermani series. The 0 gene frequency varies between 56.13 percent in the Turks of Shirvan, Khorasan and 65.33 percent in the Lurs of Luristan.

Table 4.1.1.b shows the distribution of the $A_1$ $A_2$ BO system in six out of the ten Iranian samples, expressed in term of six phenotypes after testing with three antisera.

Close agreement was found between the observed and expected phenotypic values which confirms the assumption of Hardy-weinberg equilibrium.

The frequency of the allele $A_2$ ranges from 0.56 percent in the Baluchis of Sistan and Baluchistan to 7.28 percent in the Kurds of Rezaieh and that of the $A_1$ gene from 15.25 percent in the Zoroastrians to 24.38 percent in the Turks of Rezaieh.

### 4.1.2. The Rhesus blood group system

Table 4.1.2.a illustrates the distribution of Rh types and respective gene frequencies in the ten Iranian samples, expressed in term of two phenotypes after testing with one

antiserum.

The frequency of the gene d ranges from 22.36 percent in the Turks of Shirvan, Khorasan to 39.92 percent in the Kurds of Rezaieh.

The distribution of Rh types and respective gene complex frequencies in three out of the ten Iranian samples is shown in Table 4.1.2. b. The specimens were tested with the following antisera: anti-C, anti-D, anti-E, anti-c, and anti-e. There was good agreement between the observed and expected phenotypic values in all samples, thus confirming the assumption of Hardy-weinberg equilibrium. The frequencies of the principal Rh complexes are as follows:

CDe($R_1$) ranges from 47.95 percent in the Lurs of Luristan to 59.63 percent in the Baluchis of Sistan and Baluchistan.

cde(r) varies between 21.81 percent in the Baluchis of Sistan and Baluchistan and 25.80 percent in the Zabolis of the same area.

cDE($R_2$) ranges from 14.29 percent in the Baluchis of Sistan and Baluchistan to 22.65 percent in the Lurs of Luristan.

cDe(Ro) varies between Zero in the Zabolis of Sistan and Baluchistan and 3.52 percent in the Lurs of Luristan.

4.1.3. The MNSs blood group system

The distribution of the MNSs blood groups and respective gene combination frequencies, expressed in term of nine phenotypes after testing with four antisera, in six out of the ten Iranian samples is presented in Table 4.1.3. close agreement was found between the observed and expected phenotypic values in all samples which confirms the assumption of Hardy-weinberg equilibrium and that they are random mating populations.

The frequencies of the MNSs gene complexes are as follows:

MS ranges from 11.99 percent in the Kurds of Rezaieh to 48.95 percent in the Baluchis of Sistan and Baluchistan.

Ms varies between 26.28 percent in the Baluchis of Sistan and Baluchistan and 42.80 percent in the Zoroastrians.

NS ranges from 7.02 percent in the Lurs of Luristan to 15.73 percent in the Turks of Rezaieh.

Ns varies between 17.69 percent in the Baluchis of Sistan and Baluchistan and 40.18 percent in the Kurds of Rezaieh.

4.1.4.The P blood group system

Table 4.1.4. illustrates the distribution of the P blood groups and respective gene frequencies in three out of the ten Iranian samples.

The frequency of the $P_1$ gene ranges from 23.27 percent in the Turks of Rezaieh to 31.90 percent in the Kurds of the same region.

4.1.5. The Kell blood group system

Table 4.1.5.shows the distribution of the Kell blood groups and respective gene frequencies in six out of the ten Iranian samples.

The frequency of the K gene ranges from 0.46 percent in the Baluchis of Sistan and Baluchistan to 3.41 percent in the Lurs of Luristan.

4.1.6.The Duffy blood group system

The distribution of the Duffy blood groups and respective gene frequencies in six out of the ten Iranian samples is presented in Table 4.1.6.

The frequency of the $Fy^a$ gene varies between 33.10 percent in Lurs of Luristan and 45.23 percent in the Turks of Rezaieh.

4.1.7.The Kidd blood group system

Table 4.1.7. exhibits the distribution of the Kidd blood

groups and respective gene frequencies in six out of the ten Iranian samples.

The frequency of the $JK^a$ gene ranges from 25.52 percent in the Lurs of Luristan to 45.02 percent in the Zoroastrians.

## 4.2. Serum proteins

### 4.2.1. The haptoglobin (HP) system

The distribution of haptoglobin types and respective gene frequencies in the ten Iranian samples is shown in Table 4.2.1. The gene frequencies were calculated excluding the phenotype HP0-0. All the samples showed close agreement between the observed and expected phenotypic values, thus confirming the assumption of Hardy-weinberg equilibrium. NO HP2-1 ( modified ) or other rarer phenotype was detected.

The frequency of the $HP^1$ gene varies between 21.79 percent in the Zabolis of Sistan and Baluchistan and 30.71 percent in the Kurds of Rezaieh.

### 4.2.2. The transferrin (Tf) system

Table 4.2.2. presents the distribution of transferrin types and respective gene frequencies in the ten Iranian samples. Seven samples, out of ten, exhibited only the type CC. A single Zoroastrian and a single individual from Kerman were typed as CD.

In the Kurdish sample from Shirvan, Khorasan one CD and one CB were typed.

The subtyping of the variants was not performed.

### 4.2.3. The third component of human complement (C3) system

The distribution of C3 types and respective gene frequencies in the ten Iranian samples is shown in Table 4.2.3. The observed numbers were in close agreement with those expected, assuming Hardy-weinberg equilibrium. No rare variant was detected in the present investigation.

The frequency of the $C^F3$ gene ranges from 5.78 percent in the Kermani series to 22.43 percent in the Baluchis of Sistan and Baluchistan.

## 4.3. Red cell enzymes

### 4.3.1. The acid phosphatase (AcP) system

The distribution of acid phosphatase types and respective gene frequencies in the ten Iranian samples is given in Table 4.3.1. Close agreement, calculated on the basis of Hardy-weinberg equilibrium, was found between the observed and expected phenotypic values in all the samples.

No rarer AcP phenotype was detected in the present investigation. All the samples showed an absence of the CC phenotype. The frequency of the gene $AcP^A$ ranges from 21.01 percent in the Zoroastrians to 36.99 percent in the Kurds of Rezaieh, that of the $AcP^B$ allele varies between 63.01 percent in the Kurds of Rezaieh and 77.73 percent in the Zoroastrians. The frequency of the rarest allele $AcP^C$ ranges from Zero both in the Turks and the Kurds of Rezaieh to 1.96 percent in the Lurs of Luristan.

### 4.3.2. The adenylate Kinase (AK) system

Table 4.3.2. shows the distribution of adenylate Kinase types and respective gene frequencies in the ten Iranian samples. All the samples showed close agreement between the observed and expected phenotypic values which confirms the assumption of Hardy-weinberg equilibrium and that they are random mating populations. No rare AK phenotypes were discovered in any of the samples examined.

The frequency of the $AK^2$ gene ranges from 1.38 percent in the Kurds of Rezaieh to 10.81 percent in the Baluchis of Sistan and Baluchistan.

## 4.3.3. The Phosphoglucomutase Locus 1( $PGM_1$ ) system

The frequencies of $PGM_1$ types and respective genes in the ten Iranian samples are given in Table 4.3.3.
Close agreement, calculated on the basis of Hardy-weinberg equilibrium, was found between the observed and expected phenotypic values.
No rare variant was detected in the present investigation.
The frequency of the $PGM_1^2$ gene ranges from 23.08 percent in the Zoroastrians to 43.56 percent in the Turks of Rezaieh.

## 4.3.4. The adenosine deaminase (ADA) system

Table 4.3.4 exhibits the frequencies of ADA types and respective genes in five out of the ten Iranian samples.
Good agreement was found between the observed and expected phenotypic values which confirms the assumption of Hardy-weinberg equilibrium.
The rarer $ADA^4$ and $ADA^6$ alleles were found only in the Turkish series of Rezaieh.
The frequency of the $ADA^2$ gene varies between 13.48 percent in the Turks of Rezaieh and 19.56 percent in the Zoroastrians.

## 4.3.5. The esterase D (EsD) system

The distribution of EsD types and respective gene frequencies in the ten Iranian samples is presented in Table 4.3.5.
There was good agreement between the observed and expected phenotypic values in all the samples, thus confirming the assumption of Hardy-weinberg equilibrium and that they are random mating populations.
No phenotypes other than common 1-1, 2-1 and 2-2 types were found in the present investigation.
The frequency of the $EsD^2$ gene ranges from 12.61 percent in the Turks of Shirvan, Khorasan to 29.55 percent in the Baluchis

of Sistan and Baluchistan.

4.3.6. The 6-phosphogluconate dehydrogenase (6-PGD) system

The frequncies of 6-PGD types and respective genes in five out of the ten Iranian samples are set out in Table 4.3.6. Good agreement was found between the observed and expected phenotypic values confirming the assumption of Hardy-weinberg equilibrium and that they are random mating populations. With the exception of the only CC phenotype which was found in the 301 blood specimens from Kerman, no variant other than the 'common' CA type was found in the present investigation. The frequency of the $PGD^{C}$ allele ranges from zero in the Zoroastrians to 6.43 percent in the Kurds of Shirvan, Khorasan.

Table 4.1.1.a ABO blood groups distribution in Iran tested with anti-A, and Anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | A | B | AB | O | A | B | O | $X^2$ |
| 1-Kurds.Rezaieh | 138 | obs. | 50 | 24 | 7 | 57 | 23.43 | 11.97 | 64.60 | 0.11 |
| | | Exp. | 49.36 | 23.32 | 7.74 | 57.58 | | | | |
| 2-Lurs.Luristan | 167 | obs. | 56 | 31 | 9 | 71 | 21.87 | 12.80 | 65.33 | 0.02 |
| | | Exp. | 55.69 | 30.68 | 9.35 | 71.28 | | | | |
| 3-Turks.Rezaieh | 142 | obs. | 58 | 27 | 9 | 48 | 27.46 | 13.66 | 58.88 | 0.41 |
| | | Exp. | 56.61 | 25.50 | 10.65 | 49.23 | | | | |
| 4-Tehran | 328 | obs. | 119 | 71 | 28 | 110 | 25.63 | 16.44 | 57.93 | 0.01 |
| | | Exp. | 118.93 | 71.33 | 27.63 | 110.10 | | | | |
| 5-Baluchis.Sistan & Baluchistan | 111 | obs. | 32 | 30 | 6 | 43 | 19.00 | 17.90 | 63.10 | 0.48 |
| | | Exp. | 30.63 | 28.62 | 7.55 | 44.20 | | | | |
| 6-Kurds.Shirvan Khorasan | 142 | obs. | 52 | 38 | 9 | 43 | 24.75 | 18.42 | 56.83 | 1.96 |
| | | Exp. | 48.65 | 34.54 | 12.95 | 45.86 | | | | |

Table 4.1.1.a (Cont) ABO Blood groups distribution in Iran tested with anti- A, and Anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | | A | B | AB | O | A | B | O | $X^2$ |
| 7-Zabolish.Sistan & Baluchistan | 116 | obs. | 29 | 26 | 14 | 47 | 20.32 | 18.73 | 60.95 | 4.67 |
| | | Exp. | 33.52 | 30.56 | 8.83 | 43.09 | | | | |
| 8-Turks.Shirvan Khorasan | 160 | obs. | 53 | 40 | 16 | 51 | 24.53 | 19.34 | 56.13 | 0.07 |
| | | Exp. | 53.70 | 40.71 | 15.18 | 50.41 | | | | |
| 9-Zoroastrians | 88 | obs. | 20 | 25 | 10 | 33 | 18.59 | 22.12 | 59.29 | 1.67 |
| | | Exp. | 22.43 | 27.39 | 7.24 | 30.94 | | | | |
| 10-Kerman | 317 | obs. | 82 | 101 | 28 | 106 | 19.19 | 22.99 | 57.82 | 0.00 |
| | | Exp. | 82.03 | 101.03 | 27.97 | 105.98 | | | | |

Table 4.1.1.b ABO blood groups distribution in Iran tested with anti-A, anti-B, and anti-$A_1$ sera

| Population | Number Tested | | Phenotypes | | | | | | Gene frequencies | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | $A_1$ | $A_2$ | B | $A_1B$ | $A_2B$ | 0 | $A_1$ | $A_2$ | B | 0 | $X^2$ |
| 1-Baluchis.Sistan & Baluchistan | 111 | obs | 32 | 0 | 30 | 5 | 1 | 43 | 18.50 | 0.56 | 17.89 | 63.05 | 4.49 |
| | | Exp. | 29.92 | 0.79 | 28.60 | 7.35 | 0.22 | 44.12 | | | | | |
| 2-Zabolis.Sistan & Baluchistan | 116 | obs. | 28 | 1 | 26 | 13 | 1 | 47 | 19.30 | 1.04 | 18.73 | 60.93 | 4.91 |
| | | Exp. | 32.06 | 1.48 | 30.55 | 8.39 | 0.45 | 43.06 | | | | | |
| 3-Zoroastrians | 88 | obs. | 17 | 3 | 25 | 8 | 2 | 33 | 15.25 | 3.35 | 22.12 | 59.28 | 1.72 |
| | | Exp. | 18.86 | 3.59 | 27.38 | 5.94 | 1.30 | 30.92 | | | | | |
| 4- Turks.Rezaieh | 92 | obs. | 36 | 5 | 18 | 3 | 1 | 29 | 24.38 | 4.55 | 12.94 | 58.13 | 2.12 |
| | | Exp. | 33.59 | 5.06 | 15.38 | 5.80 | 1.08 | 31.09 | | | | | |
| 5- Lurs.Luristan | 167 | obs. | 47 | 9 | 31 | 5 | 4 | 71 | 17.10 | 4.86 | 12.80 | 65.24 | 2.97 |
| | | Exp. | 44.91 | 10.98 | 30.64 | 7.31 | 2.08 | 71.07 | | | | | |
| 6- Kurds.Rezaieh | 138 | obs. | 38 | 12 | 24 | 3 | 4 | 57 | 16.26 | 7.28 | 11.97 | 64.49 | 2.47 |
| | | Exp. | 35.85 | 13.70 | 23.28 | 5.37 | 2.41 | 57.39 | | | | | |

Table 4.1.2.a Rh blood groups distribution in Iran tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | |
|---|---|---|---|---|---|
| | | D+ | D- | D | d |
| 1-Turks.Shirvan Khorasan | 160 | 152 | 8 | 77.64 | 22.36 |
| 2-Kerman | 317 | 301 | 16 | 77.53 | 22.47 |
| 3-Lurs.Luristan | 162 | 152 | 10 | 75.16 | 24.84 |
| 4-Baluchis.Sistan & Baluchistan | 111 | 104 | 7 | 74.89 | 25.11 |
| 5-Kurds.Shirvan Khorasan | 142 | 132 | 10 | 73.47 | 26.53 |
| 6-Zabolis.Sistan & Baluchistan | 115 | 104 | 11 | 69.07 | 30.93 |
| 7-Tehran | 302 | 268 | 34 | 66.40 | 33.60 |
| 8-Turks.Rezaieh | 141 | 124 | 17 | 65.27 | 34.73 |
| 9-Zoroastrians | 88 | 77 | 11 | 64.64 | 35.36 |
| 10-Kurds.Rezaieh | 138 | 116 | 22 | 60.08 | 39.92 |

Table 4.1.2.b. Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | CCDEE | CCDEe | CCDee | CCdEE | CCdEe | CCdee | CcDEE | CcDEe | CcDee | CcdEE | CcdEe | Ccdee | ccDEE | ccDEe | ccDee | ccdEE | ccdEe | ccdee |
| 1-Baluchis.Sistan & Baluchistan | 111 | obs. | 0 | 0 | 44 | 0 | 0 | 0 | 1 | 18 | 30 | 0 | 0 | 1 | 3 | 7 | 1 | 0 | 0 | 0 |
| | | Exp. | 0 | 0.78 | 42.06 | 0 | 0 | 0.04 | 0.18 | 19.84 | 31.24 | 0 | 0 | 0.95 | 2.27 | 7.47 | 0.87 | 0 | 0 | 5.28 |
| 2-Lurs.Luristan | 162 | obs. | 0 | 0 | 40 | 0 | 0 | 0 | 2 | 30 | 46 | 0 | 0 | 0 | 11 | 20 | 3 | 0 | 0 | 10 |
| | | Exp. | 0.01 | 1.26 | 37.25 | 0 | 0 | 0 | 0.59 | 35.94 | 44.42 | 0 | 0 | 0 | 8.31 | 20.98 | 3.06 | 0 | 0 | 10.18 |
| 3-Zabolis.Sistan & Baluchistan | 115 | obs. | 0 | 0 | 40 | 0 | 0 | 1 | 2 | 13 | 32 | 0 | 0 | 2 | 8 | 9 | 0 | 0 | 0 | 8 |
| | | Exp. | 0.01 | 1.31 | 35.67 | 0 | 0 | 0.31 | 0.40 | 22.79 | 30.10 | 0 | 0 | 3.10 | 3.42 | 10.23 | 0 | 0 | 0 | 7.66 |

| Population | Number Tested | Gene complex frequencies | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | $X^2$ |
| 1-Baluchis.Sistan & Baluchistan | 111 | 0.57 | 59.63 | 0.00 | 1.96 | 14.29 | 1.74 | 0.00 | 21.81 | 5.21 |
| 2-Lurs.Luristan | 162 | 0.81 | 47.95 | 0.00 | 0.00 | 22.65 | 3.52 | 0.00 | 25.07 | 6.75 |
| 3-Zabolis.Sistan & Baluchistan | 115 | 1.02 | 50.71 | 0.00 | 5.22 | 17.24 | 0.00 | 0.00 | 25.80 | 20.67 |

Table 4.1.3 MNSs blood groups distribution in Iran tested with anti-M, anti-N, anti-S, and anti-s sera

| Pupolation | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | MMSS | MMSs | MMss | MNSS | MNSs | MNss | NNSS | NNSs | NNss |
| 1-Kurds.Rezaieh | 124 | obs. 2 | 11 | 18 | 2 | 19 | 40 | 2 | 9 | 21 |
| | | Exp. 1.29 | 9.97 | 19.25 | 2.62 | 20.25 | 39.12 | 1.33 | 10.29 | 19.88 |
| 2-Turks.Rezaieh | 139 | obs. 4 | 19 | 26 | 7 | 28 | 30 | 4 | 11 | 10 |
| | | Exp. 4.79 | 20.68 | 22.32 | 6.76 | 29.18 | 31.49 | 2.38 | 10.29 | 11.11 |
| 3-Zoroastrians | 74 | obs. 2 | 12 | 13 | 4 | 16 | 16 | 2 | 4 | 5 |
| | | Exp. 3.00 | 12.48 | 13.00 | 3.86 | 16.09 | 16.75 | 1.24 | 5.18 | 5.40 |
| 4-Zabolis.Sistan & Baluchistan | 114 | obs. 6 | 28 | 18 | 4 | 18 | 23 | 2 | 4 | 11 |
| | | Exp. 5.13 | 21.34 | 22.21 | 5.44 | 22.63 | 23.56 | 1.44 | 6.00 | 6.25 |
| 5-Lurs.Luristan | 153 | obs. 22 | 41 | 20 | 9 | 30 | 15 | 1 | 6 | 9 |
| | | Exp. 16.80 | 39.31 | 22.99 | 13.13 | 30.72 | 17.97 | 2.57 | 6.00 | 3.51 |
| 6-Baluchis.Sistan & Baluchistan | 111 | obs. 27 | 28 | 8 | 10 | 22 | 9 | 1 | 2 | 4 |
| | | Exp. 20.89 | 30.67 | 11.26 | 13.75 | 20.20 | 7.42 | 2.26 | 3.33 | 1.22 |

Table 4.1.3 (Cont) MNSs blood groups distribution in Iran tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Gene complex frequencies | | | | |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | $X^2$ |
| 1-Kurds.Rezaieh | 124 | 11.99 | 37.60 | 10.23 | 40.18 | 1.39 |
| 2-Turks.Rezaieh | 139 | 16.15 | 42.48 | 15.73 | 25.64 | 2.27 |
| 3-Zoroastrians | 74 | 18.01 | 42.80 | 14.24 | 24.95 | 1.33 |
| 4-Zabolis-Sistan & Baluchistan | 114 | 24.62 | 40.73 | 7.84 | 26.81 | 8.87 |
| 5-Lurs.Luristan | 153 | 36.81 | 35.08 | 7.02 | 21.09 | 13.43 |
| 6-Baluchis.Sistan & Baluchistan | 111 | 48.95 | 26.28 | 7.08 | 17.69 | 12.04 |

Table 4.1.4. P blood groups distribution in Iran tested with anti-$P_1$ serum

| Population | Number Tested | Phenotypes | | Gene frequencies | |
|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2+P$ |
| 1-Turks.Rezaieh | 141 | 58 | 83 | 23.27 | 76.73 |
| 2-Zoroastrians | 86 | 41 | 45 | 27.66 | 72.34 |
| 3-Kurds.Rezaieh | 138 | 74 | 64 | 31.90 | 68.10 |

Table 4.1.5. Kell blood groups distribution in Iran tested with anti- K serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | |
|---|---|---|---|---|---|
| | | K(+) | K(-) | K | k |
| 1-Baluchis.Sistan & Baluchistan | 110 | 1 | 109 | 0.46 | 99.54 |
| 2-Zoroastrians | 86 | 1 | 85 | 0.58 | 99.42 |
| 3-Kurds.Rezaieh | 138 | 3 | 135 | 1.09 | 98.91 |
| 4-Zabolis.Sistan & Baluchistan | 115 | 3 | 112 | 1.31 | 98.69 |
| 5-Turks.Rezaieh | 141 | 7 | 134 | 2.51 | 97.49 |
| 6-Lurs.Luristan | 149 | 10 | 139 | 3.41 | 96.59 |

Table 4.1.6 Duffy blood groups distribution in Iran tested with anti-$Fy^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | |
|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a-) | $Fy^a$ | $Fy^b$+ Fy |
| 1-Lurs.Luristan | 143 | 79 | 64 | 33.10 | 66.90 |
| 2-Baluchis.Sistan & Baluchistan | 47 | 28 | 19 | 36.42 | 63.58 |
| 3-Zoroastrians | 86 | 52 | 34 | 37.12 | 62.88 |
| 4-Zabolis.Sistan & Baluchistan | 115 | 70 | 45 | 37.45 | 62.55 |
| 5-Kurds.Rezaieh | 138 | 89 | 49 | 40.41 | 59.59 |
| 6-Turks.Rezaieh | 140 | 98 | 42 | 45.23 | 54.77 |

Table 4.1.7 Kidd blood groups distribution in Iran tested with anti-$JK^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | |
|---|---|---|---|---|---|
| | | JK(a+ ) | JK(a-) | $JK^a$ | $Jk^b$+JK |
| 1-Lurs.Luristan | 146 | 65 | 81 | 25.52 | 74.48 |
| 2-Kurds.Rezaieh | 136 | 69 | 67 | 29.81 | 70.19 |
| 3-Baluchis.Sistan & Baluchistan | 111 | 58 | 53 | 30.90 | 69.10 |
| 4-Turks.Rezaieh | 141 | 74 | 67 | 31.07 | 68.93 |
| 5-Zabolis .Sistan & Baluchistan | 115 | 70 | 45 | 37.45 | 62.55 |
| 6-Zoroastrians | 86 | 60 | 26 | 45.02 | 54.98 |

Table 4.2.1. Haptoglobin (HP) Phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | | Phenotypes HP 1-1 | HP 2-1 | HP 2-2 | HP 0 | Gene frequencies $HP^1$ | $HP^2$ | $X^2$ |
|---|---|---|---|---|---|---|---|---|---|
| 1-Zabolis.Sistan & Baluchistan | 118 | obs. | 3 | 45 | 69 | 1 | 21.79 | 78.21 | 1.92 |
| | | Exp. | 5.56 | 39.88 | 71.56 | | | | |
| 2-Lurs.Luristan | 178 | obs. | 10 | 61 | 106 | 1 | 22.88 | 77.12 | 0.10 |
| | | Exp. | 9.27 | 62.47 | 105.27 | | | | |
| 3-Kurds.Shirvan Khorasan | 102 | obs. | 4 | 39 | 58 | 1 | 23.27 | 76.73 | 0.67 |
| | | Exp. | 5.47 | 36.06 | 59.47 | | | | |
| 4-Turks.Shirvan, Khorasan | 106 | obs. | 3 | 43 | 57 | 3 | 23.79 | 76.21 | 2.36 |
| | | Exp. | 5.83 | 37.34 | 59.83 | | | | |
| 5-Baluchis.Sistan & Baluchistan | 111 | obs. | 5 | 43 | 59 | 4 | 24.77 | 75.23 | 0.66 |
| | | Exp. | 6.56 | 39.87 | 60.56 | | | | |
| 6-Turks.Rezaieh | 145 | obs. | 11 | 51 | 79 | 4 | 25.89 | 74.11 | 0.46 |
| | | Exp. | 9.45 | 54.10 | 77.45 | | | | |
| 7-Zoroastrians | 171 | obs. | 15 | 56 | 95 | 5 | 25.90 | 74.10 | 2.44 |
| | | Exp. | 11.14 | 63.72 | 91.14 | | | | |

Table 4.2.1 (Cont) Haptoglobin (HP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | | Phentoypes HP 1-1 | HP 2-1 | HP 2-2 | HP 0 | Gene frequencies $HP^1$ | $HP^2$ | $X^2$ |
|---|---|---|---|---|---|---|---|---|---|
| 8-Tehran | 136 | obs. | 9 | 51. | 71 | 5 | 26.34 | 73.66 | 0.00 |
| | | Exp. | '9.09 | 50.83 | 71.08 | | | | |
| 9-Kerman | 294 | obs. | 31 | 108 | 141 | 14 | 30.36 | 69.64 | 2.16 |
| | | Exp. | 25.80 | 118.39 | 135.80 | | | | |
| 10-Kurds.Rezaieh | 145 | obs. | 18 | 50 | 72 | 5 | 30.71 | 69.29 | 3.62 |
| | | Exp. | 13.21 | 59.59 | 67.21 | | | | |

Table 4.2.2. Transferrin(Tf) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | |
|---|---|---|---|---|---|---|---|
| | | TfCC | TfCB | TfCD | $Tf^C$ | $Tf^B$ | $Tf^D$ |
| 1-Turks.Shirvan, Khorasan | 122 | 120 | 1 | 1 | 99.18 | 0.41 | 0.41 |
| 2-Zoroastrians | 175 | 174 | - | 1 | 99.71 | 0.00 | 0.29 |
| 3-Kerman | 313 | 312 | - | 1 | 99.84 | 0.00 | 0.16 |
| 4-Tehran | 186 | 186 | - | - | 100.00 | 0.00 | 0.00 |
| 5-Zabolis .Sistan & Baluchistan | 118 | 118 | - | - | 100.00 | 0.00 | 0.00 |
| 6-Baluchis.Sistan & Baluchistan | 111 | 111 | - | - | 100.00 | 0.00 | 0.00 |
| 7-Kurds.Shirvan, Khorasan | 112 | 112 | - | - | 100.00 | 0.00 | 0.00 |
| 8-Kurds.Rezaieh | 147 | 147 | - | - | 100.00 | 0.00 | 0.00 |
| 9-Turks.Rezaieh | 149 | 149 | - | - | 100.00 | 0.00 | 0.00 |
| 10-Lurs.Luristan | 178 | 178 | - | - | 100.00 | 0.00 | 0.00 |

Table 4.2.3. The third component of human complement(C3) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | | Phenotypes C3 SS | C3 SF | C3 FF | Gene frequencies F C3 | S C3 | 2 X |
|---|---|---|---|---|---|---|---|---|
| 1-Kerman | 199 | obs. | 177 | 21 | 1 | 5.78 | 94.22 | 0.18 |
| | | Exp. | 176.66 | 21.67 | 0.67 | | | |
| 2-Kurds.Shirvan, Khorasan | 60 | obs. | 53 | 6 | 1 | 6.67 | 93.33 | 2.32 |
| | | Exp. | 52.27 | 7.47 | 0.27 | | | |
| 3-Teheran | 51 | obs. | 44 | 7 | 0 | 6.87 | 93.13 | 0.27 |
| | | Exp. | 44.23 | 6.53 | 0.24 | | | |
| 4-Turks.Rezaieh | 90 | obs. | 77 | 12 | 1 | 7.78 | 92.22 | 0.43 |
| | | Exp. | 76.54 | 12.91 | 0.55 | | | |
| 5-Kurds.Rezaieh | 89 | obs. | 76 | 12 | 1 | 7.87 | 92.13 | 0.43 |
| | | Exp. | 75.55 | 12.90 | 0.55 | | | |
| 6-Turks.Shirvan, Khorasan | 82 | obs. | 70 | 11 | 1 | 7.92 | 92.08 | 0.55 |
| | | Exp. | 69.53 | 11.96 | 0.51 | | | |

Table 4.2.3. (Cont.) The third component of human complement (C3) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | | Phenotypes C3 SS | C3 SF | C3 FF | Gene frequencies $C3^F$ | $C3^S$ | $X^2$ |
|---|---|---|---|---|---|---|---|---|
| 7-Zabolis.Sistan & Baluchistan | 109 | obs. | 80 | 27 | 2 | 14.22 | 85.78 | 0.02 |
| | | Exp. | 80.21 | 26.59 | 2.20 | | | |
| 8-Zoroastrians | 131 | obs. | 93 | 34 | 4 | 16.03 | 83.97 | 0.17 |
| | | Exp. | 92.37 | 35.26 | 3.37 | | | |
| 9-Lurs.Luristan | 143 | Obs. | 101 | 37 | 5 | 16.43 | 83.57 | 0.60 |
| | | Exp. | 99.87 | 39.27 | 3.86 | | | |
| 10-Baluchis.Sistan & Baluchistan | 107 | Obs. | 67 | 32 | 8 | 22.43 | 77.57 | 2.11 |
| | | Exp. | 64.38 | 37.23 | 5.38 | | | |

Table 4.3.1 Red cell Acid phosphatase(AcP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | | Phenotypes | | | | | | Gene frequencies | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | AcP A | AcP BA | AcP B | AcP CA | AcP CB | AcP C | A AcP | B AcP | C AcP | $X^2$ |
| 1-Zoroastrians | 119 | obs. | 4 | 42 | 70 | - | 3 | - | 21.01 | 77.73 | 1.26 | 1.44 |
| | | Exp. | 5.25 | 38.87 | 71.90 | 0.63 | 2.33 | 0.02 | | | | |
| 2-Kurds.Shirvan Khorasan | 101 | obs. | 8 | 28 | 63 | - | 2 | - | 21.78 | 77.23 | 0.99 | 3.91 |
| | | Exp. | 4.79 | 33.98 | 60.24 | 0.44 | 1.54 | 0.01 | | | | |
| 3-Lurs.Luristan | 178 | obs. | 16 | 61 | 94 | 2 | 5 | - | 26.69 | 71.35 | 1.96 | 1.76 |
| | | Exp. | 12.68 | 67.79 | 90.62 | 1.86 | 4.98 | 0.06 | | | | |
| 4-Turks.Shirvan Khorasan | 115 | obs. | 11 | 40 | 62 | - | 2 | - | 26.96 | 72.17 | 0.87 | 2.18 |
| | | Exp. | 8.36 | 44.75 | 59.90 | 0.54 | 1.44 | 0.01 | | | | |
| 5-Turks.Rezaieh | 125 | obs. | 12 | 48 | 65 | - | - | - | 28.80 | 71.20 | 0.00 | 0.51 |
| | | Exp. | 10.37 | 51.26 | 63.37 | 0.00 | 0.00 | 0.00 | | | | |
| 6-Kerman | 307 | obs. | 31 | 133 | 134 | 4 | 5 | - | 32.41 | 66.12 | 1.47 | 0.68 |
| | | Exp. | 32.25 | 131.59 | 134.23 | 2.92 | 5.95 | 0.07 | | | | |

Table 4.3.1. (cont) Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | | Phenotypes | | | | | | Gene frequencies | | | $x^2$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | AcP A | AcP BA | AcP B | AcP CA | AcP CB | AcP C | A AcP | B AcP | C AcP | |
| 7-Teheran | 352 | obs. | 49 | 145 | 146 | 1 | 11 | - | 34.66 | 63.64 | 1.70 | 5.81 |
| | | Exp. | 42.28 | 155.27 | 142.55 | 4.16 | 7.64 | 0.10 | | | | |
| 8-Zabolis.Sistan & Baluchistan. | 117 | obs. | 19 | 44 | 51 | 1 | 2 | - | 35.47 | 63.25 | 1.28 | 3.00 |
| | | Exp. | 14.72 | 52.50 | 46.81 | 1.06 | 1.89 | 0.02 | | | | |
| 9-Baluchis.Sistan &Baluchistan. | 111 | obs. | 17 | 47 | 46 | - | 1 | - | 36.49 | 63.06 | 0.45 | 1.32 |
| | | Exp. | 14.78 | 51.08 | 44.14 | 0.37 | 0.63 | 0.00 | | | | |
| 10-Kurds.Rezaieh | 146 | obs. | 17 | 74 | 55 | - | - | - | 36.99 | 63.01 | 0.00 | 1.11 |
| | | Exp. | 19.97 | 68.05 | 57.97 | 0.00 | 0.00 | 0.00 | | | | |

Table 4.3.2. Red cell Adenylate Kinase (AK) Phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | |
|---|---|---|---|---|---|---|---|---|
| | | | AK 1-1 | AK 2-1 | AK 2-2 | $AK^1$ | $AK^2$ | $X^2$ |
| 1-Baluchis.Sistan & Baluchistan | 111 | obs. | 88 | 22 | 1 | 89.19 | 10.81 | 0.09 |
| | | Exp. | 88.30 | 21.40 | 1.30 | | | |
| 2-Zabolis.Sistan & Baluchistan | 118 | obs. | 94 | 23 | 1 | 89.40 | 10.60 | 0.10 |
| | | Exp. | 94.31 | 22.36 | 1.33 | | | |
| 3-Lurs.Luristan | 178 | obs. | 154 | 22 | 2 | 92.70 | 7.30 | 1.35 |
| | | Exp. | 152.96 | 24.09 | 0.95 | | | |
| 4-Tehran | 357 | obs. | 306 | 51 | - | 92.86 | 7.14 | 2.11 |
| | | Exp. | 307.82 | 47.36 | 1.82 | | | |
| 5-Kerman | 309 | obs. | 268 | 38 | 3 | 92.88 | 7.12 | 1.52 |
| | | Exp. | 266.57 | 40.87 | 1.57 | | | |
| 6-Zoroastrians | 120 | obs. | 105 | 15 | - | 93.75 | 6.25 | 0.53 |
| | | Exp. | 105.47 | 14.06 | 0.47 | | | |
| 7-Kurds.Shirvan Khorasan | 103 | obs. | 91 | 12 | - | 94.17 | 5.83 | 0.39 |
| | | Exp. | 91.35 | 11.30 | 0.35 | | | |

Table 4.3.2. (cont) Red cell Adneylate Kinase (AK) Phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | | Phenotypes AK 1-1 | AK 2-1 | AK 2-2 | Gene frequencies $AK^1$ | $AK^2$ | $X^2$ |
|---|---|---|---|---|---|---|---|---|
| 8-Turks.Shirvan, Khorasan | 116 | obs. | 103 | 13 | - | 94.40 | 5.60 | 0.41 |
| | | Exp. | 103.36 | 12.27 | 0.36 | | | |
| 9.Turks.Rezaieh | 127 | obs. | 120 | 7 | - | 97.24 | 2.76 | 0.10 |
| | | Exp. | 120.10 | 6.81 | 0.10 | | | |
| 10-Kurds.Rezaieh | 145 | obs. | 141 | 4 | - | 98.62 | 1.38 | 0.03 |
| | | Exp. | 141.03 | 3.94 | 0.03 | | | |

Table 4.3.3 Red cell phosphoglucomutase Locus1($PGM_1$) phenotypes and gene frequencies distribution in Iran

| Pupolation | Number Tested | | Phenotypes PGM 1-1 | PGM 2-1 | PGM 2-2 | Gene frequencies $PGM_1^1$ | $PGM_1^2$ | $X^2$ |
|---|---|---|---|---|---|---|---|---|
| 1-Turks.Rezaieh | 101 | obs. | 36 | 42 | 23 | 56.44 | 43.56 | 2.40 |
| | | Exp. | 32.17 | 49.66 | 19.17 | | | |
| 2-Kurds.Rezaieh | 138 | obs. | 47 | 68 | 23 | 58.70 | 41.30 | 0.04 |
| | | Exp. | 47.55 | 66.91 | 23.54 | | | |
| 3-Kerman | 310 | obs. | 123 | 144 | 43 | 62.91 | 37.09 | 0.01 |
| | | Exp. | 122.69 | 144.67 | 42.64 | | | |
| 4-Kurds.Shirvan, Khorasan | 92 | obs. | 40 | 40 | 12 | 65.22 | 34.78 | 0.16 |
| | | Exp. | 39.13 | 41.78 | 11.13 | | | |
| 5-Baluchis.Sistan & Baluchistan | 110 | obs. | 45 | 54 | 11 | 65.45 | 34.55 | 0.80 |
| | | Exp. | 47.12 | 49.75 | 13.13 | | | |
| 6-Tehran | 346 | obs. | 152 | 153 | 41 | 66.04 | 33.96 | 0.07 |
| | | Exp. | 150.90 | 155.20 | 39.90 | | | |

Table 4.3.3. (Cont) Red cell Phosphoglucomutase Locus 1($PGM_1$) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | | Phenotypes PGM 1-1 | PGM 2-1 | PGM 2-2 | Gene frequencies $PGM_1^1$ | $PGM_1^2$ | $X^2$ |
|---|---|---|---|---|---|---|---|---|
| 7-Zabolis.Sistan & Baluchistan | 117 | obs. | 53 | 51 | 13 | 67.09 | 32.91 | 0.02 |
| | | Exp. | 52.66 | 51.67 | 12.67 | | | |
| 8-Turks.Shirvan, Khorasan | 110 | obs. | 50 | 50 | 10 | 68.18 | 31.82 | 0.25 |
| | | Exp. | 51.13 | 47.73 | 11.14 | | | |
| 9-Lurs.Luristan | 175 | obs. | 90 | 68 | 17 | 70.86 | 29.14 | 0.61 |
| | | Exp. | 87.87 | 72.27 | 14.86 | | | |
| 10-Zoroastrians | 104 | Obs. | 60 | 40 | 4 | 76.92 | 23.08 | 0.72 |
| | | Exp. | 61.53 | 36.93 | 5.54 | | | |

Table 4.3.4. Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Iran.

| Population | Number Tested | | Phenotypes ADA 1-1 | ADA 2-1 | ADA 2-2 | ADA 4-1 | ADA 6-1 | Gene frequencies $ADA^1$ | $ADA^2$ | $ADA^4$ | $ADA^6$ | $X^2$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 1-Zoroastrians | 46 | obs. | 31 | 12 | 3 | - | - | 80.44 | 19.56 | 0.00 | 0.00 | 1.35 |
| | | Exp. | 29.76 | 14.48 | 1.76 | 0.00 | 0.00 | | | | | |
| 2-Turks.Shirvan, Khorasan | 118 | obs. | 83 | 33 | 2 | - | - | 84.33 | 15.67 | 0.00 | 0.00 | 0.39 |
| | | Exp. | 83.92 | 31.18 | 2.90 | 0.00 | 0.00 | | | | | |
| 3-Kurds.Shirvan, Khorasan | 101 | obs. | 74 | 23 | 4 | - | - | 84.66 | 15.34 | 0.00 | 0.00 | 1.54 |
| | | Exp. | 72.39 | 26.23 | 2.38 | 0.00 | 0.00 | | | | | |
| 4-Kurds.Rezaieh | 147 | obs. | 106 | 38 | 3 | - | - | 85.04 | 14.96 | 0.00 | 0.00 | 0.04 |
| | | Exp. | 106.31 | 37.40 | 3.29 | 0.00 | 0.00 | | | | | |
| 5-Turks.Rezaieh | 141 | obs. | 104 | 30 | 4 | 1 | 2 | 85.46 | 13.48 | 0.35 | 0.71 | 1.09 |
| | | Exp. | 102.98 | 32.49 | 2.56 | 0.84 | 1.71 | | | | | |

Table 4.3.5. Red cell Esterase D (EsD) Phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | |
|---|---|---|---|---|---|---|---|---|
| | | | EsD 1-1 | EsD 2-1 | EsD 2-2 | $EsD^1$ | $EsD^2$ | $X^2$ |
| 1-Baluchis.Sistan & Baluchistan | 110 | obs. | 57 | 41 | 12 | 70.45 | 29.55 | 1.21 |
| | | Exp. | 54.59 | 45.80 | 9.60 | | | |
| 2-Turks.Rezaieh | 129 | obs. | 66 | 54 | 9 | 72.09 | 27.91 | 0.21 |
| | | Exp. | 67.05 | 51.91 | 10.05 | | | |
| 3-Kurds.Rezaieh | 143 | obs. | 81 | 57 | 5 | 76.57 | 23.43 | 1.76 |
| | | Exp. | 83.85 | 51.30 | 7.85 | | | |
| 4-Kerman | 306 | obs. | 204 | 91 | 11 | 81.54 | 18.46 | 0.05 |
| | | Exp. | 203.43 | 92.14 | 10.43 | | | |
| 5-Tehran | 345 | obs. | 229 | 105 | 11 | 81.60 | 18.40 | 0.06 |
| | | Exp. | 229.72 | 103.60 | 11.68 | | | |
| 6-Zoroastrians | 111 | obs. | 74 | 35 | 2 | 82.43 | 17.57 | 0.87 |
| | | Exp. | 75.43 | 32.15 | 3.43 | | | |
| 7-Kurds.Shirvan, Khorasan | 101 | obs. | 71 | 27 | 3 | 83.66 | 16.34 | 0.05 |
| | | Exp. | 70.70 | 27.61 | 2.70 | | | |

Table 4.3.5. (Cont.) Red cell Esterase D(EsD)phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | | Phenotypes EsD 1-1 | EsD 2-1 | EsD 2-2 | Gene frequencies $EsD^1$ | $EsD^2$ | $X^2$ |
|---|---|---|---|---|---|---|---|---|
| 8-Zabolis.Sistan & Baluchistan | 117 | obs. | 86 | 28 | 3 | 85.47 | 14.53 | 0.16 |
| | | Exp. | 85.47 | 29.06 | 2.47 | | | |
| 9-Lurs.Luristan | 178 | obs. | 136 | 36 | 6 | 86.52 | 13.48 | 3.15 |
| | | Exp. | 133.25 | 41.52 | 3.23 | | | |
| 10-Turks.Shirvan, Khorasan | 115 | obs. | 88 | 25 | 2 | 87.39 | 12.61 | 0.02 |
| | | Exp. | 87.83 | 25.34 | 1.83 | | | |

Table 4. 3. 6. Red cell 6-Phosphogluconate dehydrogenase (6-PGD)phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Pehnotypes | | | | Gene frequencies | | |
|---|---|---|---|---|---|---|---|---|
| | | | PGD AA | PGD AC | PGD CC | $PGD^A$ | $PGD^C$ | $X^2$ |
| 1-Kurds.Shirvan, Khorasan | 70 | obs. | 61 | 9 | - | 93.57 | 6.43 | 0.33 |
| | | Exp. | 61.29 | 8.42 | 0.29 | | | |
| 2-Turks.Shirvan, Khorasan | 90 | obs. | 80 | 10 | - | 94.44 | 5.56 | 0.31 |
| | | Exp. | 80.28 | 9.44 | 0.28 | | | |
| 3-Kerman | 301 | obs. | 278 | 22 | 1 | 96.01 | 3.99 | 0.62 |
| | | Exp. | 277.48 | 23.04 | 0.48 | | | |
| 4-Tehran | 82 | obs. | 79 | 3 | - | 98.17 | 1.83 | 0.03 |
| | | Exp. | 79.03 | 2.95 | 0.03 | | | |
| 5-Zoroastrians | 66 | obs. | 66 | - | - | 100.00 | 0.00 | 0.00 |
| | | Exp. | 66.00 | 0.00 | 0.00 | | | |

# CHAPTER 5. DISCUSSION AND CONCLUSION

## 5.1. Blood group antigens

### 5.1.1. The ABO blood group system

The distribution of the ABO blood groups and respective gene frequencies in Iranian and neighbouring populations, expressed in terms of four and six phenotypes after testing with two and three antisera, respectively, is set out in Tables 5.1.1.a and b.

The frequency of the A gene ranges from 11.19 to 38.17 percent in Iranians, being lowest in the Bakhtiaris ( NiJenhuis, 1964) and highest in the Armenians of Esfahan ( Bowman and Walker,1961); that of the gene B varies between 9.59 percent in the Assyrians of Abadan ( Nijenhuis, 1964) and 34.54 percent in the Zoroastrians ( Bowman and Walker, 1961). The frequency of the 0 gene in Iran ranges from 50.32 percent in the Kurdish Jews ( Godber et al, 1973) to 76.80 percent in the Bakhtiaris( Nijenhuis, 1964). Values obtained in the present investigation are within this range of variation.
On the whole, with an average A gene frequency of 22.24 percent, B gene frequency of 16.98 percent and 0 gene frequency of 60.78 percent, the Iranian population appears to exhibit higher B and lower A gene frequencies than those found in Europeans ( Mourant et al, 1976).

Some differences in the distribution of the ABO gene frequencies seem to exist in Iran, as the B values increase from west to east and those of the A gene decrease. In the present investigation also two groups of Kurds and Turks of Rezaieh (north west ) exhibit lower B gene frequencies of 11.97 and 13.66 percent compared with those of 18.42 and 19.34 percent in the same two population groups of Shirvan, Khorasan ( north east).

Some other remarkable things must be noted:

It appears from the table that the Kurds of western Iran,like most other populations of the region,exhibit low B gene frequencies but the Kurdish Jews have rather high frequencies of both the A and the B genes. The frequencies of the gene A in the non-Kurdish Jews of Iran as in those of Iraq are also high. It is thus almost certain that the Jewish populations on the whole exhibit high frequencies of the A gene both in Iran and in Iraq.

The Arab population of Khuzistan shows ABO frequencies that are outside the range of the Arabian frequencies found by several investigators in various studies. However, in the course of history they must have become intermixed to a certain degree with negro elements, as can still be observed from the forms of hair and nose.

The Zoroastrians are separable from the Moslems by ABO blood groups frequencies as they appear to exhibit higher B and lower O gene frequencies compared with those found in the Moslems. Whatever the Zoroastrians were before the Islamic era, there is no doubt that they are a separate and genetically different breeding group from that of the present Moslem majority.

The Armenian and the Assyrian populations of Iran both seem to be characterized by high frequencies of the A and low frequencies of the B genes.

The high B and low O gene frequencies in the Turkmans of north eastern Iran suggest Mongoloid ancestry.

In neighbouring areas, the frequency of the A gene in the populations of the Caucasus ranges from 13.76 percent in the Adighes of north western Caucasus to 36.64 percent in the Khevsurs of highlands of Georgia (Bunak, 1960); that of the gene B

from 3.67 percent in the Megrelians, Abashi( Voronov, 1973) to 18.98 percent in the Lakts of Dagestan (Gadzhiev, 1964). The 0 gene frequency in the Caucasus varies between 52.54 percent in the Khevsurs of highlands of Georgia.( Bunak, 1960) and 78.14 percent in the Mingrelians of Georgia, Abkhazskaya (Aseyeva, 1936). With an average A gene frequency of 23.68 percent, B gene frequency of 10.73 percent and 0 gene frequency of 65.59 percent, the population of the Caucasus appears to exhibit a much lower B and a slightly higher A gene frequency than those found in Iranians. In general, ABO system frequencies show that the populations living in the Caucasus are characterized by low frequencies of the gene B and considerable presence of the 0 gene.

The frequency of the A gene in the populations of Turkey ranges from 22.94 percent in south east Anatolia to 32.97 percent in west Black sea ( Saatcioglu , 1979 ); that of the gene B from 8.89 percent in the Eti-Turks(Aksoy et al, 1958) to 17.00 percent in the Kurds ( Richard, 1976). The 0 gene frequency in Turkey varies between 53.86 percent in Aegean and 64.64 percent in East Black Sea (Saatcioglu, 1979). With an average A gene frequency of 28.97 percent, B gene frequency of 12.79 percent and 0 gene frequency of 58.24 percent, the population of Turkey seems to show a much lower B and a higher A gene frequency than those found in Iranians. The frequency of the 0 gene in Turkish population is only slightly lower than in Iranians.

The frequency of the A gene in the populations of Iraq ranges from 11.73 percent in the Kurdish Jews of the south east ( Tills et al, 1977) to 36.00 percent in the Kurdistani Jews ( Gurevitch et al, 1955); that of the B gene from 10.31

percent in the Arabs( Field, 1957) to 34.58 percent in the Karaite Jews ( Goldschmidt et al, 1976). The 0 gene frequency in Iraq varies between 42.89 percent in the Karaite Jews (Goldschmidt et al, 1976) and 64.44 percent in the Iraqis abroad(Onsi and El-Alfi, 1968). With an average A gene frequency of 24.22 percent, B gene frequency of 18.73 percent and 0 gene frequency of 57.05 percent, the population of Iraq appears to exhibit slightly higher B and A but lower 0 gene frequencies than those found in Iranians.

The extremely high B gene frequency of 34.58 percent and low 0 gene frequency of 42.89 percent in the Karaite Jews of Iraq ( Goldschmidt et al, 1976) clearly indicate the strong influence of isolation and genetic drif operating in this community.

The frequency of the A gene in the Kuwaiti populations ranges from 14.00 percent in the Suluba tribe to 18.00 percent in the Ajman tribe; that of the B gene from 4.00 percent in the Ajman tribe to 17.00 percent in the Suluba tribe. The 0 gene frequency in Kuwait varies between 69.00 percent in the Suluba tribe and 78.00 percent in the Ajman tribe (Khaled et al, 1981). With an average A gene frequency of 15.97 percent, B gene frequency of 12.63 percent and 0 gene frequency of 71.40 percent, the population of Kuwait seems to show much lower A and B but higher 0 gene frequencies than those found in Iranians.

The frequency of the gene A in Saudi Arabians ranges from 11.42 percent in the Sunnis of Asir and Najran (Maranjian et al, 1966) to 21.90 percent in the Saudi Arabian sample of Goedde et al (1979); that of the B gene from 4.12 percent in the Sunni sample of Asir, Najran (Maranjian et al, 1966) to 28.90 percent in the Saudi Arabian sample of Goedde et al

(1979). The O gene frequency in Saudi Arabia varies between 49.20 percent in the Saudi Arabian sample( Goedde et al,1979) and 84.46 percent in the Sunni sample of Asir, Najran (Maranjian et al, 1966). With an average A gene frequency of 15.38 percent, B gene frequency of 13.21 percent and O gene frequency of 71.41 percent, the Arab population of Saudi Arabia, like that of Kuwait, appears to exhibit much lower A and B but higher O gene frequencies than those found in Iranians.

Similar low A and B gene frequencies of respectively 19.60 and 8.20 percent and a high O gene frequency of 72.20 percent are reported by Kamel et al (1980) for the Abu-Dhabians of the United Arab Emirates.

In general, the Arab populations of Kuwait, Saudi Arabia and the United Arab Emirates are characterized by the high Overall frequency of O and the frequencies of the A and the B genes are correspondingly low. These findings agree with what is at present known about the frequencies of ABO in the indigenous desert populations (Mourant et al, 1976).

The frequency of the A gene in the populations of Pakistan ranges from 15.42 percent in the Multans (Lodhi,1960) to 28.37 percent in the Baltis of Baltistan (Clegg et al, 1961); that of the gene B from 22.99 percent in Dacca (Attabudin, 1954) to 29.23 percent in the Pathan, Dari, Timurgara sample of Bernhard (1967). The O gene frequency in Pakistan varies between 48.53 percent in the Baltis of Baltistan (Clegg et al, 1961) and 58.79 percent in the Multans (Lodhi,1960). With an average A gene frequency of 19.50 percent, B gene frequency of 25.24 percent and O gene frequency of 55.26 percent, the Pakistani population appears to exhibit lower A, much higher B and lower O gene frequencies than those found in Iranians.

The frequency of the gene A in the populations of Afghanistan ranges from 13.91 percent in the Uzbeks (Samin,1965) to 28.60 percent in the Pushtus ( Papiha et al, 1977); that of the B gene from 17.80 percent in the Pushtus (Papiha et al, 1977) to 36.21 percent in the Hazaras ( Samin, 1965). The 0 gene frequency in Afghanistan varies between 45.04 percent in the Hazaras ( Samin, 1965) and 60.80 percent in the Daris(Papiha et al, 1977). With an average A gene frequency of 20.42 percent, B gene frequency of 25.25 percent and 0 gene frequency of 54.33 percent, the population of Afghanistan, like that of Pakistan, seems to show lower A, much higher B and lower 0 gene frequencies than those found in Iranians.

The generalized features of blood groups frequencies in Afghanistan suggest Caucasoid ancestry on the one hand and Mongoloid on the other. The frequency of B in Afghanistan is higher than has been found in any Iranian population, except for those on the northern frontier with Soviet Turkmenistan. Further east, in northern Pakistan and India, and in the neighbouring areas north of the main mountain barrier, similar high frequencies of B are found in both Caucasoids and Mongoloids. In summary the Afghan populations appear to be intermediate in allele frequencies between Caucasoid and Mongoloid populations, with unmistakable evidence of both in their ancestry.

Regarding the A sub-alleles of the ABO system (Tables 5.1.1.b), the $A_2$ allele is present in all populations tested and its frequency ranges from 0.56 to 8.01 percent in Iranians' the lowest being in the Baluchis of Sistan and Baluchistan(present investigation) and the highest in the Kurds of Sanandaj (Lehmann et al, 1973). With an average $A_2$ allele frequency of 4.20 percent, the Iranian population on the whole appears to

exhibit a lower A2 allele frequency than that of about 10 percent found in Europeans (Mourant et al,1976). In Iranians about 19 percent of the total A genes are A2.

From the table it seems that some differences in the distribution of the A2 allele exist in Iran as the Kurds and the Kurdish Jews of western Iran appear to exhibit higher A2 allele frequencies than those found in other parts of the country.

The Arab population of Khuzistan (south west ) also seems to be characterized by high frequencies of the A2 allele. By contrast the Baluchis and the Zabolis of Sistan and Baluchistan (south east ) show the lowest frequencies of this allele in Iran. There are very few reports on the distribution of the A2 allele frequencies in the populations of eastern Iran, so it is difficult to see any west-east decrease of the A2 allele frequencies.

In neighbouring populations, the A2 allele frequency of 2.66 percent in the Svanis of Georgia, Abkhazskaya of the Caucasus (Verbitsky et al, 1971) is lower than that found in Iranians and lower than the frequency in Europeans. In the population of the Caucasus about 12 percent of the total A genes are A2.

The A2 allele frequencies of 6.77 and 8.10 percent in the Turks and the Eti-Turks of Turkey (Aksoy et al, 1958) are higher than the average for Iran but still lower than the European frequencies. In the Turkish populations about 25 percent of the total A genes are A2.

The frequency of the A2 allele in the Iraqi populations varies between 1.12 percent in the Kurdish Jews of the south east (Tills et al, 1977) and 18.27 percent in the Karaite Jews

(Goldschmidt et al, 1976). With the exception of the extremely high A2 allele frequency of 18.27 percent in the Karaite Jews, the population of Iraq with an average A2 frequency of 3.41 percent seems to exhibit lower A2 values than those found in Iranians and lower than the European frequencies. In Iraq about 16 percent of the total A genes are A2.

The very high A2 frequency of 18.27 percent in the Karaite Jews of Iraq clearly indicates the strong influence of isolation and genetic drift operating in this community.

The A2 allele frequency in the Kuwaiti populations ranges from 2.13 percent in the Kuwaiti Arabs (Sawhney, 1975) to 11.00 percent in the Ajman tribe (Khaled et al,1981). With an average A2 allele frequency of 7.03 percent, the population of Kuwait appears to show higher A2 values than those found in Iranians but lower than the frequencies in Europeans. In the Kuwaiti population about 43 percent of the total A genes are A2.

The frequency of the A2 allele in Saudi Arabians varies between 3.60 percent in the Shia sample of Qatif and Hasa Oases and 8.34 percent in the Sunni sample of Hejaz(Maranjian et al, 1966). With an average A2 allele frequency of 6.02 percent, the Arab population of Saudi Arabia, like that of Kuwait, seems to exhibit higher A2 values than those found in Iranians but lower than the European frequencies. In Saudi Arabians about 42 percent of the total A genes are A2.

The A2 allele frequency in the populations of Pakistan ranges from 2.59 percent in the Hunza, Gilgit (Ikin et al, 1959)to 4.19 percent in the Baltis of Baltistan(Clegg et al, 1961). With an average A2 allele frequency of 3.46 percent, the Pakistani population appears to show lower A2 allele fre-

quencies than those found in Iranians and lower than the frequencies in Europeans.In the population of Pakistan about 16 percent of the total A genes are A2.

The frequency of the A2 allele in the populations of Afghanistan varies between 3.62 percent in the Timuri and related tribes(Woodd-Walker et al,1967)and 8.00 percent in the Pushtus(Papiha et al,1977).With an average A2 allele frequency of 5.21 percent,the Afghan population seems to exhibit slightly higher A2 frequencies than those found in Iranians but still lower than the European frequencies. In Afghanistan about 22 percent of the total A genes are A2.

Conclusion

Frequencies of the gene A are high in Europe, especially in Scandinavia, in parts of central Europe, and in an area extending from Bulgaria into Asia Minor and Turkistan. The frequency of A2 appears to vary somewhat irregularly, but it .nowhere greatly exceeds 10 percent.

The distribution of B is more definite and less Patchy than that of A. There is a well marked maximum frequency of B in central Asia and northern India. In Europe the frequency of the B gene diminishes steadily from the borders of Asia to a minimum somewhere below 5 percent in parts of the Netherlands, France, Spain and Portugal. There is a slight but significant rise to over 7 percent further in the celtic countries and in Iceland, with higher local maxima near 10 percent in parts of wales, Scotland and Ireland (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arab populations of Kuwait, Saudi Arabia and the United Arab Emirates with their low frequencies of both the A and the B genes which seem to be characteristics of these population groups, the frequency of the A gene is lower and that

of the B gene much higher than those found in Europeans. On the whole, in the area under discussion, frequencies of the total A genes and the A2 allele decrease and those of the B gene increases from the west (Turkey) to the east (Pakistan).

Table 5.1.1.a I ABO blood groups distribution in Iran tested with anti-A, and anti-B sera

| Population | Number tested | Phenotypes | | | | Gene grequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 1-Assyrians.Abadan | 32 | 14 | 2 | 4 | 12 | 33.10 | 9.59 | 57.31 | Nijenhuis, L.E. | 1964 |
| 2-Southern Gorgan,Behshahr,Sari | 53 | 18 | 7 | 3 | 25 | 22.30 | 9.90 | 67.80 | Kirk, R.L., et al. | 1977 |
| 3-Armenians | 78 | 38 | 12 | 4 | 24 | 32.38 | 10.92 | 56.70 | Nijenhuis, L.E. | 1964 |
| 4-Armenians | 145 | 71 | 22 | 10 | 42 | 35.20 | 11.00 | 53.80 | Tabatabai, H. | 1977 |
| 5-West | 110 | 38 | 19 | 4 | 49 | 21.50 | 11.10 | 67.40 | Bajatzadeh,M.,& Walter,H. | 1969 |
| 6-Armenians.Julfa | 145 | 79 | 21 | 10 | 35 | 38.17 | 11.43 | 50.40 | Bowman,J.E.,& Walker,D.G. | 1961 |
| 7-North | 73 | 25 | 13 | 3 | 32 | 21.61 | 11.66 | 66.73 | Bajatzadeh,M.,& Walter,H. | 1969 |
| 8-Teheran | 115 | 46 | 22 | 3 | 44 | 24.52 | 11.66 | 63.82 | Bajatzadeh,M.,& Walter,H. | 1969 |
| 9-East | 78 | 38 | 13 | 4 | 23 | 32.42 | 11.66 | 55.92 | Bajatzadeh,M.,& Walter,H. | 1969 |
| 10-Mamassanis.Fars | 113 | 29 | 21 | 4 | 59 | 15.87 | 11.76 | 72.37 | Bowman,J.E., & Walker, D.,G. | 1961 |
| 11-Kurds.Rezaieh | 138 | 50 | 24 | 7 | 57 | 23.43 | 11.97 | 64.60 | Present study. | |
| 12-Bakhtiaris | 138 | 28 | 30 | 1 | 79 | 11.19 | 12.01 | 76.80 | Nijenhuis, L.E. | 1964 |
| 13-Tavalesh.Astara | 49 | 18 | 10 | 1 | 20 | 22.10 | 12.10 | 65.70 | Kirk, R.L., et al. | 1977 |
| 14-Shahsavar,Rudsar,Rudbar,Rasht Langarud,Lahijan,Bandar-Pahlavi | 87 | 18 | 15 | 5 | 49 | 14.10 | 12.20 | 73.70 | Kirk, R.L., et al. | 1977 |

Table 5.1.1.a.I (Cont.) ABO blood groups distribution in Iran tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Genefrequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | O | A | B | O | | |
| 15-Tehran | 140 | 17 | 8 | 2 | 23 | 10.90 | 12.35 | 76.75 | Beckett, P.H. | 1950 |
| 16-Lurs. Luristan | 167 | 56 | 31 | 9 | 71 | 21.87 | 12.80 | 65.33 | Present study. | |
| 17-Kurds.Baneh, Marivan | 77 | 25 | 14 | 5 | 33 | 21.79 | 13.17 | 65.04 | Lehmann,H., et al. | 1973 |
| 18-Turks.Rezaieh | 142 | 58 | 27 | 9 | 48 | 27.46 | 13.66 | 58.88 | Present study. | |
| 19-Kurdistan | 148 | 48 | 30 | 9 | 61 | 21.58 | 14.18 | 64.24 | Boue' & Boue'. | 1955 |
| 20-Central and South | 113 | 40 | 24 | 6 | 43 | 23.19 | 14.34 | 62.47 | Bajatzadeh,M.,& Walter,H. | 1969 |
| 21-Azarbaijan | 157 | 61 | 31 | 11 | 54 | 26.49 | 14.46 | 59.05 | Boue' and Boue'. | 1955 |
| 22-Ghashghais.Abadeh | 1518 | 476 | 321 | 72 | 469 | 20.57 | 14.47 | 64.96 | Montazami,K. | 1978 |
| 23-Ghashghais | 66 | 23 | 14 | 4 | 25 | 23.13 | 14.75 | 62.12 | Nijenhuis,L.E. | 1964 |
| 24-Azarbaijan | 1467 | 547 | 296 | 108 | 516 | 25.63 | 14.90 | 59.47 | Montazami, K. | 1978 |
| 25-Basseris.Fars | 101 | 30 | 24 | 40 | 43 | 18.67 | 15.08 | 66.25 | Bowman,J.E,& Walker,D.G. | 1961 |
| 26-Tehran | 20000 | 7000 | 4600 | 1000 | 7400 | 22.71 | 15.26 | 62.03 | Vazin, H. | 1969 |
| 27-Tehran | 565 | 173 | 133 | 29 | 230 | 19.93 | 15.61 | 64.46 | Motamed, M. | 1949 |
| 28-Kerman | 10000 | 3327 | 2224 | 660 | 3789 | 22.50 | 15.67 | 61.83 | Azhir, A. | 1951 |
| 29-Esfahan | 111 | 29 | 25 | 11 | 46 | 18.23 | 15.95 | 65.82 | Sawhney, K.S. | 1975 |
| 30-Jews | 225 | 83 | 45 | 22 | 75 | 26.85 | 16.13 | 57.02 | Silberstein,W., & Goldstein,N. | 1958 |

Table 5.1.1.a.I (Cont)

ABO blood groups distribution in Iran tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | O | A | B | O | | |
| 31-Khuzestan | 2043 | 525 | 503 | 113 | 902 | 17.08 | 16.42 | 66.50 | Montazami, K. | 1978 |
| 32-Tehran | 328 | 111 | 63 | 37 | 117 | 25.63 | 16.44 | 57.93 | Present study. | |
| 33-Mashhad | 17277 | 5259 | 4535 | 641 | 6842 | 19.03 | 16.47 | 64.50 | Afkari, A.H. | 1967 |
| 34-Fars.Recipients | 16368 | 4652 | 3872 | 1094 | 6750 | 19.42 | 16.52 | 64.06 | Mohallatee, E.A.,& Haghshenas, M . | 1969 |
| 35-Tehran | 150000 | 48060 | 34815 | 10995 | 56130 | 22.14 | 16.66 | 61.20 | Tehranchian.. | 1963 |
| 36-Kurds.Kermanshah | 127 | 41 | 28 | 11 | 47 | 23.12 | 16.66 | 60.22 | Nijenhuis,L.E. | 1964 |
| 37-Jews | 108 | 43 | 29 | 6 | 30 | 30.60 | 16.70 | 52.70 | Tabatabai, H. | 1977 |
| 38-Tehran | 992 | 337 | 224 | 80 | 351 | 23.86 | 16.71 | 59.43 | Roshan,G., et al. | 1969 |
| 39-Tehran | 42329 | 13037 | 7894 | 5337 | 16061 | 24.35 | 16.80 | 58.85 | Berelian-Jahanshahi,F. | 1973 |
| 40-Mobarakeh.Esfahan | 1211 | 357 | 284 | 93 | 477 | 20.68 | 16.97 | 62.35 | Montazami, K. | 1978 |
| 41-Esfahan | 9753 | 2992 | 2326 | 709 | 3726 | 21.22 | 17.01 | 61.77 | Mirdamadi, M.,et al. | 1978 |
| 42-Gorgan twon | 537 | 169 | 120 | 48 | 200 | 22.70 | 17.03 | 60.27 | Boue' and Boue'. | 1956 |
| 43-Shiraz | 5407 | 1633 | 1333 | 353 | 2088 | 20.50 | 17.07 | 62.43 | Tahmasbian A. | 1972 |
| 44-Tehran | 3049 | 1036 | 721 | 229 | 1063 | 23.56 | 17.07 | 59.37 | Boue' and Boue'. | 1956 |
| 45-South east | 348 | 91 | 86 | 23 | 148 | 17.99 | 17.08 | 64.93 | Nijenhuis, L.E. | 1964 |

Table 5.1.1.a.I (Cont)

ABO blood groups distribution in Iran tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 46-Esfahan | 4385 | 1426 | 1093 | 274 | 1592 | 21.86 | 17.12 | 61.02 | Montazami, K. | 1978 |
| 47-Khuzestani. Non Arab | 2555 | | | | | 19.40 | 17.30 | 63.30 | Fakhrai, H., et al. | 1978 |
| 48-Gonbad | 156 | 63 | 37 | 12 | 44 | 28.30 | 17.30 | 54.40 | Kirk, R.L., et al. | 1977 |
| 49-Tehran | 99 | 23 | 26 | 6 | 44 | 15.65 | 17.48 | 66.87 | Sawhney, K.S. | 1975 |
| 50-Gorgan Province | 134 | 46 | 29 | 14 | 45 | 25.53 | 17.48 | 56.99 | Boue' and Boue'. | 1956 |
| 51-Baluchis.Sistan & Baluchistan | 111 | 32 | 30 | 6 | 43 | 19.00 | 17.90 | 63.10 | Present study. | |
| 52-Tehran | 1196 | 374 | 300 | 90 | 432 | 21.79 | 17.93 | 60.28 | Defai, H. | 1971 |
| 53-Babol,Shahi,Amol | 70 | 21 | 19 | 4 | 26 | 20.00 | 18.10 | 61.90 | Kirk, R.L., et al. | 1977 |
| 54-Persian Gulf area | 4574 | 1084 | 1277 | 233 | 1980 | 15.65 | 18.19 | 66.16 | Montazami, K. | 1978 |
| 55-Tehran | 2334 | 748 | 579 | 196 | 811 | 22.82 | 18.27 | 58.91 | Sadatzadeh,H., & Hamidi, A.H. | 1972 |
| 56-Kurds.Sanandaj | 107 | 33 | 25 | 11 | 38 | 23.12 | 18.41 | 58.47 | Lehmann, H.,et al. | 1973 |
| 57-Kurds.Shirvan, Khorasan | 142 | 52 | 38 | 9 | 43 | 24.75 | 18.42 | 56.83 | Present study. | |
| 58-Jews | 200 | 75 | 51 | 16 | 58 | 26.37 | 18.59 | 55.04 | Gurevitch,J.,et al. | 1956 |

Table 5.1.1.a.I (Cont)

ABO blood groups distribution in Iran tested with anti-A, and Anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 59-Arabs.Khuzestan | 97 | | | | | 21.20 | 18.60 | 60.20 | Marzban, M. | 1978 |
| 60-Zabolis.Sistan & Baluchistan | 116 | 29 | 26 | 14 | 47 | 20.32 | 18.73 | 60.95 | Present study. | |
| 61-North west | 76 | 16 | 23 | 3 | 34 | 13.51 | 18.95 | 67.54 | Bajatzadeh, M., & Walter, H. | 1969 |
| 62-Moslems.Fars | 768 | 211 | 205 | 60 | 292 | 19.53 | 19.05 | 61.42 | Bowman,J.E., & Walker, D.G. | 1961 |
| 63-Ghashgais | 103 | 22 | 25 | 11 | 45 | 17.30 | 19.06 | 63.64 | Bowman,J.E., & Walker, D.G. | 1961 |
| 64-Gilan.Province | 225 | 77 | 54 | 24 | 70 | 25.67 | 19.10 | 55.23 | Boue' and Boue'. | 1956 |
| 65-Turks.Shirvan,Khorasan | 160 | 53 | 40 | 16 | 51 | 24.53 | 19.34 | 56.13 | Present study . | |
| 66-Northern Gorgan | 43 | 19 | 13 | 2 | 9 | 29.30 | 19.90 | 50.80 | Kirk, R.L.,et al. | 1977 |
| 67-Turkish.Shahsavan Azerbaijan | 247 | 89 | 62 | 28 | 68 | 27.42 | 20.26 | 52.32 | Boue' and Boue'. | 1956 |
| 68-Hamadan.Province | 115 | 38 | 31 | 12 | 34 | 24.80 | 20.86 | 54.34 | Boue' and Boue'. | 1956 |
| 69-Kurdish Jews | 94 | 34 | 24 | 12 | 24 | 28.29 | 21.39 | 50.32 | Godber, Marilyn J.,et al . | 1973 |

Table 5.1.1.a.I(Cont) ABO blood groups distribution in Iran tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 70-Kurdish Jews | 106 | 35 | 29 | 12 | 30 | 25.31 | 21.66 | 53.03 | Tills, D., et al. | 1977 |
| 71-Zoroastrians | 88 | 20 | 25 | 10 | 33 | 18.59 | 22.12 | 59.29 | Present study. | |
| 72-Arabs.Khuzestan | 364 | | | | | 16.50 | 22.90 | 60.60 | Fakhrai, H.,et al. | 1978 |
| 73- Kerman | 317 | 82 | 101 | 28 | 106 | 19.19 | 22.99 | 57.82 | Present study. | |
| 74-Arabs.Abadan | 158 | 33 | 51 | 15 | 59 | 16.46 | 23.50 | 60.04 | Nijenhuis, L.E. | 1964 |
| 75-Shi'a . Yazd | 307 | 81 | 100 | 30 | 96 | 20.09 | 24.06 | 55.85 | Sunderland,E., & Smiht, H.M. | 1966 |
| 76-Moslems. Yazd | 258 | 57 | 90 | 29 | 82 | 17.14 | 25.44 | 57.41 | Boue' and Boue'. | 1956 |
| 77-Turkmans | 1163 | 296 | 401 | 116 | 350 | 19.64 | 25.48 | 54.88 | Montazami, K. | 1978 |
| 78-Turkmans | 374 | 101 | 134 | 44 | 95 | 21.78 | 27.64 | 50.58 | Boue' and Boue'. | 1956 |
| 79-Zoroastrians. Tehran and Yazd | 150 | 25 | 69 | 17 | 39 | 15.09 | 34.54 | 50.37 | Bowman, J.E., & Walker D.G. | 1961 |

Table 5.1.1.a. II

ABO blood groups distribution in the Caucasus tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 1-Megrelians.Abashi | 195 | 66 | 11 | 3 | 115 | 19.62 | 3.67 | 76.71 | Voronov, A.A. | 1973 |
| 2-Abkhazians Ochamchiri | 91 | | | | | 18.29 | 4.27 | 77.44 | Voronov, A.A. | 1973 |
| 3-Zugdidi.Mingrelians.Georgia | 168 | 58 | 15 | 3 | 92 | 20.22 | 5.52 | 74.26 | Aseyeva, S.M. | 1936 |
| 4-Georgia.Except Gudauta | 397 | 121 | 40 | 9 | 227 | 18.00 | 6.37 | 75.63 | Aseyeva, S.M. | 1936 |
| 5-Mingrelians .Georgia,Abkhazeskaya | 229 | 58 | 24 | 6 | 141 | 15.09 | 6.77 | 78.14 | Aseyeva, S.M. | 1936 |
| 6-Svanis.Georgia,Abkhazeskaya | 659 | 234 | 62 | 26 | 337 | 22.12 | 6.89 | 70.99 | Verbitsky,M.Sh.,et al. | 1971 |
| 7-Imeretians.Trans.Caucasia | 287 | 85 | 32 | 9 | 161 | 17.97 | 7.40 | 74.63 | Bunak, V.V. | 1960 |
| 8-Imeretians.Southern Caucasus | 80 | 28 | 9 | 3 | 40 | 21.71 | 7.80 | 70.49 | Aseyeva, S.M. | 1936 |
| 9-Gudauta | 191 | 57 | 25 | 4 | 105 | 17.54 | 7.93 | 74.53 | Aseyeva, S.M. | 1936 |
| 10-Gurians.Southern Caucasus | 32 | 16 | 4 | 1 | 11 | 31.81 | 8.22 | 59.97 | Aseyeva, S.M. | 1936 |
| 11-Gurians.Lunchkhuti | 98 | | | | | 14.32 | 9.68 | 75.99 | Voronov, A.A. | 1973 |
| 12-Azerbaijanians.Barda | 147 | 76 | 17 | 11 | 43 | 36.07 | 10.02 | 53.93 | Voronov, A.A. | 1973 |
| 13-Armenians | 317 | 170 | 43 | 17 | 87 | 36.27 | 10.05 | 53.68 | Aseyeva, S.M. | 1936 |
| 14-Goris | 428 | 180 | 61 | 24 | 163 | 27.68 | 10.49 | 61.83 | Solovyeva, T.G. | 1967 |

Table 5.1.1.a. II(Cont) ABO blood groups distribution in the Caucasus tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | O | A | B | O | | |
| 15-Osetes.Northern Caucasus | 220 | 82 | 33 | 11 | 94 | 24.03 | 10.56 | 65.41 | Bunak, V.V. | 1960 |
| 16-Khevsurs.High lands of Georgia | 69 | 37 | 10 | 4 | 18 | 36.64 | 10.82 | 52.54 | Bunak, V.V. | 1960 |
| 17-Azerbaijanians.Nukha | 287 | 110 | 48 | 14 | 115 | 24.71 | 11.53 | 63.75 | Voronov, A.A. | 1973 |
| 18-Adighes.North-Western Caucasus | 486 | 115 | 98 | 9 | 264 | 13.76 | 11.75 | 74.49 | Bunak, V.V. | 1960 |
| 19-Karachay-Cherkesskaya oblast | 811 | 282 | 145 | 35 | 349 | 22.03 | 11.83 | 66.14 | Grikurov, V.S. | 1963 |
| 20-Ingushes.North-Eastern Caucasus | 407 | 165 | 76 | 17 | 149 | 25.87 | 12.27 | 61.86 | Bunak, V.V. | 1960 |
| 21-Khumarin,Uchkulan | 269 | 100 | 49 | 13 | 107 | 23.94 | 12.33 | 63.73 | Lauer, V.V. | 1929 |
| 22-Kakhetians.Gurdjaani | 196 | 86 | 27 | 19 | 64 | 32.45 | 12.36 | 55.18 | Voronov, A.A. | 1973 |
| 23-Lezgians.Dagestan | 208 | 72 | 38 | 13 | 85 | 23.08 | 13.11 | 63.81 | Gadzhiev,A.G. | 1964 |
| 24-Darghians.Dagestan | 189 | 66 | 35 | 12 | 76 | 23.35 | 13.32 | 63.33 | Gadzhiev, A.G. | 1964 |
| 25-Chechens.North-Eastern Caucasus | 756 | 299 | 159 | 38 | 260 | 25.78 | 14.14 | 60.08 | Bunak, V.V. | 1960 |
| 26- Azerbaijanians.Shemakha | 180 | 72 | 35 | 14 | 59 | 27.80 | 14.70 | 57.49 | Voronov, A.A. | 1973 |

Table 5.1.1.a.II (Cont) ABO blood groups distribution in the Caucasus tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 27-Kabardins.Northern Caucasus | 106 | 29 | 23 | 6 | 48 | 18.14 | 14.75 | 67.11 | Bunak, V.V. | 1960 |
| 28-Kumyks.Dagestan | 187 | 61 | 38 | 18 | 70 | 23.83 | 16.18 | 59.99 | Gadzhiev, A.G. | 1964 |
| 29-Avars.Dagestan | 210 | 68 | 45 | 25 | 72 | 25.07 | 18.14 | 56.79 | Gadzhiev, A.G. | 1964 |
| 30-Lakts.Dagestan | 168 | 52 | 41 | 17 | 58 | 23.12 | 18.98 | 57.90 | Gadzhiev, A.G. | 1964 |

Table 5.1.1.a.III ABO blood groups distribution in Turkey tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 1-Eti-Turks.Asia Minor, near Mersin | 118 | 55 | 14 | 6 | 43 | 30.59 | 8.89 | 60.55 | Aksoy,M.,et al. | 1958 |
| 2-Black sea region·Eastern | 5295 | 2284 | 652 | 259 | 2100 | 27.93 | 9.01 | 63.06 | Mizan, N., et al. | 196? |
| 3-East Black sea | 195 | 78 | 29 | 8 | 80 | 25.33 | 10.03 | 64.64 | Saatci oglu, Armagan. | 1979 |
| 4-Marmara | 649 | 292 | 93 | 42 | 222 | 30.00 | 11.00 | 59.00 | Saatci oglu, Armagan. | 1979 |
| 5-Turks.Istanbul | 8674 | 3833 | 1245 | 592 | 3004 | 30.00 | 11.22 | 58.78 | Benbanaste, M. | 1963 |
| 6-Black sea region.Western | 3612 | 1581 | 550 | 253 | 1228 | 29.84 | 11.82 | 58.34 | Mizan, N.,et al. | 196? |
| 7-Anatolia central.Eastern part | 7023 | 3126 | 1091 | 511 | 2295 | 30.59 | 12.15 | 57.26 | Mizan, N., et al. | 196? |
| 8-Anatolia.Eastern | 7598 | 3474 | 1148 | 593 | 2383 | 31.83 | 12.20 | 55.97 | Mizan, N., et al. | 196? |
| 9-Anatolia central.Middle part | 5453 | 2501 | 818 | 444 | 1690 | 32.15 | 12.32 | 55.53 | Mizan, N., et al. | 196? |
| 10-Middle Black sea | 360 | 157 | 54 | 30 | 119 | 30.59 | 12.40 | 57.01 | Saatci oglu,Armagan. | 1979 |
| 11-West Black sea | 173 | 82 | 28 | 13 | 50 | 32.97 | 12.70 | 54.33 | Saatci oglu,Armagan. | 1979 |
| 12-Marmar and Thrace | 8430 | 3681 | 1390 | 626 | 2733 | 30.10 | 12.79 | 57.11 | Mizan, N., et al.. | 196? |
| 13-Mediterranean region | 3122 | 1251 | 548 | 211 | 1112 | 27.11 | 13.02 | 59.87 | Mizan, N., et al. | 196? |

Table 5.1.1.a.III (Cont) ABO blood groups distribution in Turkey tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 14.Turks.Abroad.Recruits | 364 | 146 | 64 | 25 | 129 | 27.21 | 13.09 | 59.70 | Paidoussis, M. | 1967 |
| 15-East Anatolia | 399 | 158 | 72 | 26 | 143 | 26.64 | 13.17 | 60.19 | Saatci oglu, Armagan. | 1979 |
| 16-Central Anatolia | 1141 | 486 | 193 | 91 | 371 | 29.68 | 13.33 | 56.99 | Saatci oglu, Armagan. | 1979 |
| 17-Mediterranean region | 321 | 122 | 51 | 30 | 118 | 27.20 | 13.40 | 59.40 | Saatci oglu, Armagan. | 1979 |
| 18-Asia Minor Aegean region | 3166 | 1291 | 546 | 249 | 1080 | 28.31 | 13.45 | 58.24 | Mizan, N., et al. | 196? |
| 19-Anatolia Central.Western part | 4096 | 1819 | 700 | 331 | 1246 | 31.12 | 13.51 | 55.37 | Mizan, N., et al. | 196? |
| 20-Anatolia.South-Eastern | 5200 | 2020 | 1016 | 390 | 1774 | 26.91 | 14.59 | 58.50 | Mizan, N., et al. | 196? |
| 21-Turks.Asia Minor,Mersin | 108 | 43 | 20 | 10 | 35 | 28.54 | 14.94 | 56.52 | Aksoy,M.,et al. | 1958 |
| 22-Aegean | 423 | 178 | 79 | 42 | 124 | 30.67 | 15.47 | 53.86 | Saatci oglu, Armagan. | 1979 |
| 23-South east Anatolia | 220 | 76 | 50 | 13 | 81 | 22.94 | 15.60 | 61.46 | Saatci oglu, Armagan. | 1979 |
| 24-Kurds | | | | | | 27.00 | 17.00 | 56.00 | Richard, P. | 1976 |

Table 5.1.1.a IV ABO blood groups distribution in Iraq tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 1-Arab | 123 | 51 | 18 | 6 | 48 | 26.81 | 10.31 | 62.88 | Field, H. | 1957 |
| 2-Kurdistani Jews | 250 | 117 | 34 | 32 | 67 | 36.00 | 14.04 | 49.96 | Gurevitch, J.,et al. | 1955 |
| 3-Kurds | | | | | | 23.00 | 16.00 | 61.00 | Al-Khafaji, S.D.,et al. | 1976 |
| 4-Kurdish Jews.North West | 61 | 27 | 12 | 6 | 16 | 32.42 | 16.08 | 51.50 | Tills, D., et al. | 1977 |
| 5-Kurdistani Jews | 129 | 53 | 23 | 17 | 36 | 32.02 | 16.76 | 51.22 | Gurevitch, J.,et al. | 1956 |
| 6-Iraqi abroad | 313 | 89 | 81 | 16 | 127 | 18.56 | 17.00 | 64.44 | Onsi, A.A., & El-Alfi, | 1968 |
| 7-Jews | 308 | 128 | 61 | 38 | 81 | 31.94 | 17.53 | 50.53 | Silberstein,W., & Goldstein, N. | 1958 |
| 8-Civilian donors | 1185 | 406 | 286 | 98 | 395 | 24.23 | 17.81 | 57.96 | Fakhrie, S.D. | 1966 |
| 9-Bedouin.Baghdad | 338 | 90 | 87 | 23 | 138 | 18.43 | 17.89 | 63.68 | Kayassi,A.I.,et al. | 1938 |
| 10-Assyrians | 99 | 27 | 25 | 8 | 39 | 19.53 | 18.26 | 62.21 | Ikin,Elizabeth W.,et al. | 1965 |
| 11-Paid donors | 4056 | 1209 | 1037 | 320 | 1490 | 21.06 | 18.42 | 60.52 | Fakhrie, S.D. | 1966 |
| 12-Kurds | 1500 | 449 | 293 | 227 | 531 | 25.29 | 18.73 | 55.98 | Kennedy, W.P., and MaC-farlane, J. | 1936 |
| 13-Kurdish Jews | 27 | 10 | 8 | 1 | 8 | 23.81 | 18.74 | 57.45 | Godber,Marilyn J.,et al. | 1973 |

Table 5.1.1.a. IV (Cont) ABO blood groups distribution in Iraq tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 14-Arabs | 493 | 150 | 133 | 36 | 174 | 21.15 | 18.98 | 59.87 | Kennedy,W.P., and MaCfarlane, J. | 1936 |
| 15-Moslems | 386 | 121 | 109 | 26 | 130 | 21.54 | 19.49 | 59.06 | Boyd,W.C., and Boyd,L.G. | 1941 |
| 16-Military donors | 1067 | 306 | 300 | 77 | 384 | 19.98 | 19.63 | 60.39 | Fakhrie, S.D. | 1966 |
| 17-Baghdadi Jews | 162 | 62 | 39 | 21 | 40 | 30.09 | 20.60 | 49.31 | Gurevitch,J.,et al. | 1956 |
| 18-Kurdish Jews.South East | 50 | 8 | 19 | 3 | 20 | 11.73 | 25.17 | 63.10 | Tills, D., et al. | 1977 |
| 19-Karaite Jews | 72 | 22 | 34 | 6 | 10 | 22.53 | 34.58 | 42.89 | Goldschmidt,E.,et al. | 1976 |

Table 5.1.1.a. V ABO blood groups distribution in Kuwait tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | O | A | B | O | | |
| 1-Ajman tribe | 52 | 17 | 3 | 1 | 31 | 18.00 | 4.00 | 78.00 | Khaled, E., et al. | 1981 |
| 2-Kuwaiti Arabs | 162 | 45 | 32 | 4 | 81 | 17.31 | 12.68 | 70.01 | Sawhney, K.S. | 1975 |
| 3-General population | 74 | 19 | 17 | 2 | 36 | 15.00 | 14.00 | 71.00 | Khaled,E., et al. | 1981 |
| 4-Kuwaitis | 2632 | 638 | 635 | 115 | 1244 | 15.53 | 15.47 | 69.00 | Onsi, A. A.,et al. | 1968 |
| 5-Suluba tribe | 52 | 12 | 14 | 2 | 24 | 14.00 | 17.00 | 69.00 | Khaled, E., et al. | 1981 |

Table 5.1.1.a. VI ABO blood groups distribution in Saudi Arabia tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | O | A | B | O | | |
| 1-Sunnis.Asir,Najran | 74 | 14 | 4 | 2 | 54 | 11.42 | 4.12 | 84.46 | Maranjian,G.,et al. | 1966 |
| 2-Sunnis. Hejaz | 102 | 27 | 14 | 4 | 57 | 16.50 | 9.22 | 74.28 | Maranjian,G.,et al. | 1966 |
| 3-Bedouins | 178 | 49 | 28 | 7 | 94 | 17.25 | 10.35 | 72.40 | Maranjian, G.,et al. | 1966 |
| 4-Sunnis. Najd | 180 | 42 | 35 | 6 | 97 | 14.44 | 12.12 | 73.44 | Maranjian, G.,et al. | 1966 |
| 5-Western Saudi Arabia | 210 | 54 | 41 | 8 | 107 | 16.18 | 12.57 | 71.38 | Saha, N.,et al. | 1980 |
| 6-Sunnis.Qatif & Hasa Oases | 323 | 73 | 74 | 7 | 169 | 13.36 | 13.49 | 73.15 | Maranjian, G.,et al. | 1966 |
| 7-Shias.Qatif & Hasa Oases | 465 | 88 | 112 | 17 | 248 | 12.05 | 14.98 | 72.97 | Maranjian, G.,et al. | 1966 |
| 8-Saudi Arabians | 311 | 63 | 85 | 10 | 153 | 21.90 | 28.90 | 49.20 | Goedde, H.W., et al. | 1979 |

Table 5.1.1.a. VII

ABO blood groups distribution in the United Arab Emirates

tested with

anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | |
| 1- Abu-Dhabians | 624 | 208 | 86 | 12 | 318 | 19.60 | 8.20 | 72.20 | Kamel,K., et al. 1980 |

Table 5.1.1.a. VIII ABO blood groups distribution in Pakistan tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | |
| 1-Dacca | 1000 | 254 | 311 | 97 | 338 | 19.38 | 22.99 | 57.63 | Attabudin.ed.by Boyde, 1954 W.C. & Boyde,Lyle G. |
| 2-Baltis.Baltistan | 80 | 23 | 9 | 3 | 25 | 28.37 | 23.10 | 48.53 | Clegg, E.J., et al. 1961 |
| 3-Hunza.Gilgit | 23 | 5 | 8 | 2 | 8 | 16.77 | 24.78 | 58.45 | Ikin,Elizabeth W.,et al.1959 |
| 4-Pathans.Swat,Saidu Sharif | 133 | 31 | 42 | 18 | 42 | 20.32 | 25.54 | 54.14 | Alciati, G. 1968 |
| 5-Multans | 201 | 43 | 76 | 14 | 68 | 15.42 | 25.79 | 58.79 | Lodhi, M.A.K. 1960 |
| 6-Pathan.Dari,Timurgara | 78 | 16 | 31 | 8 | 23 | 16.76 | 29.23 | 54.01 | Bernhard, W. 1967 |

Table 5.1.1.a. IX ABO blood groups distribution in Afghanistan tested with anti-A, and anti-B sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | A | B | AB | 0 | A | B | 0 | | |
| 1-Pushtus | 104 | 42 | 24 | 7 | 31 | 28.60 | 17.80 | 53.60 | Papiha,S.S.,et al. | 1977 |
| 2-Daris | 179 | 51 | 48 | 14 | 66 | 20.10 | 19.10 | 60.80 | Papiha,S.S.,et al. | 1977 |
| 3-Timuri & related tribes | 118 | 30 | 38 | 14 | 36 | 20.73 | 25.02 | 54.25 | Woodd-Walker,R.B.,et al. | 1967 |
| 4-Uzbeks | 369 | 90 | 171 | 3 | 105 | 13.91 | 28.10 | 57.99 | Samin, P. | 1965 |
| 5-Hazaras | 1052 | 263 | 533 | 77 | 179 | 18.75 | 36.21 | 45.04 | Samin, P. | 1965 |

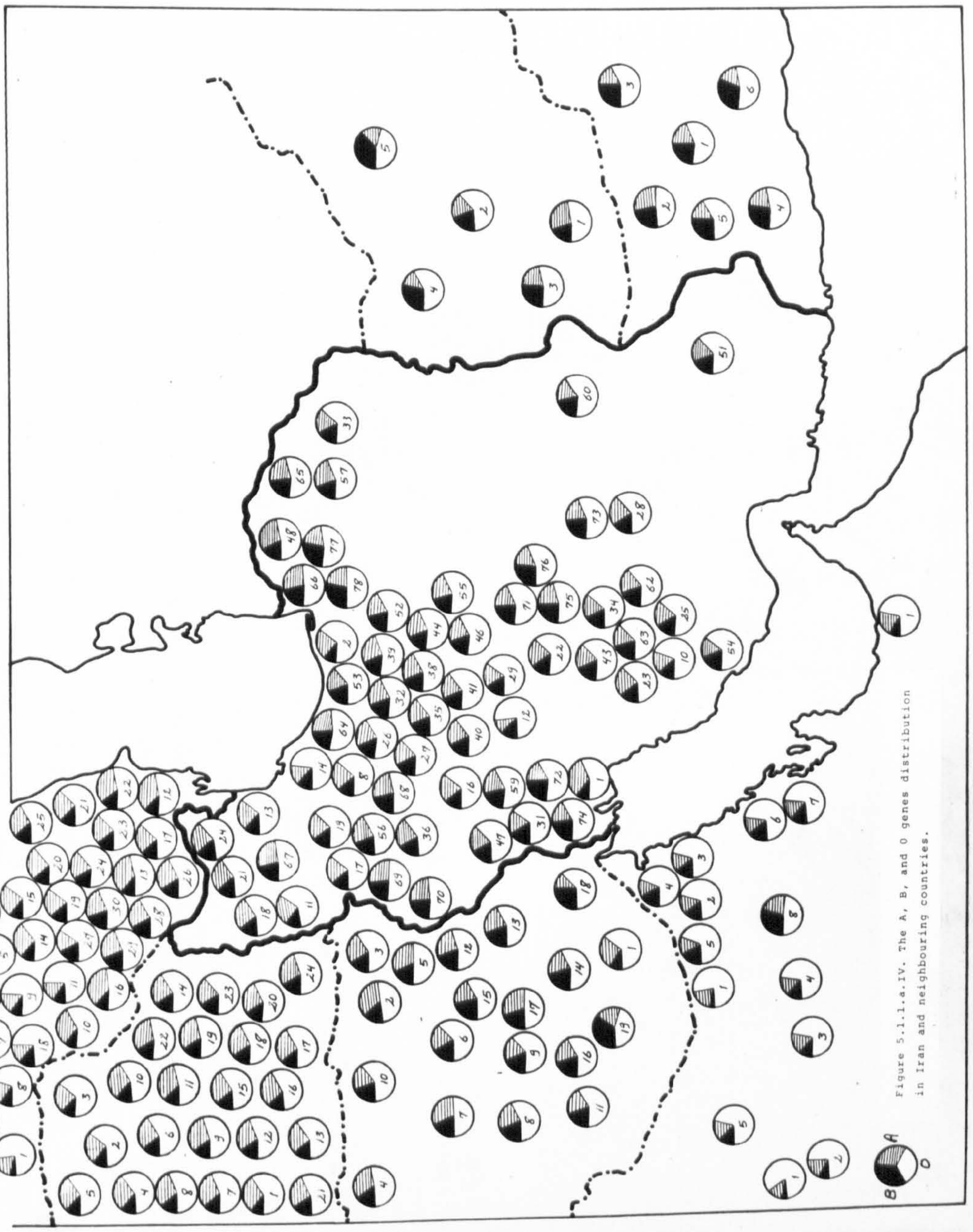



Figure 5.1.1.a.IV. The A, B, and O genes distribution in Iran and neighbouring countries.

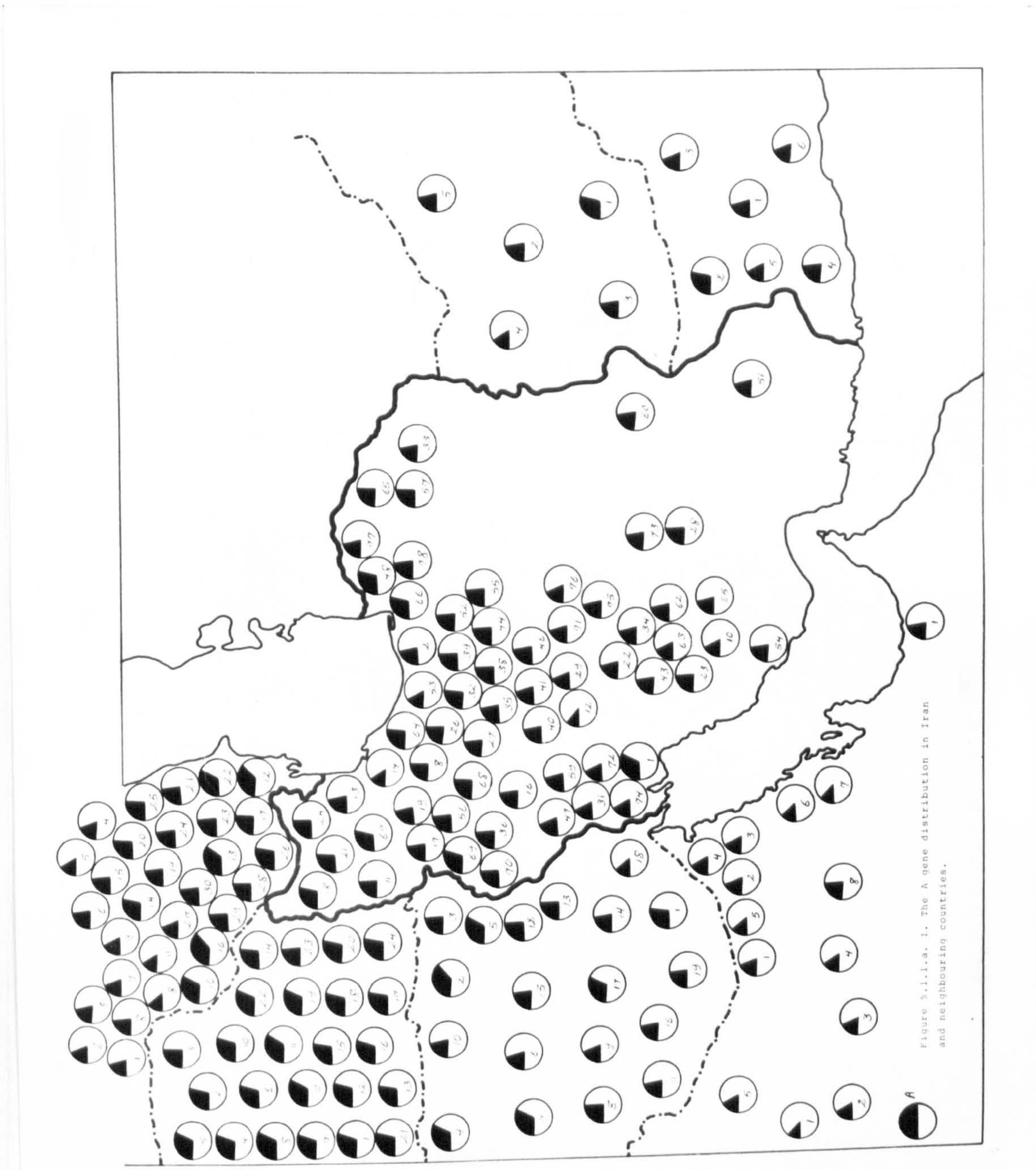


Figure 5.1.1.a. 1. The A gene distribution in Iran and neighbouring countries.

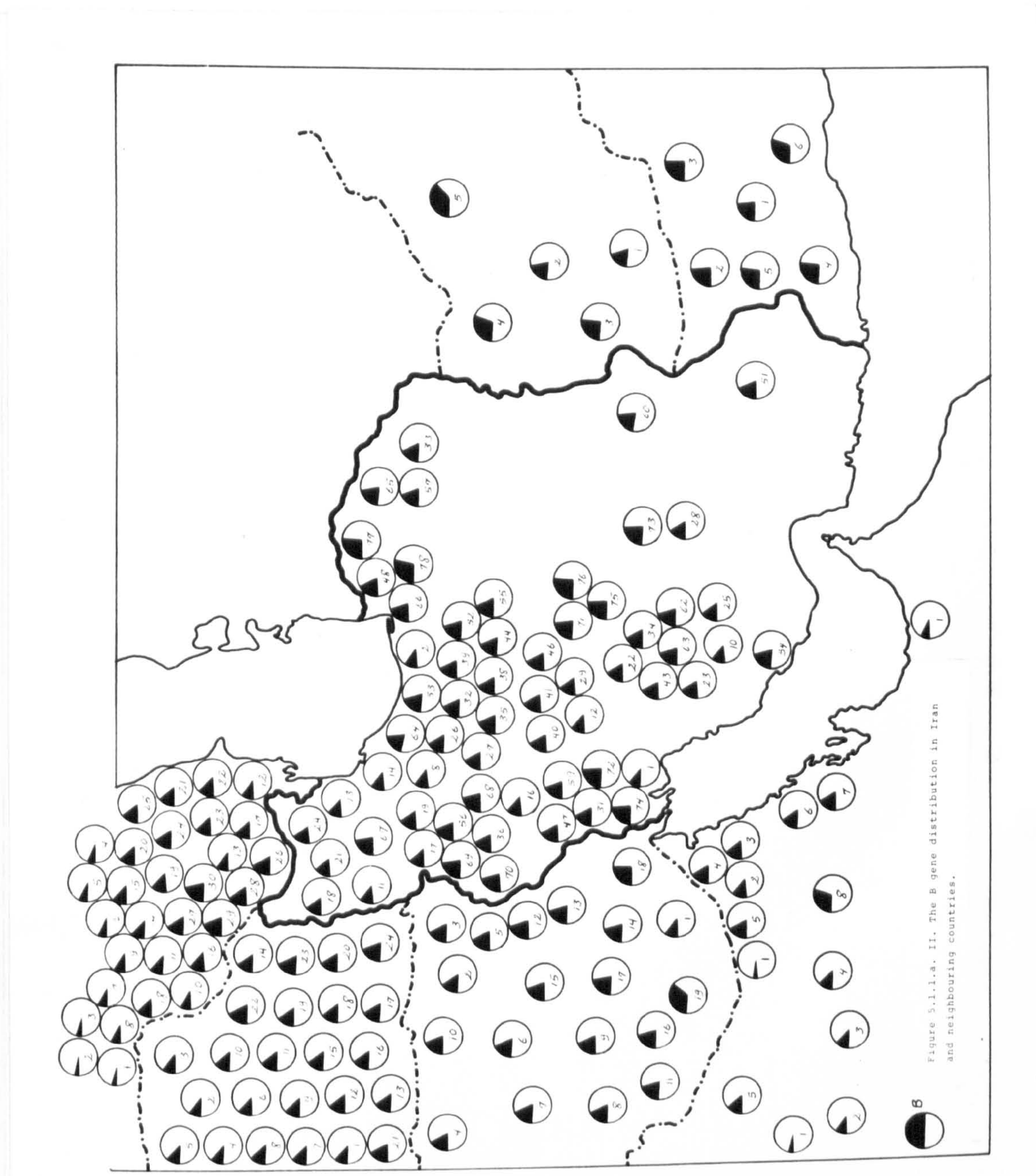


Figure 5.1.1.a. II. The B gene distribution in Iran and neighbouring countries.

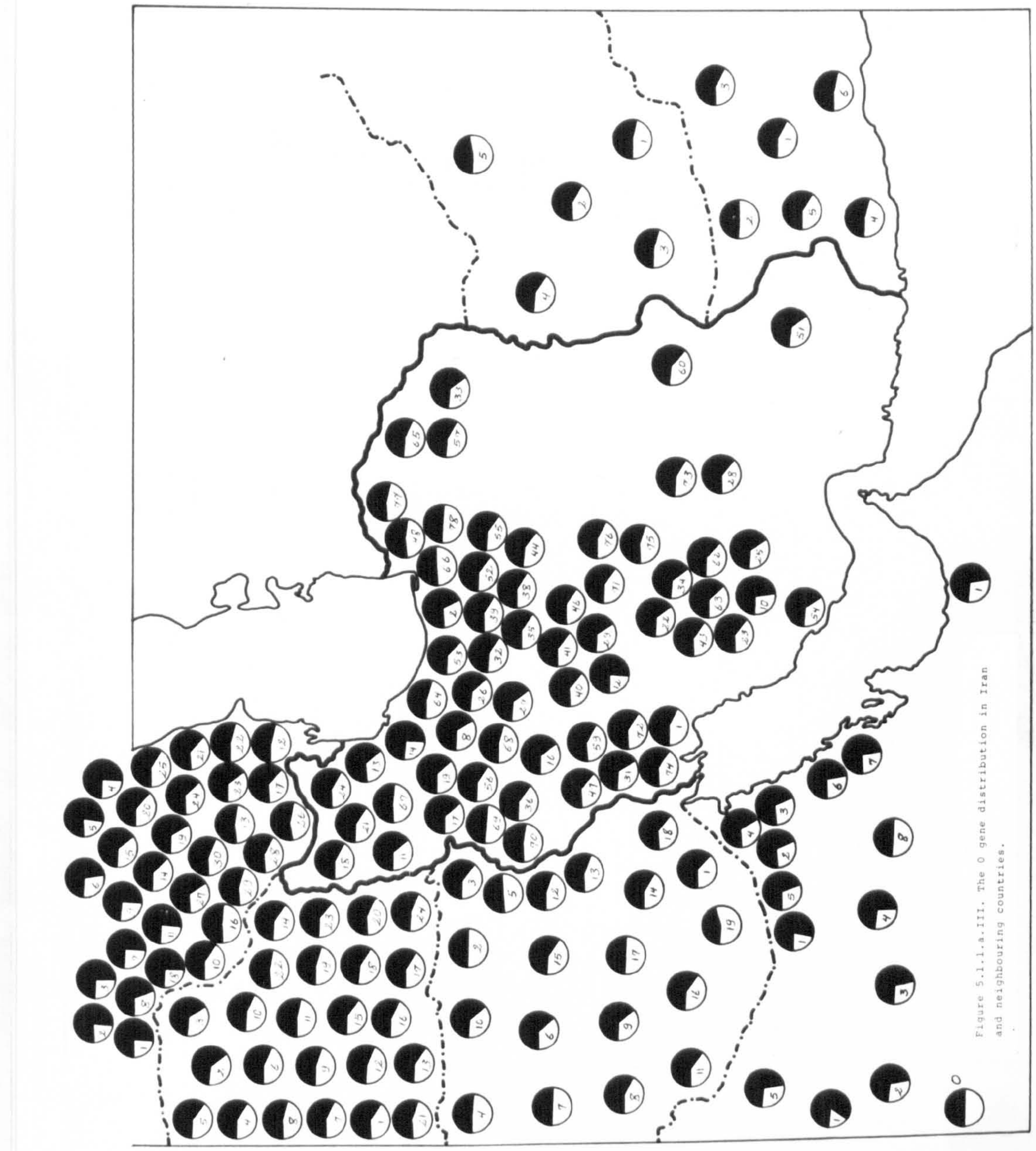


Figure 5.1.1.a.III. The O gene distribution in Iran and neighbouring countries.

Table 5.1.1.b.I ABO blood groups distribution in Iran tested with anti-A, anti-B, and anti-A1 sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A1 | A2 | B | A1B | A2B | 0 | A1 | A2 | B | 0 | |
| 1-Baluchis.Sistan & Baluchistan | 111 | 32 | - | 30 | 5 | 1 | 43 | 18.50 | 0.56 | 17.89 | 63.05 | Present study. |
| 2-Zabolis. Sistan & Baluchistan | 116 | 28 | 1 | 26 | 13 | 1 | 47 | 19.30 | 1.04 | 18.73 | 60.93 | Present study.. |
| 3-Southern Goran,Sari, Behshahr | 53 | 18 | - | 7 | 2 | 1 | 25 | 21.10 | 1.20 | 9.90 | 67.80 | Kirk,R.L.,et al. 1977 |
| 4-Tavalesh,Astara | 49 | 18 | - | 10 | - | 1 | 20 | 20.80 | 1.30 | 12.10 | 65.70 | Kirk,R.L.,et al. 1977 |
| 5-Bakhtiaris | 138 | 25 | 3 | 30 | 1 | - | 79 | 9.99 | 1.32 | 12.01 | 76.77 | Nijenhuis,L.E. 1964 |
| 6-Shahsavar,Rudsar,Langa-rud,Lahijan,Bandar-Pah-Pahlvi,Rudbar,Rasht | 87 | 16 | 2 | 15 | 4 | 1 | 49 | 12.20 | 1.90 | 12.20 | 73.70 | Kirk,R.L.,et al. 1977 |
| 7-Armenians | 145 | 68 | 3 | 22 | 8 | 2 | 42 | 33.30 | 1.90 | 11.00 | 53.80 | Tabatabai,H. 1977 |
| 8-North West | 76 | 14 | 2 | 23 | 2 | 1 | 34 | 11.23 | 2.28 | 18.95 | 67.54 | Bajatzadeh,M.,& Walter, H. 1969 |
| 9-Babol, Shahi,Amol | 70 | 18 | 3 | 19 | 4 | - | 26 | 17.20 | 2.70 | 18.20 | 61.90 | Kirk,R.L.,et al. 1977 |
| 10-Esfahan | 111 | 25 | 4 | 25 | 8 | 3 | 46 | 15.42 | 2.81 | 15.95 | 65.82 | Sawhney,K.S. 1975 |
| 11-Tehran | 115 | 41 | 5 | 22 | 3 | - | 44 | 21.63 | 2.89 | 11.66 | 63.82 | Bajatzadeh,M.,& Walter, H. 1969 |

Table 5.1.1.b.I(Cont)

ABO blood groups distribution in Iran tested with anti-A, anti-B, and anti-A1 sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A1 | A2 | B | A1B | A2B | 0 | A1 | A2 | B | 0 | | |
| 12-South-east | 348 | 78 | 13 | 86 | 19 | 4 | 148 | 15.07 | 2.92 | 17.08 | 64.93 | Nijenhuis,L.E. | 1964 |
| 13-Zoroastrians | 88 | 17 | 3 | 25 | 8 | 2 | 33 | 15.25 | 3.35 | 22.12 | 59.28 | Present study. | |
| 14-Jews | 108 | 39 | 4 | 29 | 4 | 2 | 30 | 27.20 | 3.40 | 16.70 | 52.70 | Tabatabai,H. | 1977 |
| 15-Gonbad | 156 | 57 | 6 | 37 | 10 | 2 | 44 | 24.70 | 3.60 | 17.30 | 54.40 | Kirk,R.L.,et al. | 1977 |
| 16-Tehran | 94 | 21 | 5 | 17 | 5 | - | 46 | 13.94 | 3.73 | 11.99 | 70.34 | Sawhney,K.S. | 1975 |
| 17-Ghashghais | 66 | 19 | 4 | 14 | 4 | - | 25 | 19.27 | 3.86 | 14.75 | 62.12 | Nijenhuis,L.E. | 1964 |
| 18-Moslems.Yazd | 258 | 45 | 12 | 90 | 19 | 10 | 82 | 13.13 | 4.01 | 25.44 | 57.41 | Boue' and Boue'. | 1956 |
| 19-Tehran | 1239 | 351 | 61 | 301 | 83 | 16 | 427 | 19.51 | 4.04 | 17.92 | 58.53 | Boue' and Boue'. | 1956 |
| 20-West | 110 | 31 | 7 | 19 | 3 | 1 | 49 | 16.96 | 4.54 | 11.10 | 64.70 | Bajatzadeh,M., & Walter, H. | 1969 |
| 21-Turks.Rezaieh | 92 | 36 | 5 | 18 | 3 | 1 | 29 | 24.38 | 4.55 | 12.94 | 58.13 | Present study. | |
| 22-Arabs.Abadan | 158 | 24 | 9 | 51 | 11 | 4 | 59 | 11.72 | 4.74 | 23.50 | 60.04 | Nijenhuis,L.E. | 1964 |
| 23-Lurs.Luristan | 167 | 47 | 9 | 31 | 5 | 4 | 71 | 17.10 | 4.86 | 12.80 | 65.24 | Present study. | |
| 24-Kurdish Jews | 106 | 29 | 6 | 29 | 10 | 2 | 30 | 20.42 | 4.89 | 21.66 | 53.03 | Tills,D.,et al. | 1977 |
| 25-Northern Gorgan | 43 | 16 | 3 | 13 | 2 | - | 9 | 24.30 | 5.00 | 19.90 | 50.80 | Kirk,R.L.,et al. | 1977 |

Table 5.1.1.b.I(Cont.) ABO blood groups distribution in Iran tested with anti-A, anti-B, and anti-A1 sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A1 | A2 | B | A1B | A2B | 0 | A1 | A2 | B | 0 | | |
| 26-Armenians | 78 | 33 | 5 | 12 | 3 | 1 | 24 | 26.83 | 5.55 | 10.92 | 56.70 | Nijenhuis,L.E. | 1964 |
| 27-Kurds.Baneh,Marivan | 77 | 19 | 6 | 14 | 4 | 1 | 33 | 16.21 | 5.58 | 13.17 | 65.04 | Lehmann,H.,et al. | 1973 |
| 28-Central & South | 113 | 32 | 8 | 24 | 4 | 2 | 43 | 17.57 | 5.62 | 14.34 | 62.47 | Bajatzadeh,M., & Walter, H. | 1969 |
| 29-Kurdish Jews | 94 | 27 | 7 | 24 | 11 | 1 | 24 | 22.63 | 5.66 | 21.39 | 50.32 | Godber,Marilyn J., et al. | 1973 |
| 30-North | 73 | 19 | 6 | 13 | 2 | 1 | 32 | 15.68 | 5.93 | 11.66 | 66.73 | Bajatzadeh,M., & Walter, H. | 1969 |
| 31-Shi'a.Yazd | 151 | 28.42 | 11.58 | 48 | 9.69 | 4.31 | 49 | 13.55 | 6.32 | 23.17 | 56.96 | Sunderland,E.,& Smith,H.M. | 1966 |
| 32-Assyrians.Abadan | 32 | 12 | 2 | 2 | 3 | 1 | 12 | 26.69 | 6.41 | 9.59 | 57.31 | Nijenhuis,L.E. | 1964 |
| 33-Arabs.Khuzestan | 97 | | | | | | | 14.70 | 6.50 | 18.60 | 60.20 | Marzban,M. | 1978 |
| 34-Kurds.Rezaieh | 138 | 38 | 12 | 24 | 3 | 4 | 57 | 16.26 | 7.28 | 11.97 | 64.49 | Present study. | |
| 35-East | 78 | 31 | 7 | 13 | 3 | 1 | 23 | 25.09 | 7.33 | 11.66 | 55.92 | Bajatzadeh,M., & Walter, H. | 1969 |

Table 5.1.1.b.I (Cont.) ABO blood groups distribution in Iran tested with anti-A, anti-B, and anti-A1 sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A1 | A2 | B | A1B | A2B | O | A1 | A2 | B | O | |
| 36-Kurds.Kermanshah | 127 | 29 | 12 | 28 | 7 | 4 | 47 | 15.36 | 7.76 | 16.66 | 60.22 | Nijenhuis,L.E. 1964 |
| 37-Kurds.Sanandaj | 107 | 23 | 10 | 25 | 7 | 4 | 38 | 15.11 | 8.01 | 18.41 | 58.47 | Lehmann,H.,et al. 1973 |

Table 5.1.1.b.II ABO blood groups distribution in the Caucasus tested with anti-A, anti-B, and anti-Al sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A1 | A2 | B | A1B | A2B | 0 | A1 | A2 | B | 0 | |
| 1-Svanis . Georgia.Abkhazskaya | 358 | 119 | 12 | 36 | 11 | 3 | 177 | 20.18 | 2.66 | 7.23 | 69.93 | Verbitsky,M.Sh., 1971 et al. |

Table 5.1.1.b.III ABO blood groups distribution in Turkey tested with anti-A, anti-B, and anti-Al sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A1 | A2 | B | A1B | A2B | 0 | A1 | A2 | B | 0 | |
| 1-Turks.Asia Minor,Mersin | 108 | 34 | 9 | 20 | 8 | 2 | 35 | 21.77 | 6.77 | 14.94 | 56.52 | Aksoy,M.,et al. 1958 |
| 2-Eti-Turks.Asia Minor,near Mersin | 118 | 43 | 12 | 14 | 4 | 2 | 43 | 22.49 | 8.10 | 8.89 | 60.55 | Aksoy,M.,et al. 1958 |

Table 5.1.1.b.IV ABO blood groups distribution in Iraq tested with anti-A, anti-B, and anti-A1 sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A1 | A2 | B | A1B | A2B | 0 | A1 | A2 | B | 0 | |
| 1-Kurdish Jews.South east | 50 | 8 | - | 19 | 2 | 1 | 20 | 10.61 | 1.12 | 25.17 | 63.10 | Tills,D.,et al. 1977 |
| 2-Kurdish Jews | 27 | 10 | - | 8 | - | 1 | 8 | 21.31 | 2.50 | 18.74 | 57.45 | Godber,Marilyn J., et al. 1973 |
| 3-Assyrians | 99 | 23 | 4 | 25 | 7 | 1 | 39 | 16.47 | 3.06 | 18.26 | 62.21 | Ikin,Elizabeth W., et al. 1965 |
| 4- Kurdis Jews.North west | 61 | 23 | 4 | 12 | 4 | 2 | 16 | 25.45 | 6.97 | 16.08 | 51.50 | Tills ,D.,et al. 1977 |
| 5-Karaite Jews | 72 | 4 | 18 | 34 | 2 | 4 | 10 | 4.26 | 18.27 | 34.58 | 42.89 | Goldschmidt,E., et al. 1976 |

Table 5.1.1.b V ABO blood groups distribution in Kuwait tested with anti-A, anti-B, and anti-Al sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Al | A2 | B | A1B | A2B | O | Al | A2 | B | O | |
| 1-Kuwaiti Arabs | 162 | 40 | 5 | 32 | 2 | 2 | 81 | 15.18 | 2.13 | 12.68 | 70.01 | Sawhney, K.S. 1975 |
| 2-General population | 74 | 13 | 6 | 17 | - | 2 | 36 | 9.00 | 6.00 | 14.00 | 71.00 | Khaled,E.,et al. 1981 |
| 3-Suluba tribe | 52 | 5 | 7 | 14 | - | 2 | 24 | 5.00 | 9.00 | 17.00 | 69.00 | Khaled,E.,et al. 1981 |
| 4-Ajman tribe | 52 | 7 | 10 | 3 | - | 1 | 31 | 7.00 | 11.00 | 4.00 | 78.00 | Khaled,E.,et al. 1981 |

Table 5.1.1.b. VI

ABO blood groups distribution in Saudi Arabia tested with anti-A, anti-B, and anti-A1 sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A1 | A2 | B | A1B | A2B | 0 | A1 | A2 | B | 0 | | |
| 1-Shias.Qatif & Hasa Oases | 465 | 66 | 22 | 112 | 9 | 8 | 248 | 8.45 | 3.60 | 14.98 | 72.97 | Maranjian,G., et al. | 1966 |
| 2-Sunnis.Qatif & Hasa Oases | 323 | 54 | 19 | 74 | 5 | 2 | 169 | 9.65 | 3.71 | 13.49 | 73.15 | Maranjian,G., et al. | 1966 |
| 3- Western Saudi Arabia | 210 | 37 | 17 | 41 | 7 | 1 | 107 | 10.72 | 5.46 | 12.57 | 71.38 | Saha,N.,et al. | 1980 |
| 4-Sunnis.Najd | 180 | 26 | 16 | 35 | 2 | 4 | 97 | 8.15 | 6.29 | 12.12 | 73.44 | Maranjian,G., et al. | 1966 |
| 5-Sunnis.Asir,Najran | 74 | 5 | 9 | 4 | 1 | 1 | 54 | 4.14 | 7.28 | 4.12 | 84.46 | Maranjian,G., et al. | 1966 |
| 6-Bedouins | 178 | 30 | 19 | 28 | 3 | 4 | 94 | 9.78 | 7.47 | 10.35 | 72.40 | Maranjian,G., et al. | 1966 |
| 7-Sunnis. Hejaz | 102 | 13 | 14 | 14 | 3 | 1 | 57 | 8.16 | 8.34 | 9.22 | 74.28 | Maranjian,G., et al. | 1966 |

Table 5.1.1.b. VIII ABO blood groups distribution in Pakistan tested with anti-A, anti-B, and anti-A1 sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A1 | A2 | B | A1B | A2B | O | A1 | A2 | B | O | |
| 1-Hunza.Gilgit | 23 | 5 | - | 8 | 1 | 1 | 8 | 14.18 | 2.59 | 24.77 | 58.44 | Ikin,Elizabeth W., et al. 1959 |
| 2-Pathans.Swat, Saidu Sharif | 133 | 27 | 4 | 42 | 14 | 4 | 42 | 16.71 | 3.61 | 25.54 | 54.14 | Alciati, G. 1968 |
| 3-Baltis.Baltistan | 60 | 2 | 21 | 9 | 3 | - | 25 | 24.18 | 4.19 | 23.10 | 48.53 | Clegg,E.J., et al. 1961 |

Table 5.1.1.b. IX

ABO blood groups distribtution in Afghanistan tested with anti-A, anti-B, and anti-Al sera

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | A1 | A2 | B | A1B | A2B | O | A1 | A2 | B | O | | |
| 1-Timuri & related tribes | 118 | 26 | 4 | 38 | 11 | 3 | 36 | 17.11 | 3.62 | 25.02 | 54.25 | Woodd-Walker, R. B., et al. | 1967 |
| 2- Daris | 179 | 42 | 9 | 48 | 12 | 2 | 66 | 16.10 | 4.00 | 19.10 | 60.80 | Papiha, S.S., et al. | 1977 |
| 3- Pushtus | 104 | 32 | 10 | 24 | 6 | 1 | 31 | 20.60 | 8.00 | 17.80 | 53.60 | Papiha, S.S., et al. | 1977 |

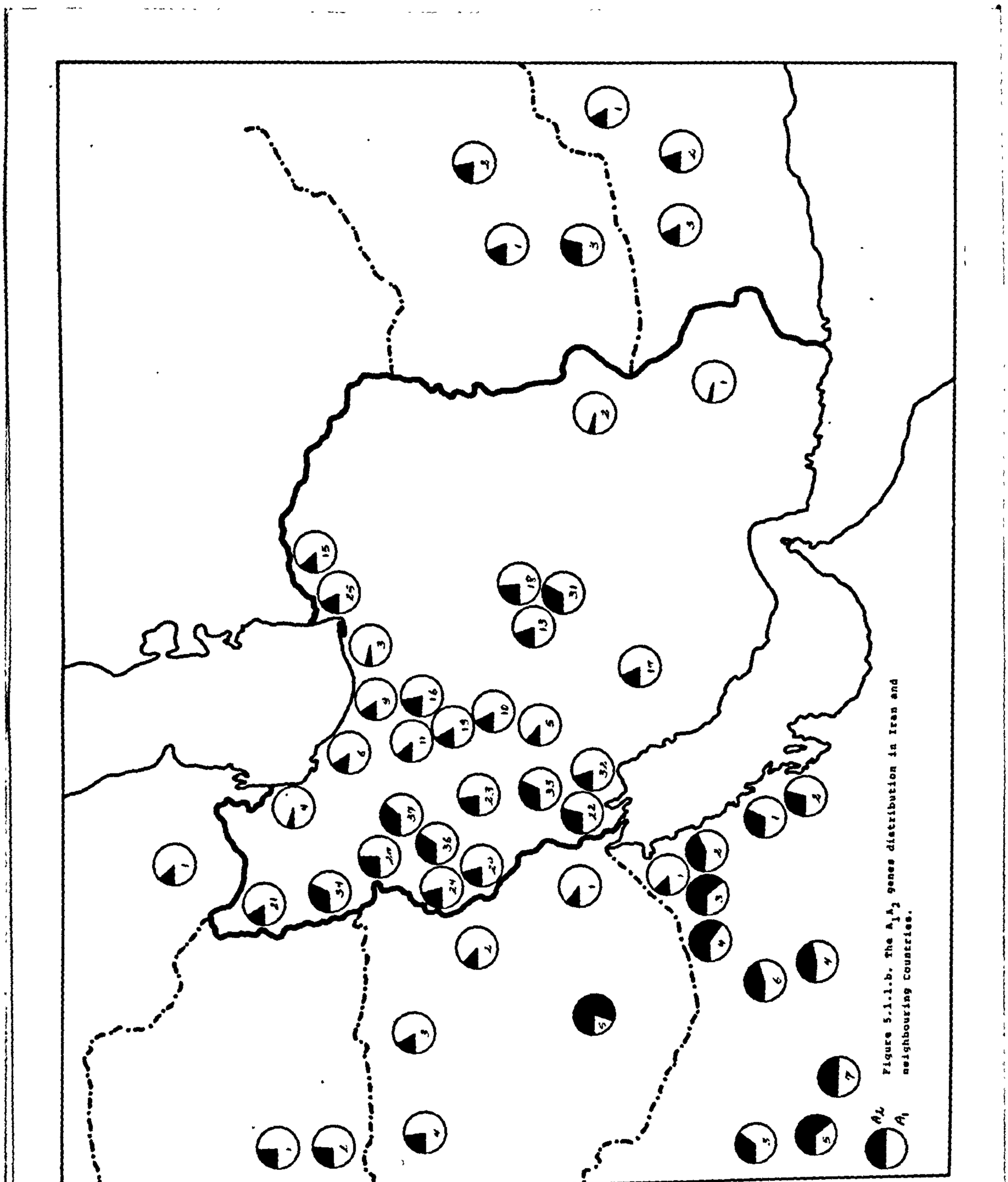


Figure 5.1.1.b. The $A_1A_2$ genes distribution in Iran and neighbouring Countries.

## 5.1.2. The Rhesus blood group system

The distribution of the Rhesus blood groups and respective gene frequencies in Iranian and neighbouring populations, expressed in term of two phenotypes after testing with one antiserum, is shown in Tables 5.1.2.a.

The frequency of the d gene ranges from 12.17 to 48.20 percent in Iranians, being lowest in the Kurdish Jews of western Iran ( Gurevitch et al, 1947) and highest in the Gilani series ( Shahsavar, Rudsar, Rasht, Langarud, Lahijan, Rudbar, Bandar-Pahlavi) of Kirk et al (1977). Values obtained in the present investigation are within this range of variation.
On the whole, with an average d gene frequency of 30.08 percent, the Iranian population appears to exhibit a much lower d gene frequency than that of around 40 percent found in Europeans ( Mourant et al, 1976).
It seems that some differences in the distribution of the d gene frequencies exist in Iran as with the exception of the Kurdish Jews and the Arabs of Khuzistan, the populations of western Iran appear to exhibit higher d gene frequencies than those found in the populations of eastern Iran. In the present investigation also two population groups, the Kurds and the Turks, of Rezaieh(north west ) show higher d gene frequencies of 39.92 and 34.73 percent compared with those of 26.53 and 22.36 percent in the population groups of Shirvan, Khorasan (north east). The d gene frequency of 26.53 percent obtained in the present investigation in the Kurds of Shirvan is also lower than all d gene frequencies reported in various studies in the Kurds of western Iran.

The d gene frequency seems also to decrease from north to south in Iran, in addition to its decreasing frequency from west to east.

The Kurds of western Iran like most other populations of the region exhibit high frequencies of the gene d but the Kurdish Jews like the Jews themselves have low frequencies of this gene.

The Arabs of south western Iran, unlike the other population groups in western Iran, also seem to exhibit low frequencies of the gene d.

The Turkmans of north eastern Iran appear to be characterized by low frequencies of the gene d, suggesting Mongoloid features.

High frequencies of the gene d seem to be characteristics of the Armenians and the Assyrians of Iran.

Regarding neighbouring populations, the frequency of the gene d in the populations of the Caucasus ranges from 21.49 percent in the Azerbaijanians of Barda (Voronov, 1973) to 41.50 percent in the Goris of Georgia( Solovyeva, 1967). With an average d gene frequency of 31.81 percent, the population of the Caucasus appears to exhibit a higher d gene frequency than that found in Iranians but lower than the European frequency.

The d gene frequency in Turkish populations varies between 29.68 percent in the Mediterranean region( Mizan et al , 1963) and 35.55 percent in the Turkish sample of Paidoussis (1967). With an average d gene frequency of 32.61 percent, the population of Turkey, like that of the Caucasus, seems to show a higher d gene frequency than that found in Iranians but still lower than the European frequency.

The frequency of the gene d in the Iraqi populations

ranges from 14.14 percent in the Kurdish Jews of the south east (Tills et al, 1977) to 52.70 percent in the Karaite Jews ( Goldschmidt et al, 1976). With an average d gene frequency of 30.13 percent, the population of Iraq appears to exhibit nearly similar d gene frequencies to those found in Iranians and lower than the European frequencies.

The extremely high d gene frequency of 52.70 percent reported by Goldschmidt et al (1976) in the Karaite Jews of Iraq, clearly indicates the strong influence of isolation and genetic drift operating in this community.

On the whole the Kurdish Jews and the Jewish populations of Iraq seem to show higher d gene frequencies compared with those found in the same two population groups of Iran.

The d gene frequency in the populations of Kuwait varies between 19.61 percent in the Suluba tribe and 39.22 percent in the Ajman tribe (Khaled et al, 1981). With an average d gene frequency of 31.34 percent, the Kuwaiti population appears to exhibit a higher d gene frequency than that found in Iranians though lower than the European frequency.

The frequency of the d gene in Saudi Arabians ranges from 26.68 percent in the Sunni sample of Qatif and Hasa Oases to 31.98 percent in the Sunni sample of Asir, Hejaz, Najran(Maranjian et al, 1966). With an average d gene frequency of 29.28 percent, the Arab population of Saudi Arabia seems to show a slightly lower d gene frequency than that found in Iranians and lower than the European frequency.

The d gene frequency of 27.44 percent reported by Kamel et al (1980) in the Abu-Dhabians of the United Arab Emirates is also lower than that found in Iranians and lower than the European frequency.

The frequency of the d gene in the populations of Pakistan ranges from 20.85 percent in the Hunza, Gilgit (Ikin et al, 1959) to 33.12 percent in Peshawar ( Boyd and Boyd, 1954).With an average d gene frequency of 27.82 percent, the population of Pakistan appears to exhibit a lower d gene frequency than Iranians and lower than the European frequency.

The d gene frequency in the Afghan populations varies between 18.41 percent in the Timuri and related tribes (Woodd-Walker et al, 1967) and 35.06 percent in the Daris (Papiha et al,1977). With an average d gene frequency of 26.91 percent, the population of Afghanistan, like that of Pakistan, seems to show a lower d gene frequency than that found in Iranians and lower than the European frequency.

The distribution of Rh types and respective gene complex frequencies in Iranian and neighbouring populations, expressed in terms of 8, 12, 18, ..... phenotypes after testing with 3,4,5,.....antisera respectively, is presented in Tables 5.1.2. b, c, d, .....

The gene complex frequencies were combined in Tables 5.1.2.* for comparative purposes.

The frequencies of the principal Rh complexes in the Iranian populations are as follows:

CDe($R_1$) ranges from 36.50 percent in a sample from north of Iran ( Bajatzadeh and Walter, 1969) to 60.50 percent in the Jews ( Gurevitch et al, 1956).

cde (r) varies between 17.02 percent in the Kurdish Jews (Godber et al, 1973) and 38.90 percent in the Armenians (Nijenhuis, 1964).

cDE($R_2$) ranges from 4.00 percent in a sample from east of Iran (Bajatzadeh and Walter, 1969) to 25.56 percent in the Ma-

massani tribe of Fars ( Bowman, 1969).

cDe(Ro) varies between zero in the Kurdish Jews (Godber et al, 1973, and Tills et al, 1977); in the Zabolis of Sistan and Baluchistan (present study); and in the Ghashghais (Nijenhuis, 1964), and 20.58 percent in the Arabs of Khuzistan ( Marzban, 1978).

Furthermore, the frequencies of the rare gene complexes do not show much variation. Values obtained in the present investigation are within this range of variation.

The approximate frequencies of Rh gene complexes in the English population are as follows:

| Gene complex | % frequency |
|---|---|
| CDe($R_1$) | 41 |
| cde(r ) | 39 |
| cDE($R_2$) | 14 |
| cDe(Ro) | 3 |
| $C^wDe(R_1^w)$ | 1 |
| Cde(r') | 1 |
| cdE(r") | 1 |
| CDE($R_Z$) | 0.2 |

Most northern and central European populations differ only slightly (Mourant et al, 1976).

On the whole, with average Rh gene complex frequencies of:

| Gene complex | % frequencies |
|---|---|
| CDe($R_1$) | 48 |
| cde(r) | 28 |
| cDE($R_2$) | 14 |
| cDe(Ro) | 6 |
| Cde(r') | 3 |
| CDE($R_z$) | 1 |
| cdE(r") | 0.57 |

the Iranian population appears to exhibit much lower cde (r) but higher CDe($R_1$) and cDe(Ro) gene complex frequencies than those found in northern European populations. The Rh gene complex frequencies in Iranians seem to be similar to those found in southern Europe and the Mediterranean region.

The Kurdish Jews of western Iran are characterized by the relatively low frequency of the gene complex cde(r), but high frequencies of both the CDe($R_1$) and cDE($R_2$) gene complexes.

The Iranian Jews appear to exhibit low frequencies of both the gene complex cde(r) and cDE($R_2$), but very high CDe($R_1$) frequency.

The Arabs of south western Iran seem to show low frequencies of both the gene complexes cde(r) and cDE($R_2$), but high frequencies of CDe($R_1$) and African cDe(Ro).

The Turkmans of north eastern Iran appear to be characterized by low frequency of the gene complex cde(r), but a high CDe($R_1$) frequency, suggesting Mongoloid features.

High frequencies of both the gene complexes cde(r) and cDE ($R_2$), but a low frequency of CDe($R_1$) seem to be characteristics of the Armenians and the Assyrians of Iran.

Regarding neighbouring populations, the frequencies of the principal Rh complexes in the populations of the Caucasus are as follows:

CDe($R_1$) ranges from 34.76 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al,1972 ) to 46.30 percent in the

Georgians (Kherumian et al, 1954).

cde (r) varies between 34.09 percent in the Georgians (Kherumian et al, 1954) and 35.61 percent in the Svanis of Georgia, Abkhazskaya ( Verbitsky et al, 1972 ).

cDE($R_2$)ranges from 15.61 percent in the Armenians (Kherumian et al, 1954) to 17.73 percent in the Svanis of Georgia, Abkhazskaya ( Verbitsky et al, 1972 ).

cDe(Ro) varies between 3.25 percent in the Georgians and 4.82 percent in the Armenians(Kherumian et al, 1954). With average Rh gene complex frequencies of :

| Gene complex | % Frequency |
|---|---|
| CDe($R_1$) | 41 |
| cde(r ) | 35 |
| cDE($R_2$) | 17 |
| cDe(Ro) | 4 |
| Cde(r') | 2 |
| CDE($R_Z$) | 1 |
| cdE(r") | 0.26 |

the population of the Caucasus seems to exhibit higher cde(r) but lower CDe($R_1$) and cDe(Ro) gene complex frequencies than those found in Iranians and more similar to the European frequencies.

The frequencies of the principal Rh complexes in the populations of Turkey( Aksoy et al, 1958) are as follows:

CDe($R_1$) ranges from 48.18 percent in the Turks to 54.66 percent in the Eti-Turks.

cde(r) varies between 30.85 percent in the Eti-Turks and 32.00 percent in the Turks.

cDE($R_2$) ranges from 13.14 percent in the Eti-Turks to 17.14 percent in the Turks.

cDe(Ro) varies between 1.30 percent in the Turks and 1.35 percent in the Eti-Turks.

With average Rh gene complex frequencies of:

| Gene complex | %Frequency |
|---|---|
| CDe($R_1$) | 51 |
| cde(r) | 32 |
| cDE($R_2$) | 15 |
| cDe(Ro) | 1 |
| Cde(r') | 1 |
| CDE($R_Z$) | 0 |
| cdE(r") | 0 |

the Turkish population appears to exhibit higher frequencies of both cde(r) and CDe($R_1$) gene complexes compared with those found in Iranians, but surprisingly much lower African cDe(Ro) values which are relatively common in Iranian and in nearly every other Mediterranean populations.

The frequencies of the principal Rh complexes in the populations of Iraq are as follows:

CDe ($R_1$) ranges from 29.93 percent in the Karaite Jews (Goldschmidt et al, 1976) to 64.51 percent in the Kurdish Jews of the south east ( Tills et al, 1977).

cde(r) varies between 7.49 percent in the Kurdish Jews of the south east ( Tills et al, 1977) and 52.70 percent in the Karaite Jews ( Goldschmidt et al, 1976).

cDE($R_2$) ranges from 3.47 percent in the Karaite Jews (Goldschmidt et al, 1976) to 20.51 percent in the Kurdish Jews of the south east ( Tills et al, 1977).

cDe(Ro) varies between zero in the Kurdish Jews (Godber et al, 1973; and Tills et al, 1977) and 20.90 percent in the Karaite Jews ( Goldschmidt et al, 1976). With average Rh gene complex frequencies of :

| Gene complex | %Frequency |
|---|---|
| CDe($R_1$) | 48 |
| cde(r) | 27 |
| cDE($R_2$) | 15 |
| cDe(Ro) | 6 |
| Cde(r') | 1 |
| CDE($R_Z$) | 3 |
| cdE(r") | 0.30 |

the population of Iraq seems to exhibit Rh gene complex frequencies similar to those found in Iranians.

The very high frequency of the so called "African gene",cDe ( 20.90 percent ) in the Karaite Jews of Iraq is notable, as is the frequency of cde (52.70 percent). This results clearly indicate the strong influence of isolation and genetic drift operating in this community.

The frequencies of the principal Rh complexes in the populations of Kuwait are as follows:

CDe($R_1$) ranges from 30.00 percent in the Ajman tribe (Khaled et al, 1981) to 49.20 percent in the Kuwaiti Arabs (Sawhney, 1975).

cde(r) varies between 15.00 percent in the Suluba tribe and 37.00 percent in the Ajman tribe (Khaled et al, 1981).

cDE ($R_2$) ranges from 5.04 percent in the Kuwaiti Arabs(Sawhney, 1975) to 33.00 percent in the Suluba tribe (Khaled et al, 1981).

cDe(Ro) varies between 3.00 percent in the Suluba tribe(Khaled et al, 1981) and 14.42 percent in the Kuwaiti sample of Onsi et al (1969). With average Rh gene complex frequencies of:

| Gene complex | % Frequency |
|---|---|
| CDe($R_1$) | 41 |
| cde (r) | 28 |
| cDE ($R_2$) | 18 |
| cDe (Ro) | 9 |
| Cde (r') | 2 |
| CDE ($R_z$) | 1 |
| cdE (r") | 1 |

the population of Kuwait appears to exhibit lower CDe($R_1$) but higher African cDe(Ro) frequencies than those found in Iranians.

The frequencies of the principal Rh complexes in the po-

pulations of Saudi Arabia are as follows:

CDe($R_1$) ranges from 38.46 percent in the Sunni sample of Qatif and Hasa Oases to 42.46 percent in the Sunni sample of Asir, Hejaz, Najran (Maranjian et al, 1966).

cde (r) varies between 25.02 percent in Saudi Arabians and 28.74 percent in the Sunni sample of Asir, Hejaz, Najran (Maranjian et al, 1966).

cDE($R_2$) ranges from 9.96 percent in western Saudi Arabia(Saha et al, 1980) to 16.72 percent in the Sunni sample of Qatif and Hasa Oases (Maranjian et al, 1966).

cDe(Ro) varies between 14.77 percent in the Sunni sample of Asir, Hejaz, Najran (Maranjian et al, 1966) and 22.61 percent in western Saudi Arabia (Saha et al, 1980). With average Rh gene complex frequencies of :

| Gene complex | % Frequency |
|---|---|
| CDe( $R_1$ ) | 14 |
| cde ( r ) | 26 |
| cDE ($R_2$ ) | 13 |
| cDe (Ro ) | 18 |
| Cde ( r') | 1 |
| CDE ($R_Z$ ) | 0.25 |
| cdE (r" ) | 1 |

the Arab population of Saudi Arabia, like that of Kuwait,seems to exhibit lower frequencies of both CDe($R_1$) and cde ( r ) , but much higher African cDe(Ro) frequency than those found in Iranians.

Generally, if seems that the frequency of the typical negro gene cDe(Ro) in the Arab populations is higher than in Europeans, but lower than in Africans. Rh is only one of the several systems suggesting the flow of African genes in the Arabs

of Peninsula (Mourant et al, 1976). On the whole, it appears that the gene frequency distribution of the Rh system in Arab populations is intermediate between Caucasoids and Negroids.

The frequencies of the Rh principal complexes in the populations of Pakistan are as follows:

CDe ($R_1$) ranges from 55.02 percent in the Baltis of west Pakistan (Clegg et al, 1961) to 66.69 percent in the Moslems of Punjab and west Pakistan (Chaudhri et al, 1952.).

cde (r) varies between 17.08 percent in Dacca, Bengal (Boyd et al,1954) and 28.26 percent in the Hunza , Gilgit( Ikin et al, 1959).

cDE ($R_2$) ranges from 7.71 percent in Dacca, Bengal (Boyd et al, 1954) to 21.39 percent in the Baltis of west Pakistan(Clegg et al, 1961).

cDe(Ro) varies between zero in the Hunza, Gilgit (Ikin et al, 1959) and 7.21 percent in the Punjabis of Lahore (Boyd et al, 1954). With average Rh gene complex frequencies of:

| Gene complex | % Frequency |
|---|---|
| CDe ($R_1$) | 60 |
| cde ( r) | 24 |
| cDE($R_2$ ) | 11 |
| cDe(Ro ) | 3 |
| Cde(r' ) | 1.50 |
| CDE($R_Z$ ) | 0.76 |
| cdE(r'' ) | 0.10 |

the population of Pakistan appears to exhibit lower frequencies of both cde (r ) and cDe(Ro), but much higher CDe($R_1$)frequency than those found in Iranians.

The frequencies of the Rh principal complexes in the populations of Afghanistan are as follows:

CDe($R_1$) ranges from 46.10 percent in the Pushtus (Papiha et al, 1977) to 60.61 percent in the Timuri and related tribes (woodd-Walker et al, 1967).

cde (r) varies between 13.63 percent in the Timuri and related tribes (Woodd-Walker et al, 1967) and 28.60 percent in the Daris (Papiha et al, 1977).

cDE($R_2$) ranges from 13.80 percent in the Daris (Papiha et al, 1977) to 24.15 percent in the Timuri and related tribes (Woodd-Walker et al, 1967).

cDe (Ro) varies between 1.61 percent in the Timuri and related tribes (Woodd-Walker et al, 1967) and 6.80 percent in the Pushtus (Papiha et al, 1977).

With average Rh gene complex frequencies of:

| Gene complex | % Frequency |
|---|---|
| CDe($R_1^1$) | 53 |
| cde(r) | 23 |
| cDE($R_2$) | 19 |
| cDe(Ro) | 4 |
| Cde(r') | 0.26 |
| CDE($R_Z$) | 0.13 |
| cdE(r") | 0 |

the population of Afghanistan, like that of Pakistan, seems to exhibit lower frequencies of both cde(r) and cDe(Ro), but much higher CDe($R_1$) frequency than those found in Iranians. The low frequency of cde(r) seems to be suggestive of some Mongoloid features in the Afghan population.

Conclusion

In northern Europe the frequency of the d gene is about 40 percent and the frequencies of the principal Rh complexes are approximately:

CDe($R_1$) 40 percent; cDE($R_2$) 15 percent; cde(r) 40 percent; cDe (Ro) 3 percent.

In southern Europe and the Mediterranean area generally, the frequency of cde(r) is usually much lower and that of CDe ($R_1$) higher than further north; and the variations between populations are greater.

South-western Asia, including most of the Indian region, shows a similar Rh distribution to the Mediterranean region but with a Marked admixture of African genes in the Arab countries.

Africa, south of Sahara Desert, shows a uniform absolute preponderance of cDe(Ro) which is unique. Here the cde(r)complex has a frequency of about 20 percent and there are lower frequencies of CDe($R_1$) and cDE($R_2$) (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arab populations of Kuwait and Saudi Arabia with their lower CDe($R_1$) and much higher African cDe(Ro), the frequencies of the total d gene and the cde(r)complex appear to be much lower but that of the CDe($R_1$) complex higher than those found in Europeans. Therefore with slightly lower cde (r) and higher CDe($R_1$), the frequencies of the principal Rh complexes in Iranian and neighbouring populations seem to be more similar to those in southern European and Mediterranean populations.

In general, regarding the whole area under discussion,the frequencies of the total d gene and the cde(r) complex seem to decrease from the west(Turkey) to the east (Pakistan) and that of CDe($R_1$) increases.

Table 5.1.2. a I

Rh blood groups distribution in Iran tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | | |
| 1-Kurdish Jews | 203 | 200 | 3 | 87.83 | 12.17 | Gurevitch,J.,et al. | 1947 |
| 2-Turkmans | 1263 | 1244 | 19 | 87.74 | 12.26 | Montazami, K. | 1978 |
| 3-Arabs.Khuzestan | 97 | 94 | 3 | 82.41 | 17.59 | Marzban, M. | 1978 |
| 4-Southern Gorgan, Behshahr,Sari | 53 | 51 | 2 | 80.60 | 19.40 | Kirk,R.L.,et al. | 1977 |
| 5-Mashhad | 11240 | 10767 | 473 | 79.49 | 20.51 | Afkari,A.H. | 1967 |
| 6-Jews | 225 | 214 | 11 | 77.89 | 22.11 | Silberstein W., & Goldstein. | 1958 |
| 7-Jews | 200 | 190 | 10 | 77.64 | 22.36 | Gurevitch,J.,et al. | 1956 |
| 8-Turks.Shirvan,Khorasan | 160 | 152 | 8 | 77.64 | 22.36 | Present study. | |
| 9-Kerman | 317 | 301 | 16 | 77.53 | 22.47 | Present study. | |
| 10-Kurdish Jews | 94 | 89 | 5 | 76.94 | 23.06 | Godber, Marilyn.J., et al. | 1973 |
| 11-Turkmans | 292 | 276 | 16 | 76.59 | 23.41 | Boue' and Boue'. | 1956 |
| 12-Arabs.Abadan | 158 | 149 | 9 | 76.13 | 23.87 | Nijenhuis, L.E. | 1964 |

Table 5.1.2.a. I (Cont.) $^{Rh}$ blood groups distribution in Iran tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | |
| 13-Zoroastrians. Yazd | 182 | 171 | 11 | 75.42 | 24.58 | Boue' and Boue'. 1956 |
| 14-Lurs.Luristan | 162 | 152 | 10 | 75.16 | 24.84 | Present study. |
| 15-Gonbad | 143 | 134 | 9 | 74.90 | 25.10 | Kirk,R.L., et al. 1977 |
| 16-Baluchis.Sistan & Baluchistan | 111 | 104 | 7 | 74.89 | 25.11 | Present study. |
| 17-Bakhtiaris | 138 | 129 | 9 | 74.46 | 25.54 | Nijenhuis, L.E. 1964 |
| 18-Shahsavan.Azari Dashte Moghan | 137 | 128 | 9 | 74.37 | 25.63 | Boue' and Boue'. 1956 |
| 19-Kurdish Jews | 106 | 99 | 7 | 74.30 | 25.70 | Tills, D., et al. 1977 |
| 20-Tehran | 992 | 925 | 67 | 74.01 | 25.99 | Roshan, G.,et al. 1969 |
| 21-Gorgan | 133 | 124 | 9 | 73.99 | 26.01 | Boue' and Boue'. 1956 |
| 22-Ghashghais.Abadeh | 1516 | 1412 | 104 | 73.81 | 26.19 | Montazami,K. 1978 |
| 23-Khuzestan | 2043 | 1902 | 141 | 73.73 | 26.27 | Montazami, K. 1978 |
| 24-Moslems. Yazd | 145 | 135 | 10 | 73.73 | 26.27 | Boue' and Boue'. 1956 |
| 25-Bassaris.Fars | 101 | 94 | 7 | 73.67 | 26.33 | Bowman, J.E. 1959 |

Table 5.1.2.a.I (Cont.)

Rh blood groups distribution in Iran tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | | |
| 26-Kurds.Shirvan,Khorasan | 142 | 132 | 10 | 73.47 | 26.53 | Present study. | |
| 27-Mamassanis.Fars | 113 | 105 | 8 | 73.39 | 26.61 | Bowman, J.E. | 1959 |
| 28-Moslems.Fars | 768 | 712 | 56 | 73.00 | 27.00 | Bowman,J.E., & Ronaghy, H. | 1967 |
| 29-Mobarakeh.Esfahan | 1211 | 1121 | 90 | 72.74 | 27.26 | Montazami, K. | 1978 |
| 30-Kurds.Sanandaj | 107 | 99 | 8 | 72.66 | 27.34 | Lehmann, H.,et al. | 1973 |
| 31-Shiraz | 1171 | 1083 | 88 | 72.59 | 27.41 | Tahmasbian,A. | 1972 |
| 32-Gilan.Turkish speakers | 198 | 183 | 15 | 72.48 | 27.52 | Boue' and Boue'. | 1956 |
| 33-Tehran | 1196 | 1103 | 93 | 72.12 | 27.88 | Defai, H. | 1971 |
| 34-Kurds.Baneh,Marivan | 77 | 71 | 6 | 72.09 | 27.91 | Lehmann,H.,et al. | 1973 |
| 35-Esfahan | 4385 | 4031 | 354 | 71.59 | 28.41 | Montazami, K. | 1978 |
| 36-Tehran | 500 | 457 | 43 | 70.67 | 29.33 | Boue' and Boue'. | 1956 |
| 37-Shiraz | 16368 | 14942 | 1426 | 70.49 | 29.51 | Mohallatee,E.A., & Haghshenas. | 1969 |

Table 5.1.2.a.I(Cont.) Rh blood groups distribution in Iran tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | | |
| 38-North west | 68 | 62 | 6 | 70.30 | 29.70 | Bajatzadeh,M., & Walter, H. | 1969 |
| 39-Azarbaijan | 1467 | 1326 | 131 | 70.12 | 29.88 | Montazami.K. | 1978 |
| 40-Jews | 108 | 98 | 10 | 69.57 | 30.43 | Tabatabai, H. | 1977 |
| 41-Moslems.Tehran | 203 | 184 | 19 | 69.41 | 30.59 | Boue' and Boue'. | 1956 |
| 42-South east | 348 | 315 | 33 | 69.21 | 30.79 | Njenhuis, L.E. | 1964 |
| 43-Zabolis.Sistan & Baluchistan | 115 | 104 | 11 | 69.07 | 30.93 | Present study. | |
| 44-Babol,Shahi,Amol | 70 | 63 | 7 | 68.40 | 31.60 | Kirk,R.L.,et al. | 1977 |
| 45-Esfahan | 9753 | 8779 | 974 | 68.40 | 31.60 | Mirdamadi,M.,et al. | 1978 |
| 46-Tehran | 20000 | 18000 | 2000 | 68.38 | 31.62 | Vazin, H. | 1969 |
| 47-Kurds.Sanandaj | 129 | 116 | 13 | 68.26 | 31.74 | Boue' and Boue'. | 1955 |
| 48-Tavalesh.Astara | 49 | 44 | 5 | 68.10 | 31.90 | Kirk, R.L.,et al. | 1977 |
| 49-Tehran | 150000 | 134640 | 15360 | 68.00 | 32.00 | Tehranchian .. | 1963 |

Table 5.1.2.a.I (Cont.) Rh blood groups distribution in Iran tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | | |
| 50-Assyrians.Abadan | 32 | 29 | 3 | 67.84 | 32.16 | Nijenhuis,L.E. | 1964 |
| 51-Hamadan,Akinlou Turkish Speakers | 115 | 103 | 12 | 67.70 | 32.30 | Boue' and Boue'. | 1956 |
| 52-Ghashghais | 66 | 59 | 7 | 67.43 | 32.57 | Nijenhuis,L.E. | 1964 |
| 53-Tehran | 42330 | 37631 | 4699 | 66.68 | 33.32 | Berelian-Jahanshahi,F. | 1973 |
| 54-Tehran | 302 | 268 | 34 | 66.40 | 33.60 | Present study. | |
| 55-Turks.Rezaieh | 141 | 124 | 17 | 65.27 | 34.73 | Present study. | |
| 56-Shi'a Yazd | 270 | 237 | 33 | 65.04 | 34.96 | Sunderland,E., & Smith,H.M. | 1966 |
| 57-Tehran | 196 | 172 | 24 | 65.01 | 34.99 | Sawhney, K.S. | 1975 |
| 58-Zoroastrians | 88 | 77 | 11 | 64.64 | 35.36 | Present study. | |
| 59-Kurds.Kermanshah | 127 | 111 | 16 | 64.51 | 35.49 | Nijenhuis, L.E. | 1964 |
| 60-Esfahan | 110 | 96 | 14 | 64.33 | 35.67 | Sawhney,K.S. | 1975 |
| 61-Kurds.Mahabad | 125 | 108 | 17 | 63.12 | 36.88 | Boue' and Boue'. | 1955 |

Table 5.1.2.a I.(Cont.) Rh blood groups distribution in Iran tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | | |
| 62-Northern Gorgan | 43 | 37 | 6 | 62.60 | 37.40 | Kirk, R.L.,et al. | 1977 |
| 63-East | 70 | 60 | 10 | 62.20 | 37.80 | Bajatzadeh,M., & Walter, H. | 1969 |
| 64-Central & South | 109 | 93 | 16 | 61.69 | 38.31 | Bajatzadeh,M., & Walter, H. | 1969 |
| 65-Armenians | 144 | 122 | 22 | 60.91 | 39.09 | Tabatabai, H. | 1977 |
| 66-Kurds.Rezaieh | 138 | 116 | 22 | 60.08 | 39.92 | Present study. | |
| 67-Armenians.Esfahan | 144 | 119 | 25 | 58.33 | 41.67 | Bowman, J.E. | 1959 |
| 68-Tehran | 113 | 93 | 20 | 57.93 | 42.07 | Bajatzadeh,M., & Walter, H. | 1969 |
| 69-Armenians | 78 | 64 | 14 | 57.63 | 42.37 | Nijenhuis, L.E. | 1964 |
| 70-West | 107 | 86 | 21 | 55.70 | 44.30 | Bajatzadeh,M., & Walter H. | 1969 |
| 71-Tehran | 1432 | 1145 | 287 | 55.23 | 44.77 | Sadatzadeh,H., Hamidi, A.H. | 1972 |

Table 5.1.2.a.I (Cont.) Rh blood groups distribution in Iran tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | | |
| 72-North | 63 | 49 | 14 | 52.86 | 47.14 | Bajatzadeh,M., & Walter, H. | 1969 |
| 73- Shahsavar, Rudsar, Rudbar, Rasht, Langarud, Lahijan,Bandar Pahlavi | 86 | 66 | 20 | 51.80 | 48.20 | Kirk,R.L., et al. | 1977 |

Table 5.1.2.a. II Rh blood groups distribution in the Caucasus tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | | |
| 1-Azerbaijanians.Barda | 136 | 130 | 6 | 78.51 | 21.49 | Voronov, A.A. | 1973 |
| 2-Kakhetians.Gurgaani | 195 | 184 | 11 | 76.25 | 23.75 | Voronov, A.A. | 1973 |
| 3-Georgia, Gurdzhaani. Autochthonous | 699 | 653 | 46 | 74.35 | 25.65 | Voronov, A.A.,& Potapov, M.I.. | 1967 |
| 4-Azerbaijanians Shemakha | 176 | 162 | 14 | 71.79 | 28.21 | Voronov, A.A. | 1973 |
| 5-Azerbaijanians.Nukha | 191 | 175 | 16 | 71.06 | 28.94 | Voronov, A.A. | 1973 |
| 6-Megrelians.Mikha-Tsakai | 121 | 108 | 13 | 67.23 | 32.77 | Voronov, A.A. | 1973 |
| 7-Georgian abroad.Autochthonous | 40 | 35 | 5 | 64.65 | 35.35 | Kherumian,R.,et al. | 1954 |
| 8-Georgians.Different region | 802 | 695 | 107 | 63.48 | 36.52 | Vaisblit,R.I.,et al. | 1965 |
| 9-Armenians.Autochthonous | 199 | 171 | 28 | 62.49 | 37.51 | Kherumian,R.,et al. | 1954 |

Table 5.1.2.a. II (Cont.) Rh blood groups distribution in the Caucasus tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | |
| 10-Svanis. Georgia.Abkhazskaya | 643 | 549 | 94 | 61.77 | 38.23 | Verbitsky,M.Sh., et al. 1972 |
| 11-Goris.Georgia | 331 | 274 | 57 | 58.50 | 41.50 | Solovyeva,T.G. 1967 |

Table 5.1.2.a.III

Rh blood groups distribution in Turkey tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | | |
| 1-Mediterranean region | 3122 | 2847 | 275 | 70.32 | 29.68 | Mizan,N., et al. | 1963 |
| 2-Asia Minor.Aegean region | 3166 | 2875 | 291 | 69.68 | 30.32 | Mizan,N., et al. | 1963 |
| 3-Eti-Turks.Asia Minor.near Mersin | 118 | 107 | 11 | 69.47 | 30.53 | Aksoy,M., et al. | 1958 |
| 4-Western part | 4096 | 3711 | 385 | 69.34 | 30.66 | Mizan,N., et al. | 1963 |
| 5-Anatolia,South-Eastern | 5200 | 4695 | 505 | 68.84 | 31.16 | Mizan,N., et al. | 1963 |
| 6-Middle part | 5453 | 4890 | 563 | 67.88 | 32.12 | Mizan,N., et al. | 1963 |
| 7-Marmar and Thrace | 8430 | 7517 | 913 | 67.09 | 32.91 | Mizan,N., et al. | 1963 |
| 8-Anatolia Central | 7023 | 6255 | 768 | 66.92 | 33.08 | Mizan,N., et al. | 1963 |
| 9-Black sea region. western | 3612 | 3202 | 410 | 66.31 | 33.69 | Mizan,N., et al. | 1963 |
| 10-Anatolia,Eastern | 7598 | 6684 | 914 | 65.32 | 34.68 | Mizan,N., et al. | 1963 |
| 11-Turks.Asia Minor.Mersin | 108 | 95 | 13 | 65.31 | 34.69 | Aksoy,M., et al. | 1958 |

Table 5.1.2.a. III (Cont.) Rh blood groups distribution in Turkey tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | |
| 12-Black sea region. Eastern | 5295 | 4650 | 645 | 65.10 | 34.90 | Mizan, N., et al. 1963 |
| 13-Turks abroad.Recruits | 364 | 318 | 46 | 64.45 | 35.55 | Paidoussis, M. 1967 |

Table 5.1.2.a:IV Rh blood groups distribution in Iraq tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | | |
| 1-Kurdish Jews. south east | 50 | 49 | 1 | 85.86 | 14.14 | Tills,D., et al. | 1977 |
| 2-Kurdish Jews | 129 | 124 | 5 | 80.31 | 19.69 | Gurevitch,J., et al. | 1956 |
| 3-Jews | 1146 | 1063 | 83 | 73.09 | 26.91 | Levence, C. | 1968 |
| 4-Kurdish Jews | 250 | 231 | 19 | 72.44 | 27.56 | Gurevitch,J., et al. | 1953 |
| 5-Jews.Baghdad | 162 | 149 | 13 | 71.67 | 28.33 | Gurevitch,J., et al. | 1956 |
| 6-Kurdish Jews.North West | 61 | 56 | 5 | 71.37 | 28.63 | Tills,D., et al. | 1977 |
| 7-Military donors | 1067 | 977 | 90 | 70.97 | 29.03 | Fakhrie, S.D., et al. | 1966 |
| 8-Paid donors | 4056 | 3697 | 359 | 70.25 | 29.75 | Fakhrie, S.D., et al. | 1966 |
| 9-Baghdadi Jews | 200 | 181 | 19 | 69.18 | 30.82 | Sirat,Satyavati M. | 1956 |
| 10-Assyrians | 99 | 89 | 10 | 68.22 | 31.78 | Ikin,Elizabeth W.,et al. | 1965 |
| 11-Jews | 308 | 276 | 32 | 67.77 | 32.23 | Silberstein,W., & Goldstein. | 1958 |
| 12-Civilian donors | 1185 | 1060 | 125 | 67.52 | 32.48 | Fakhrie,S.D., et al. | 1966 |

Table 5.1.2.a. IV (Cont.) Rh blood groups distribution in Iraq tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | |
| 13-Kurdish Jews | 27 | 24 | 3 | 66.67 | 33.33 | Godber,Marilyn J., et al. 1973 |
| 14-Arabs.Baghdad | 300 | 264 | 36 | 65.36 | 34.64 | Kayssi, A.I. 1940 |
| 15-Karaite Jews | 72 | 52 | 20 | 47.30 | 52.70 | Goldschmidt,E.,et al. 1976 |

Table 5.1.2. a V

Rh blood groups distribution in Kuwait tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | | |
| 1-Suluba tribe | 52 | 50 | 2 | 80.39 | 19.61 | Khaled, E., et al. | 1981 |
| 2-Kuwaitis | 2328 | 2154 | 174 | 72.67 | 27.33 | Onsi,A., et al. | 1969 |
| 3-General population | 74 | 65 | 9 | 65.13 | 34.87 | Khaled,E.,et al. | 1981 |
| 4-Kuwaiti Arabs | 110 | 96 | 14 | 64.33 | 35.67 | Sawhney, K.S. | 1975 |
| 5-Ajman tribe | 52 | 44 | 8 | 60.78 | 39.22 | Khaled,E., et al. | 1981 |

Table 5.1.2.a. VI Rh blood groups distribution in Saudi Arabia tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | |
| 1-Sunnis. Qatif and Hasa Oases | 323 | 300 | 23 | 73.32 | 26.68 | Maranjian,G.,et al. 1966 |
| 2-Saudi Arabians | 355 | 328 | 27 | 72.42 | 27.58 | Goedde,H.W., et al. 1979 |
| 3-Western Saudi Arabia | 178 | 161 | 17 | 69.10 | 30.90 | Saha,N., et al. 1981 |
| 4-Sunnis. Asir,Hejaz,Najran | 176 | 158 | 18 | 69.02 | 31.98 | Maranjian,G.,et al. 1966 |

Table 5.1.2. a VII

Rh blood groups distribution in the United Arab Emirates tested with anti -D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | |
| 1-Abu-Dhabians | 624 | 577 | 47 | 72.56 | 27.44 | Kamel,K.,et al. 1980 |

Table 5.1.2. a. VIII Rh blood groups distribution in Pakistan tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | D+ | D- | D | d´ | | |
| 1-Hunza.Gilgit | 23 | 22 | 1 | 79.15 | 20.85 | Ikin,Elizabeth W., et al. | 1959 |
| 2-Bengalis.Dacca | 236 | 222 | 14 | 75.64 | 24.36 | Boyd,W.C., & Boyd, lyle.G. | 1954 |
| 3-Moslems.Borne in Punjab and West Pakistan | 101 | 95 | 6 | 75.63 | 24.37 | Chaudhri,I.M., et al. | 1952 |
| 4- Punjabis.Lahore | 203 | 189 | 14 | 73.74 | 26.26 | Boyd,W.C., & Boyd, lyle.G. | 1954 |
| 5-Moslems.Karachi | 150 | 139 | 11 | 72.92 | 27.08 | Moten,A.N., et al. | 1956 |
| 6-Baltis.Baltistan | 80 | 74 | 6 | 72.61 | 27.39 | Clegg,E.J., et al. | 1961 |
| 7-Pathans.Swat. Saidu Sharif | 133 | 121 | 12 | 69.96 | 30.04 | Alciati, G. | 1968 |
| 8-Pathan. Dari,Timurgara | 78 | 70 | 8 | 67.97 | 32.02 | Bernhard, W. | 1967 |
| 9-Parsis.Karachi | 103 | 92 | 11 | 67.32 | 32.68 | Moten,A.N.,et al. | 1956 |
| 10-Peshawar | 155 | 138 | 17 | 66.88 | 33.12 | Boyd,W.C.,& Boyd Lyle.G. | 1954 |

Table 5.1.2.a. IX Rh blood groups distribution in Afghanistan tested with anti-D serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | D+ | D- | D | d | |
| 1-Timuri and related tribes | 118 | 114 | 4 | 81.59 | 18.41 | Woodd-Walker,R.B., et al. 1967 |
| 2-Fara,Girishk | 98 | 92 | 6 | 75.26 | 24.74 | Boue',A., et al. 1956 |
| 3-Pushtus | 104 | 95 | 9 | 70.58 | 29.42 | Papiha,S.S., et al. 1977 |
| 4-Daris | 179 | 157 | 22 | 64.94 | 35.06 | Papiha,S.S., et al. 1977 |

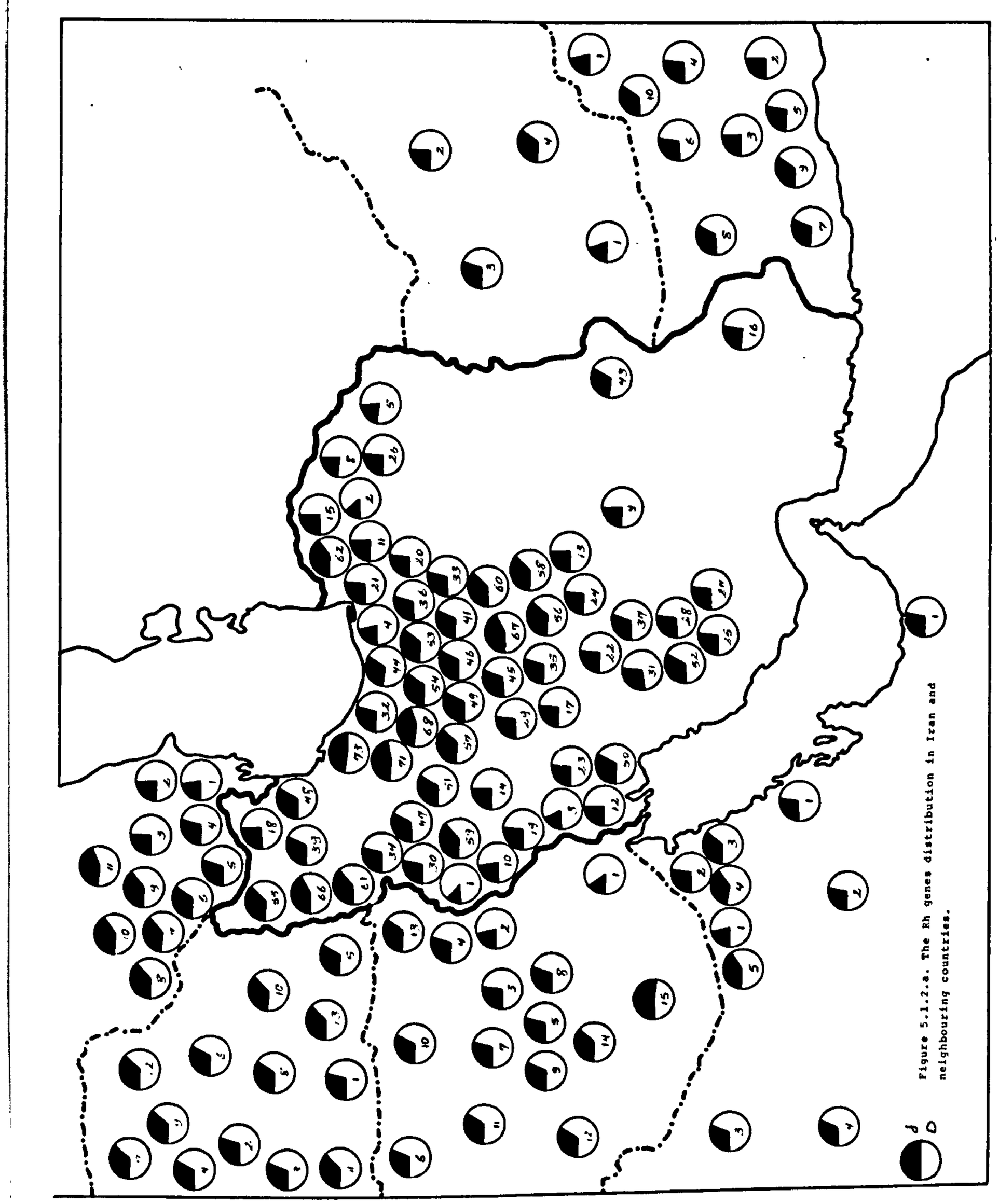


Figure 5.1.2.a. The Rh genes distribution in Iran and neighbouring countries.

Table 5.1121b.I

Rh blood groups distribution in Iran tested with anti-C, anti-D, and anti-E sera

| Population | Number Tested | Phenotypes | | | | | | | | Gene complex frequencies | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDee | CCddEE | CCddee | ccDEE | ccDee | ccddEE | ccddee | CDe | Cde | cDE | cDe | cdE | cde | |
| 1-Zoroastrians . Yazd | 182 | 42 | 111 | - | 2 | 16 | 2 | - | 9 | 57.79 | 2.61 | 17.15 | 2.14 | 0.00 | 20.31 | Boue' & Boue'. 1956 |

Table 5.1.2.c.I

Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, and anti-c sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDee | CCddEE | CCddee | CcDEE | CcDee | CcddEE | Ccddee | ccDEE | ccDee | ccddEE | ccddee |
| 1-Turkmans. Gonbad | 292 | 3 | 104 | - | - | 34 | 82 | - | 3 | 44 | 9 | - | 13 |
| 2-Gorgan | 133 | - | 46 | - | - | 19 | 45 | - | 1 | 7 | 7 | - | 8 |
| 3-Jews | 200 | - | 73 | - | - | 26 | 70 | - | - | 15 | 6 | - | 10 |
| 4-Gilan.Turkish Speakers | 198 | 4 | 49 | - | 1 | 43 | 56 | - | 1 | 27 | 4 | - | 13 |
| 5-North West | 68 | 1 | 17 | - | - | 10 | 23 | - | 2 | 7 | 4 | - | 4 |
| 6-Kurds.Sanandaj | 129 | 3 | 21 | - | 1 | 26 | 49 | - | 2 | 9 | 8 | - | 10 |
| 7-Kurds.Mahabad | 125 | 3 | 34 | - | - | 18 | 35 | - | 2 | 6 | 12 | - | 15 |
| 8-Moslems.Tehran | 203 | - | 38 | - | - | 42 | 82 | - | 2 | 18 | 4 | - | 17 |
| 9-Tehran | 113 | 3 | 24 | - | - | 11 | 36 | - | 6 | 10 | 9 | 2 | 12 |
| 10-North | 63 | 1 | 16 | - | - | 1 | 19 | - | 4 | 6 | 6 | 3 | 7 |
| 11-Central and South | 109 | 4 | 28 | - | - | 5 | 41 | - | 2 | 7 | 8 | - | 14 |
| 12-East | 70 | 2 | 17 | - | - | 5 | 29 | - | 1 | 3 | 4 | - | 9 |
| 13-West | 107 | 1 | 24 | - | - | 9 | 37 | - | 3 | 8 | 7 | 2 | 16 |

Table5.1.2.c.I.(Cont) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, and anti-c sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Turkmans. Gonbad | 292 | 0.73 | 54.01 | 0.00 | 2.30 | 14.78 | 6.45 | 0.00 | 21.73 | Boue' and Boue'. 1956 |
| 2-Gorgan | 133 | 0.00 | 57.40 | 0.00 | 1.64 | 10.15 | 8.27 | 0.00 | 22.54 | Boue' and Boue'. 1956 |
| 3-Jews | 200 | 0.00 | 60.50 | 0.00 | 0.00 | 10.85 | 6.00 | 0.00 | 22.65 | Gurevitch,J.,et al. 1956 |
| 4-Gilan.Turkish Speakers | 198 | 2.04 | 47.81 | 0.00 | 2.70 | 18.75 | 3.80 | 0.00 | 24.90 | Boue' and Boue'. 1956 |
| 5-North West | 68 | 1.50 | 44.90 | 0.00 | 5.80 | 12.70 | 9.80 | 0.00 | 25.30 | Bajatzadeh,M.,& Walter, H. 1969 |
| 6-Kurds.Sanandaj | 129 | 3.92 | 41.07 | 0.00 | 4.27 | 11.97 | 10.42 | 0.00 | 28.35 | Boue' and Boue'. 1956 |
| 7-Kurds.Mahabad | 125 | 3.39 | 45.20 | 0.00 | 3.06 | 8.04 | 10.21 | 0.00 | 30.10 | Boue' and Boue'. 1956 |
| 8-Moslems. Tehran | 203 | 0.00 | 48.49 | 0.00 | 1.29 | 15.60 | 3.46 | 0.00 | 31.16 | Boue' and Boue'. 1956 |
| 9-Tehran | 113 | 2.70 | 36.90 | 0.00 | 7.70 | 7.20 | 10.50 | 2.20 | 32.70 | Bajatzadeh, M., & Walter, H. 1969 |
| 10-North | 63 | 1.30 | 36.50 | 0.00 | 8.20 | 4.20 | 11.50 | 3.70 | 34.60 | Bajatzadeh, M., & Walter, H. 1969 |
| 11-Central and South | 109 | 3.30 | 45.70 | 0.00 | 2.40 | 4.30 | 8.90 | 0.00 | 35.40 | Bajatzadeh, M., & Walter, H. 1969 |

Table 5.1.2.c.I.(cont.) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, and anti-c sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 12- East | 70 | 2.90 | 47.30 | 0.00 | 1.90 | 4.00 | 7.30 | 0.00 | 36.50 | Bajatzadeh, M.,& Walter, H. 1969 |
| 13-West | 107 | 1.00 | 41.80 | 0.00 | 3.40 | 6.50 | 7.50 | 2.30 | 37.40 | Bajatzadeh, M., & Walter, H. 1969 |

Table 5.1.2.d.I

Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddEE | CCddEe | CCddee | CcDEE | CcDEe | CcDee | CcddEE | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1-Kurdish Jews | 94 | - | 2 | 32 | - | - | - | - | 23 | 18 | - | - | 2 | 8 | 6 | - | - | - | 3 |
| 2-Arabs.Khuzestan | 97 | - | 1 | 24 | - | - | - | 3 | 12 | 33 | - | - | 1 | 1 | 13 | 7 | - | - | 2 |
| 3-Bakhtiaris | 138 | - | 4 | 40 | - | - | 1 | 1 | 30 | 31 | - | 1 | - | 1 | 18 | 4 | - | 1 | 6 |
| 4-Kurdish Jews | 106 | - | 5 | 31 | - | - | - | 3 | 28 | 21 | - | - | 1 | 6 | 5 | - | - | - | 6 |
| 5-Kurds.Baneh,Marivan | 77 | - | - | 21 | - | - | - | - | 25 | 15 | - | - | - | 2 | 8 | - | - | - | 6 |
| 6-Kurds.Sanandaj | 107 | - | - | 39 | - | - | - | - | 15 | 28 | - | - | 1 | 3 | 11 | 3 | - | - | 7 |
| 7-Baluchis.Sistan & Baluchistan | 111 | - | - | 44 | - | - | - | 1 | 18 | 30 | - | - | 1 | 3 | 7 | 1 | - | - | 6 |
| 8-Arabs.Abadan | 158 | - | - | 44 | - | - | - | - | 21 | 59 | - | - | - | 6 | 13 | 6 | 1 | 1 | 7 |
| 9-Lurs.Luristan | 162 | - | - | 40 | - | - | - | 2 | 30 | 46 | - | - | - | 11 | 20 | 3 | - | - | 10 |
| 10-Zabolis.Sistan & Baluchistan | 115 | - | - | 40 | - | - | 1 | 2 | 13 | 32 | - | - | 2 | 8 | 9 | - | - | - | 8 |
| 11-Moslems.Fars | 768 | - | 1 | 196 | - | - | - | 1 | 134 | 262 | - | - | 3 | 21 | 73 | 24 | - | 1 | 52 |
| 12-South east | 348 | - | 4 | 87 | - | - | - | - | 59 | 114 | - | - | 3 | 7 | 30 | 14 | - | 1 | 29 |
| 13-Ghashghais | 66 | - | 1 | 20 | - | - | - | - | 11 | 16 | - | - | 1 | 3 | 8 | - | - | - | 6 |

Table 5.1.2. d. I (Cont)  Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddEE | CCddEe | CCddee | CcDEE | CcDEe | CcDee | CcddEE | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 14-Bassaris. Fars | 101 | - | - | 17 | - | - | - | - | 15 | 46 | - | - | - | 4 | 6 | 6 | - | - | 7 |
| 15-Mamassanis. Fars | 113 | - | 3 | 20 | - | - | - | - | 25 | 29 | - | - | - | 7 | 20 | 1 | - | - | 8 |
| 16-Esfahan | 110 | - | 1 | 27 | - | - | - | 2 | 25 | 36 | - | - | 2 | - | 4 | 1 | - | 1 | 11 |
| 17-Assyrians. Abadan | 32 | - | - | 7 | - | - | - | - | 5 | 9 | - | - | - | - | 7 | 1 | - | - | 3 |
| 18-Tehran | 196 | - | - | 45 | - | - | - | 2 | 37 | 75 | - | - | 2 | 3 | 8 | 2 | - | - | 22 |
| 19-Kurds.Kermanshah | 127 | - | - | 28 | - | - | - | - | 17 | 46 | - | - | - | 1 | 16 | 3 | - | - | 16 |
| 20-Shi'a.Yazd | 151 | - | - | 31 | - | - | - | 1 | 18 | 54 | - | - | 1 | 2 | 18 | 5 | - | - | 21 |
| 21-Armenians.Esfahan | 144 | - | 1 | 23 | - | - | - | 1 | 23 | 45 | - | - | 1 | 2 | 16 | 8 | - | 1 | 23 |
| 22-Armenians | 78 | - | - | 13 | - | - | - | - | 12 | 24 | - | - | - | 3 | 9 | 3 | - | - | 14 |

Table 5.1.2.d I (Cont.) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | | |
| 1-Kurdish Jews | 94 | 1.06 | 53.43 | 0.00 | 4.55 | 23.94 | 0.00 | 0.00 | 17.02 | Godber,Marilyn. J., et al.. | 1973 |
| 2-Arabs.Khuzestan | 97 | 1.04 | 46.28 | 0.00 | 3.59 | 10.15 | 20.58 | 0.00 | 18.36 | Marzban, M. | 1978 |
| 3-Bakhtiaris | 138 | 2.00 | 48.73 | 0.00 | 4.62 | 16.48 | 6.97 | 2.17 | 18.93 | Nijenhuis,L.E. | 1964 |
| 4-Kurdish Jews | 106 | 4.73 | 51.86 | 0.00 | 2.37 | 21.69 | 0.00 | 0.00 | 19.35 | Tills,D., et al. | 1977 |
| 5-Kurds.Baneh,Marivan | 77 | 0.00 | 53.25 | 0.00 | 0.00 | 21.44 | 1.71 | 2.59 | 21.01 | Lehmann,H., et al. | 1973 |
| 6-Kurds.Sanandaj | 107 | 0.93 | 51.07 | 0.00 | 5.00 | 14.02 | 7.20 | 0.00 | 21.78 | Lehmann,H., et al. | 1973 |
| 7-Baluchis.Sistan & Baluchistan | 111 | 0.57 | 59.63 | 0.00 | 1.96 | 14.29 | 1.74 | 0.00 | 21.81 | Present study. | |
| 8-Arabs.Abadan | 158 | 0.00 | 53.18 | 0.00 | 0.00 | 12.13 | 8.91 | 3.37 | 22.41 | Nijenhuis,L.E. | 1964 |
| 9-Lurs.Luristan | 162 | 0.81 | 47.95 | 0.00 | 0.00 | 22.65 | 3.52 | 0.00 | 25.07 | Present study. | |
| 10-Zabolis.Sistan & Baluchistan | 115 | 1.02 | 50.71 | 0.00 | 5.22 | 17.24 | 0.00 | 0.00 | 25.80 | Present study. | |
| 11-Moslems. Fars | 768 | 0.19 | 50.83 | 0.00 | 0.68 | 16.03 | 5.52 | 0.26 | 26.49 | Bowman,J.E.,& Ronaghy, H. | 1967 |

Table 5.1.2.d I (Cont.) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | | |
| 12-South east | 348 | 0.46 | 49.52 | 0.00 | 1.48 | 14.24 | 6.01 | 0.53 | 27.76 | Nijenhuis,L.E. | 1964 |
| 13-Ghashgais.Fars | 66 | 0.99 | 49.30 | 0.00 | 2.47 | 18.71 | 0.00 | 0.00 | 28.26 | Nijenhuis,L.E. | 1964 |
| 14-Bassaris.Fars | 101 | 0.00 | 47.10 | 0.00 | 0.00 | 14.39 | 10.24 | 0.00 | 28.27 | Bowman,J.E. | 1959 |
| 15-Mamassanis. Fars | 113 | 1.88 | 42.39 | 0.00 | 0.00 | 25.56 | 1.72 | 0.00 | 28.54 | Bowman,J.E. | 1959 |
| 16-Esfahan | 110 | 2.56 | 49.99 | 0.00 | 2.44 | 10.99 | 1.32 | 2,35 | 30.33 | Sawhney, K.S. | 1975 |
| 17-Assyrians. Abadan | 32 | 0.00 | 43.80 | 0.00 | 0.00 | 18.76 | 5.01 | 0.00 | 32.43 | Nijenhuis,L.E. | 1964 |
| 18-Tehran | 196 | 0.93 | 50.27 | 0.00 | 1.36 | 13.10 | 1.46 | 0.00 | 32.89 | Sawhney, K.S. | 1975 |
| 19-Kurds.Kermanshah | 127 | 0.00 | 46.88 | 0.00 | 0.00 | 13.79 | 3.24 | 0.00 | 36.09 | Nijenhuis,L.E. | 1964 |
| 20-Shi'a.Yazd | 151 | 0.58 | 43.72 | 0.00 | 0.89 | 13.35 | 4.20 | 0.00 | 37.26 | Sunderland,E.,& Smith, H.M/ | 1966 |
| 21-Armenians.Esfahan | 144 | 1.52 | 38.55 | 0.00 | 0.94 | 13.84 | 6.12 | 0.97 | 38.06 | Bowman,J.E. | 1959 |
| 22-Armenians | 78 | 0.00 | 39.81 | 0.00 | 0.00 | 17.32 | 3.97 | 0.00 | 38.90 | Nijenhuis, L.E. | 1964 |

Table 5.1.2.e.I Rh blood groups distribtuion in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$C^W$ sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | $C^WCDEe$ | CCDee | $C^WCDee$ | $C^WC^WDee$ | CCddee | $C^WCddee$ | $C^WC^Wddee$ | CcDEE | CcDEe | $C^WcDEe$ | CcDee | $C^WcDee$ | CcddEe | $C^WcddEe$ | Ccddee | $C^Wccddee$ | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1-Bakhtiaris | 138 | - | 3 | 1 | 40 | - | - | 1 | - | - | 1 | 30 | - | 31 | - | 1 | - | - | - | 1 | 18 | 4 | - | 1 | 6 |
| 2-South east | 348 | - | 2 | 2 | 87 | - | - | - | - | - | - | 59 | - | 114 | - | - | - | 3 | - | 7 | 30 | 14 | - | 1 | 29 |
| 3-Esfahan | 110 | - | 1 | - | 26 | 1 | - | - | - | - | 2 | 25 | - | 36 | - | - | - | 2 | - | - | 4 | 1 | - | 1 | 11 |
| 4-Tehran | 196 | - | - | - | 42 | 3 | - | - | - | - | 2 | 35 | 2 | 72 | 3 | - | - | 2 | - | 3 | 8 | 2 | - | - | 22 |
| 5-Kurds.Kermanshah | 127 | - | - | - | 27 | 1 | - | - | - | - | - | 17 | - | 46 | - | - | - | - | - | 1 | 16 | 3 | - | - | 16 |

Table 5.1.2.e I (Cont)

Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$C^W$ sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | $C^W$De | Cde | $C^W$de | cDE | cDe | cdE | cde | | |
| 1-Bakhtiaris | 138 | 2.00 | 48.47 | 0.36 | 4.62 | 0.00 | 16.48 | 6.97 | 2.17 | 18.93 | Nijenhuis,L.E. | 1964 |
| 2-South east | 348 | 0.46 | 49.23 | 0.29 | 1.48 | 0.00 | 14.24 | 6.01 | 0.53 | 27.76 | Nijenhuis,L.E. | 1964 |
| 3-Esfahan | 110 | 2.56 | 49.54 | 0.45 | 2.44 | 0.00 | 10.99 | 1.32 | 2.35 | 30.33 | Sawhney,K.S. | 1975 |
| 4-Tehran | 196 | 0.93 | 48.23 | 2.04 | 1.36 | 0.00 | 13.10 | 1.46 | 0.00 | 32.89 | Sawhney,K.S. | 1975 |
| 5-Kurds.Kermanshah | 127 | 0.00 | 46.49 | 0.39 | 0.00 | 0.00 | 13.79 | 3.24 | 0.00 | 36.09 | Nijenhuis,L.E. | 1964 |

able 5.1.2.f I

Rh blood groups distribution in Iran tested with

anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$D^u$ sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCD$^u$ee | CCddee | CcDEE | CcDEe | CcD$^u$Ee | CcDee | CcD$^u$ee | CcddEe | Ccddee | ccDEE | ccD$^u$EE | ccDEe | ccD$^u$Ee | ccDee | ccD$^u$ee | ccddEE | ccddEe | ccddee |
| 1-Kurds.Baneh, Marivan | 77 | - | - | 21 | - | - | - | 25 | - | 15 | - | - | - | 2 | - | 7 | 1 | - | - | - | - | 6 |

Table 5.1.2. f. I ( cont.) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$D^u$ sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | $CD^ue$ | Cde | cDE | $cD^uE$ | cDe | $cD^uc$ | cdE | cde | |
| 1-Kurds.Baneh,Marivan | 77 | 0.00 | 53.25 | 0.00 | 0.00 | 21.44 | 0.00 | 0.00 | 1.71 | 2.59 | 21.01 | Lehmann,H., 1973 et al. |

Table 5.1.2.h.I Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-Ce(f) sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCdee | CcDEE | CE/D/Ce | Ce/D/cE | CcDee | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccDee |
| 1-Kurdish Jews | 94 | - | 2 | 32 | - | - | - | 23 | 18 | - | 2 | 8 | 6 | - | - | - | 3 |

Table 5.1.2.h.I (cont.)

Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-Ce(f) sera

| Population | Number Tested | Gene complex frequencies | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | Cde | cDE | cDe | cdE | cde | |
| 1-Kurdish Jews | 94 | 1.06 | 53.43 | 4.55 | 23.94 | 0.00 | 0.00 | 17.02 | Godber,Marilyn.J., et al. 1973 |

Table 5.1.2.i.I

Rh blood groups distribution in Iran tested with

anti-C, anti-D, anti-E, anti-$^{c}$, anti-$^{e}$, anti-CE, anti-$C^{w}$, and anti-$D^{u}$ sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | $C^{w}$CDEe | CCDee | $c^{w}$CDee | $C^{w}C^{w}$Dee | CCddee | CcDEE | CE/D/Ce | Ce/D/cE | $C^{w}$cDEe | CcDee | $C^{w}$cDee | $CcD^{u}$ee | Ccddee | ccDEE | ccDEe | ccDee | $ccD^{u}$ee | ccddee |
| 1-Kurds. Sanandaj | 107 | - | - | - | 38 | 1 | - | - | - | 2 | 12 | 1 | 26 | - | 2 | 1 | 3 | 11 | 3 | - | 7 |

Table 5.1.2. i.I (Cont.) Rh blood groups distribution in Iran tested with anti-C, anti-D, anti-E, anti-c, anti-e, anti-CE, anti-$C^w$, and anti-$D^u$, sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | $C^w De$ | Cde | cDE | cDe | $cD^u e$ | cde | |
| 1-Kurds.Sanandanj | 107 | 0.93 | 50.14 | 0.93 | 5.00 | 14.02 | 4.45 | 2.75 | 21.78 | Lehmann, H., et al. 1973 |

Table 5.1.2. k I. Rh blood groups distribution in Iran tested with anti-$C^w$ serum

| Population | Number Tested | $C^w$+ | $C^w$- | $C^w$+% | Authors | |
|---|---|---|---|---|---|---|
| 1-Arabs | 158 | 158 | | CDEce | Nijenhuis,L.E. | 1964 |
| 2-Armenians | 78 | 78 | | CDEce | Nijenhuis,L.E. | 1964 |
| 3-Assyrians | 32 | 32 | | CDEce | Nijenhuis,L.E. | 1964 |
| 4-Ghashgais | 66 | 66 | | CDEce | Nijenhuis,L.E. | 1964 |
| 5-Kurds.Baneh,Marivan | 77 | 77 | | CDEce | Lehmann,H.,et al. | 1973 |
| 6-Kurdish Jews | 121 | 121 | | CDEce | Godber,Marylin J.,et al. | 1973 |

Table 5.1.2. 1 I. Rh blood groups distribution in Iran tested with anti-V serum

| Population | Number Tested | V+ | V- | V+% | Authors |
|---|---|---|---|---|---|
| Kurdish Jews | 94 | 94 | CDEcef | | Godber,Maryline,J.,et al. 1973 |

Table 5.1.2. * I. Rh gene complex frequencies distribution in Iran

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Kurdish Jews | 94 | 1.06 | 53.43 | 0.00 | 4.55 | 23.94 | 0.00 | 0.00 | 17.02 | Godber,Marilyn J., et al. 1973 |
| 2-Arabs.Khuzestan | 97 | 1.04 | 46.28 | 0.00 | 3.59 | 10.15 | 20.58 | 0.00 | 18.36 | Marzban, M. 1978 |
| 3-Bakhtiaris | 138 | 2.00 | 48.73 | 0.00 | 4.62 | 16.48 | 6.97 | 2.17 | 18.93 | Nijenhuis,L.E. 1964 |
| 4-Kurdish Jews | 106 | 4.73 | 51.86 | 0.00 | 2.37 | 21.69 | 0.00 | 0.00 | 19.35 | Tills,D., et al. 1977 |
| 5-Kurds.Baneh,Marivan | 77 | 0.00 | 53.25 | 0.00 | 0.00 | 21.44 | 1.71 | 2.59 | 21.01 | Lehmann,H., et al. 1973 |
| 6-Turkmans.Gobad | 292 | 0.73 | 54.01 | 0.00 | 2.30 | 14.78 | 6.45 | 0.00 | 21.73 | Boue' and Boue'. 1956 |
| 7-Kurds.Sanandaj | 107 | 0.93 | 51.07 | 0.00 | 5.00 | 14.02 | 7.20 | 0.00 | 12.78 | Lehmann,H.,et al. 1973 |
| 8-Baluchis.Sistan & Baluchistan | 111 | 0.57 | 59.63 | 0.00 | 1.96 | 14.29 | 1.74 | 0.00 | 21.81 | Present study. |
| 9-Arabs.Abadan | 158 | 0.00 | 53.18 | 0.00 | 0.00 | 12.13 | 8.91 | 3.37 | 22.41 | Nijenhuis,L.E. 1964 |
| 10-Gorgan | 133 | 0.00 | 57.40 | 0.00 | 1.64 | 10.15 | 8.27 | 0.00 | 22.54 | Boue' and Boue'. 1956 |
| 11-Jews | 200 | 0.00 | 60.50 | 0.00 | 0.00 | 10.85 | 6.00 | 0.00 | 22.65 | Gurevitch,J.,et al.1956 |
| 12-Gilan.Turkish Speakers | 198 | 2.04 | 47.81 | 0.00 | 2.70 | 18.75 | 3.80 | 0.00 | 24.90 | Boue' and Boue'. 1956 |
| 13-Lurs.Luristan | 162 | 0.81 | 47.95 | 0.00 | 0.00 | 22.65 | 3.52 | 0.00 | 25.07 | Present study. |

Table 5.1.2.*I(Cont.) Rh gene complex frequencies distribution in Iran

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | | |
| 14-North west | 68 | 1.50 | 44.90 | 0.00 | 5.80 | 12.70 | 9.80 | 0.00 | 25.30 | Bajatzadeh,M.,& Walter,H. | 1969 |
| 15-Zabolis.Sistan & Baluchistan | 115 | 1.02 | 50.71 | 0.00 | 5.22 | 17.24 | 0.00 | 0.00 | 25.80 | Present study. | |
| 16-Moslems.Fars | 768 | 0.19 | 50.83 | 0.00 | 0.68 | 16.03 | 5.52 | 0.26 | 26.49 | Bowman,J.E.,et al. | 1967 |
| 17-South east | 348 | 0.46 | 49.52 | 0.00 | 1.48 | 14.24 | 6.01 | 0.53 | 27.76 | Nijenhuis,L.E. | 1964 |
| 18-Ghashghais | 66 | 0.99 | 49.30 | 0.00 | 2.47 | 18.71 | 0.00 | 0.00 | 28.26 | Nijenhuis,L.E. | 1964 |
| 19-Bassaris.Fars | 101 | 0.00 | 47.10 | 0.00 | 0.00 | 14.39 | 10.24 | 0.00 | 28.27 | Bowman,J.E. | 1959 |
| 20-Kurds.Sanandaj | 129 | 3.92 | 41.07 | 0.00 | 4.27 | 11.97 | 10.42 | 0.00 | 28.35 | Boue' and Boue'. | 1956 |
| 21-Mamassanis.Fars | 113 | 1.88 | 42.39 | 0.00 | 0.00 | 25.56 | 1.72 | 0.00 | 28.54 | Bowman,J.E. | 1959 |
| 22-Kurds.Mahabad | 125 | 3.39 | 45.20 | 0.00 | 3.06 | 8.04 | 10.21 | 0.00 | 30.10 | Boue' and Boue'. | 1956 |
| 23-Esfahan | 110 | 2.56 | 49.99 | 0.00 | 2.44 | 10.99 | 1.32 | 2.35 | 30.33 | Sawhney,K.S. | 1975 |
| 24-Moslems. Tehran | 203 | 0.00 | 48.49 | 0.00 | 1.29 | 15.60 | 3.46 | 0.00 | 31.16 | Boue' and Boue'. | 1956 |
| 25-Assyrians.Abadan | 32 | 0.00 | 43.80 | 0.00 | 0.00 | 18.76 | 5.01 | 0.00 | 32.43 | Nijenhuis,L.E. | 1964 |
| 26-Tehran | 113 | 2.70 | 36.90 | 0.00 | 7.70 | 7.20 | 10.50 | 2.20 | 32.70 | Bajatzadeh,M.,& Walter,H. | 1969 |

Table 5.1.2.* I(Cont) Rh gene complex frequencies distribution in Iran

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | | |
| 27-Tehran | 196 | 0.93 | 50.27 | 0.00 | 1.36 | 13.10 | 1.46 | 0.00 | 32.89 | Sawhney, K.S. | 1975 |
| 28-North | 63 | 1.30 | 36.50 | 0.00 | 8.20 | 4.20 | 11.50 | 3.70 | 34.60 | Bajatzadeh,M., & Walter, H. | 1969 |
| 29-Central and South | 109 | 3.30 | 45.70 | 0.00 | 2.40 | 4.30 | 8.90 | 0.00 | 35.40 | Bajatzadeh,M., & Walter, H. | 1969 |
| 30-Kurds.Kermanshah | 127 | 0.00 | 46.88 | 0.00 | 0.00 | 13.79 | 3.24 | 0.00 | 36.09 | Nijenhuis, L.E. | 1964 |
| 31-East | 70 | 2.90 | 47.30 | 0.00 | 1.90 | 4.00 | 7.30 | 0.00 | 36.50 | Bajatzadeh,M., & Walter, H. | 1969 |
| 32-Shi'a . Yazd | 151 | 0.58 | 43.72 | 0.00 | 0.89 | 13.35 | 4.20 | 0.00 | 37.26 | Sunderland,E., and Smith, H. M. | 1966 |
| 33-West | 107 | 1.00 | 41.80 | 0.00 | 3.40 | 6.50 | 7.50 | 2.30 | 37.40 | Bajatzadeh,M., & Walter,H. | 1969 |
| 34-Armenians.Esfahan | 144 | 1.52 | 38.55 | 0.00 | 0.94 | 13.84 | 6.12 | 0.97 | 38.06 | Bowman, J.E. | 1959 |
| 35-Armenians | 78 | 0.00 | 39.81 | 0.00 | 0.00 | 17.32 | 3.97 | 0.00 | 38.90 | Nijenhuis,L.E. | 1964 |

Table 5.1.2.C. 11 Rh blood groups distribution in the Caucasus tested with anti-C, anti-D, anti-E, and anti-c sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDee | CCddEE | CCddee | CcDEE | CcDee | CcddEE | Ccddee | ccDEE | ccDee | ccddEE | ccddee |
| 1-Georgians abroad. Autocthonous | 40 | - | 9 | - | - | 6 | 13 | - | - | 6 | 1 | - | 5 |
| 2-Armenians autochthonous | 199 | 2 | 41 | - | - | 28 | 62 | - | 1 | 30 | 8 | - | 27 |

Table 5.1.2.C II (Cont)

Rh blood groups distribution in the Caucasus tested with anti-C, anti-D, anti-E, and anti-c sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Georgians abroad. Autocthonous | 40 | 0.00 | 46.30 | 0.00 | 0.00 | 16.36 | 3.25 | 0.00 | 34.09 | Kherumian,R., et al. 1954 |
| 2-Armenians autochthonous | 199 | 0.88 | 42.78 | 0.00 | 0.77 | 15.61 | 4.82 | 0.00 | 35.14 | Kherumian,R., et al. 1954 |

Table 5.1.2.d. II

Rh blood groups distribution in the Caucasus tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddEE | CCddEe | CCddee | CcDEE | CcDEe | CcDee | CcddEE | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1-Svanis.Georgia, Abkhazskaya | 643 | - | 10 | 75 | - | - | - | 6 | 113 | 217 | - | - | 24 | 18 | 92 | 18 | - | 4 | 66 |

Table 5.1.2.d II (Cont.)

Rh blood groups distribution in the Caucasus tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Svanis. Georgia, Abkhazskaya. | 643 | 2.24 | 34.76 | 0.00 | 4.21 | 17.73 | 4.65 | 0.80 | 35.61 | Verbitsky,M. Sh., et al. 1972 |

Table 5.1.2. K II Rh blood groups distribution in the Caucasus tested with anti-$C^w$ serum

| Population | Number Tested | $C^w$+ | $C^w$- | $C^w$+% | | Authors | |
|---|---|---|---|---|---|---|---|
| Svanis.Georgia. Abkhazskaya | 643 | 6 | 637 | 0.93 | CDEce | Verbitsky,M.Sh.,et al. | 1972 |

Table 5.1.2. * II Rh gene complex frequencies distribution in the Caucasus

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Georgians abroad., autochthonous | 40 | 0.00 | 46.30 | 0.00 | 0.00 | 16.36 | 3.25 | 0.00 | 34.09 | Kherumian,R., et al. 1954 |
| 2-Armenians.auto-chthonous | 199 | 0.88 | 42.78 | 0.00 | 0.77 | 15.61 | 4.82 | 0.00 | 35.14 | Kherumian,R., et al. 1954 |
| 3-Svanis.Georgia, Abkhazskaya | 643 | 2.24 | 34.76 | 0.00 | 4.21 | 17.73 | 4.65 | 0.80 | 35.61 | Verbitsky,M. Sh., et al. 1972 |

Table 5.1.2. d * III Rh blood groups distribution in Turkey tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddEE | CCddEe | CCddee | CcDEE | CcDEe | CcDee | CcddEE | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1-Eti-Turks.Asia Minor, near Mersin | 118 | - | - | 33 | - | - | - | - | 17 | 46 | - | - | - | 4 | 6 | 1 | - | - | 11 |
| 2-Turks.Asia Minor, Mersin | 108 | - | - | 25 | - | - | - | - | 21 | 35 | - | - | 1 | 3 | 10 | 1 | - | - | 12 |

Table 5.1.2. d.* III (Cont.) Rh blood groups distribution in Turkey tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | | |
| 1-Eti-Turks.Asia Minor, near Mersin | 118 | 0.00 | 54.66 | 0.00 | 0.00 | 13.14 | 1.35 | 0.00 | 30.85 | Aksoy,M., et al. | 1958 |
| 2-Turks.Asia Minor, Mersin | 108 | 0.00 | 48.18 | 0.00 | 1.38 | 17.14 | 1.30 | 0.00 | 32.00 | Aksoy,M., et al. | 1958 |

Table 5.1.2. f. III

Rh blood groups distribution in Turkey tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$D^u$ sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | $CCD^{u}ee$ | CCddee | CcDEE | CcDEe | $CcD^{u}Ee$ | CcDee | $CcD^{u}ee$ | CcddEe | Ccddee | ccDEE | $ccD^{u}EE$ | ccDEe | $ccD^{u}Ee$ | ccDee | $ccD^{u}ee$ | ccddEE | ccddEe | ccddee |
| 1-Eti-Turks.Asia Minor , near Mersin | 118 | - | - | 33 | - | - | - | 17 | - | 42 | 4 | - | - | 4 | - | 6 | - | 1 | - | - | - | 11 |

Table 5.1.2. f III (Cont.)

Rh blood groups distribution in Turkey tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$D^u$ sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | $C^uDe$ | Cde | cDE | $c^uDE$ | cDe | $c^uDe$ | cdE | cde | |
| 1-Eti-Turks.Asia Minor, near Mersin | 118 | 0.00 | 50.20 | 4.46 | 0.00 | 13.14 | 0.00 | 1.35 | 0.00 | 0.00 | 30.85 | Aksoy,M.,et al. 1958 |

Table 5.1.2. K III Rh blood groups distribution in Turkey tested with anti-$C^W$ serum

| Population | Number Tested | $C^W$+ | $C^W$- | $C^W$+% | Authors |
|---|---|---|---|---|---|
| Eti-Turks.Asia Minor,near Mersin | 118 | 118 | CDEceD$^u$ | | Aksoy,M., et al. 1968 |

Table 5.1.2.b. IV

Rh blood groups distribution in Iraq tested with anti-C, anti-D, and anti-E sera

| Population | Number Tested | Phenotypes | | | | | | | | Gene complex frequencies | | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDee | CCdEE | CCddee | ccDEE | ccDee | ccddEE | ccddee | CDe | Cde | cDE | cDe | cdE | cde | | |
| 1-Kurdish Jews | 250 | 54 | 103 | - | 2 | 48 | 26 | 4 | 13 | 37.42 | 1.59 | 20.15 | 15.61 | 3.55 | 21.68 | Gurevitch,J., et al. | 1955 |

Table 5.1.2. c IV

Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, and anti-c sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDee | CCddEE | CCddee | CcDEE | CcDee | CcddEE | Ccddee | ccDEE | ccDee | ccddEE | ccddee |
| 1-Kurdish Jews | 129 | 14 | 38 | - | - | 34 | 22 | - | - | 12 | 4 | - | 5 |
| 2-Baghdad.Jews | 162 | 9 | 55 | - | - | 34 | 28 | - | 1 | 18 | 5 | 1 | 11 |
| 3-Baghdad.Arabs | 300 | - | 72 | - | - | 47 | 79 | - | 3 | 41 | 25 | 2 | 31 |

Table 5.1.2.c IV (Cont.) Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, and anti-c sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | | |
| 1-Kurdish Jews | 129 | 9.82 | 52.24 | 0.00 | 0.00 | 17.01 | 5.32 | 0.00 | 15.61 | Gurevitch,J., et al. | 1956 |
| 2-Baghdad.Jews | 162 | 4.77 | 52.37 | 0.00 | 1.83 | 15.46 | 4.11 | 1.29 | 20.17 | Gurevitch,J., et al. | 1956 |
| 3-Baghdad.Arabs | 300 | 0.00 | 43.56 | 0.00 | 1.95 | 15.13 | 9.73 | 1.13 | 28.50 | Kayssi,A.I. | 1940 |

Table 5.1.2.d. IV Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddEE | CCddEe | CCddee | CcDEE | CcDEe | CcDee | CcddEE | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1-Kurdish Jews. South east | 50 | - | - | 24 | - | - | - | 7 | 12 | 5 | - | - | - | 1 | - | - | - | - | 1 |
| 2-Jews | 1146 | 1 | 4 | 338 | - | - | 1 | 5 | 208 | 340 | - | - | 8 | 29 | 115 | 23 | - | 2 | 72 |
| 3-Kurdish Jews. North west | 61 | - | - | 13 | - | - | - | 4 | 15 | 19 | - | - | 1 | 1 | 4 | - | - | - | 4 |
| 4-Assyrians | 99 | - | - | 18 | - | - | - | - | 12 | 39 | - | - | - | - | 11 | 9 | - | - | 10 |
| 5-Kurdish Jews | 27 | - | - | 7 | - | - | - | - | 3 | 9 | - | - | - | 1 | 4 | - | - | - | 3 |
| 6-Karaite Jews | 72 | - | - | 4 | - | - | - | - | 1 | 24 | - | - | - | - | 4 | 19 | - | - | 20 |

Table 5.1.2.d. IV (Cont.) Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Kurdish Jews.South east | 50 | 7.49 | 64.51 | 0.00 | 0.00 | 20.51 | 0.00 | 0.00 | 7.49 | Tills,D.,et al, 1977 |
| 2-Jews | 1146 | 0.67 | 52.20 | 0.00 | 1.63 | 16.43 | 3.74 | 0.31 | 25.02 | Levence,C. 1968 |
| 3-Kurdish Jews.North west | 61 | 4.86 | 46.17 | 0.00 | 2.24 | 18.91 | 0.00 | 0.00 | 27.82 | Tills,D.,et al. 1977 |
| 4-Assyrians | 99 | 0.00 | 43.99 | 0.00 | 0.00 | 11.63 | 12.92 | 0.00 | 31.46 | Ikin,Elizabeth W., et al. 1965 |
| 5-Kurdish Jews | 27 | 0.00 | 48.15 | 0.00 | 0.00 | 16.67 | 0.00 | 0.00 | 35.18 | Godber,Marilyn. J., et al.. 1973 |
| 6-Karaite Jews | 72 | 0.00 | 22.93 | 0.00 | 0.00 | 3.47 | 20.90 | 0.00 | 52.70 | Goldschmidt,E., et al. 1976 |

Table 5.1.2.f. IV

Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$D^u$ sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | $CCD^ue$ | CCddee | CcDEE | CcDEe | $CcD^uEe$ | CcDee | $CcD^uee$ | CcddEe | Ccddee | ccDEE | $ccD^uEE$ | ccDEe | $ccD^uEe$ | ccDee | $ccD^uee$ | ccddEE | ccddEe | ccddee |
| 1-Jews | 1146 | 1 | 4 | 338 | - | 1 | 5 | 208 | - | 339 | 1 | - | 8 | 29 | - | 115 | - | 23 | - | - | 2 | 72 |

Table 5.1.2.f IV (Cont.) Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$D^u$ sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | $CD^ue$ | Cde | cDE | $cD^uE$ | cDe | $cD^ue$ | cdE | cde | |
| 1-Jews | 1146 | 0.67 | 52.04 | 0.16 | 1.63 | 16.43 | 0.00 | 3.74 | 0.00 | 0.31 | 25.02 | Levence,C. 1968 |

Table 5.1.2.g. IV

Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-V sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddee | CcDEE | CcDEeV | CcDEe | CcDeeV | CcDee | CcddeeV | Ccddee | ccDEE | ccDEeV | ccDEe | ccDeeV | ccDee | ccddeeV | ccddee |
| 1- Assyrians | 99 | - | - | 18 | - | - | - | 12 | 2 | 37 | - | - | - | - | 11 | - | 9 | - | 10 |

Table 5.1.2.g. IV (Cont.)

Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-V sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | Cde | cDE | cDeV | cDe | cdeV | cde | |
| 1-Assyrians | 99 | 0.00 | 43.99 | 0.00 | 11.63 | 1.01 | 11.91 | 0.00 | 31.46 | Ikin,Elizabeth W., et al. 1965 |

Table 5.1.2. h. IV

Rh blood groups distribution in Iraq tested with

anti-C, anti-D, anti-E, anti-c, anti-e, and anti-Ce(f)sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddee | CcDEE | CE/D/Ce | Ce/D/cE | CcDee | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1- Kurdish Jews | 27 | - | - | 7 | - | - | - | 3 | 9 | - | - | 1 | 4 | - | - | - | 3 |

Table 5.1.2.h. IV (Cont.) Rh blood groups distribution in Iraq tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-Ce (f) sera

| Population | Number Tested | Gene complex frequencies | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | Cde | cDE | cDe | cdE | cde | |
| 1-Kurdish Jews | 27 | 0.00 | 48.15 | 0.00 | 16.67 | 0.00 | 0.00 | 35.18 | Godber,Marilyn.J., 1973 et al. |

Table 5.1.2. K IV. Rh blood groups distribution in Iraq tested with anti-$C^W$ serum

| Population | Number Tested | $C^W$+ | $C^W$- | $C^W$+% | Authors |
|---|---|---|---|---|---|
| Assyrians | 99 | 99 | | CDEceV | Ikin,Elizabeth W.,et al. 1965 |

Table 5.1.2. 1 IV Rh blood groups distribution in Iraq tested with anti-V serum

| Population | Number Tested | V+ | V- | V+% | Authors |
|---|---|---|---|---|---|
| Kurdish Jews | 27 | 27 | CDEcef | | Godber,Marylin,J.,et al. 1973 |

Table 5.1.2. * IV Rh gene complex frequencies distribution in Iraq

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Kurdish Jews, South east | 50 | 7.49 | 64.51 | 0.00 | 0.00 | 20.51 | 0.00 | 0.00 | 7.49 | Tills,D., et al. 1977 |
| 2-Kurdish Jews | 129 | 9.82 | 52.24 | 0.00 | 0.00 | 17.01 | 5.32 | 0.00 | 15.61 | Gurevitch,J., et al. 1956 |
| 3-Baghdad.Jews | 162 | 4.77 | 52.37 | 0.00 | 1.83 | 15.46 | 4.11 | 1.29 | 20.17 | Gurevitch,J., et al. 1956 |
| 4-Jews | 1146 | 0.67 | 52.20 | 0.00 | 1.63 | 16.43 | 3.74 | 0.31 | 25.02 | Levence, C. 1968 |
| 5-Kurdish Jews.North west | 61 | 4.86 | 46.17 | 0.00 | 2.24 | 18.91 | 0.00 | 0.00 | 27.82 | Tills,D., et al. 1977 |
| 6-Baghdad.Arabs | 300 | 0.00 | 43.56 | 0.00 | 1.95 | 15.13 | 9.73 | 1.13 | 28.50 | Kayssi,A.I. 1940 |
| 7-Assyrians | 99 | 0.00 | 43.99 | 0.00 | 0.00 | 11.63 | 12.92 | 0.00 | 31.46 | Ikin,Elizabeth W.,1965 et al. |
| 8-Kurdish Jews | 27 | 0.00 | 48.15 | 0.00 | 0.00 | 16.67 | 0.00 | 0.00 | 35.18 | Godber,Marilyn.J.,1973 et al. |
| 9-Karaite Jews | 72 | 0.00 | 22.93 | 0.00 | 0.00 | 3.47 | 20.90 | 0.00 | 52.70 | Goldschmidt,E., 1976 et al. |

Table 5.1.2.d. * V

Rh blood groups distribution in Kuwait tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddEE | CCddEe | CCddee | CcDEE | CcDEe | CcDee | CcddEE | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1-Suluba tribe | 52 | - | - | 13 | - | - | - | - | 16 | 9 | - | - | - | 7 | 4 | 1 | - | - | 2 |
| 2-Kuwaitis | 200 | 1 | 4 | 44 | - | - | 1 | 5 | 24 | 55 | - | 1 | 3 | 6 | 30 | 15 | 1 | - | 10 |
| 3-General population | 74 | - | - | 16 | - | - | - | - | 8 | 24 | - | - | 2 | 1 | 11 | 5 | - | - | 7 |
| 4-Kuwaiti Arabs | 110 | - | 1 | 27 | - | - | - | 1 | 14 | 42 | - | - | - | - | 3 | 8 | - | 1 | 13 |
| 5-Ajman tribe | 52 | - | - | 4 | - | - | - | - | 9 | 14 | - | - | - | 4 | 9 | 4 | - | - | 8 |

Table 5.1.2.d.*V( Cont.)

Rh blood groups distribution in Kuwait tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Suluba tribe | 52 | 0.00 | 49.00 | 0.00 | 0.00 | 33.00 | 3.00 | 0.00 | 15.00 | Khaled,E., et al. 1981 |
| 2-Kuwaitis | 200 | 3.53 | 38.12 | 0.00 | 5.37 | 15.39 | 14.42 | 2.25 | 20.92 | Onsi,A., et al,. 1969 |
| 3-General population | 74 | 0.00 | 41.00 | 0.00 | 4.00 | 14.00 | 10.00 | 0.00 | 32.00 | Khaled,E.,et al. 1981 |
| 4-Kuwaiti Arabs | 110 | 2.17 | 49.20 | 0.00 | 0.00 | 5.04 | 8.80 | 2.34 | 32.46 | Sawhney,K.S. 1975 |
| 5-Ajman | 52 | 0.00 | 30.00 | 0.00 | 0.00 | 25.00 | 8.00 | 0.00 | 37.00 | Khaled,E., et al.. 1981 |

Table 5.1.2.d. VI

Rh blood groups distribution in Saudi Arabia tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddEE | CCddEe | CCddee | CcDEE | CcDEe | CcDee | CcddEE | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1-Saudi Arabians | 1167 | - | 2 | 225 | - | - | - | - | 137 | 413 | - | - | 7 | 32 | 129 | 148 | - | - | 74 |
| 2-Sunnis.Qatif and Hasa Oases | 323 | - | - | 58 | - | - | - | - | 30 | 110 | - | - | 3 | 15 | 48 | 39 | - | - | 20 |
| 3-Western Saudi Arabia | 178 | - | - | 39 | - | - | - | 1 | 8 | 53 | - | - | - | 14 | 6 | 40 | - | 2 | 15 |
| 4-Sunnis.Asir,Hejaz, Najran | 176 | - | 2 | 37 | - | - | - | - | 17 | 60 | - | - | 1 | 5 | 16 | 21 | - | 1 | 16 |

Table 5.1.2.d. VI(Cont.) Rh blood groups distribution in Saudi Arabia tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | | |
| 1-Saudi Arabians | 1167 | 0.13 | 42.06 | 0.00 | 1.13 | 14.09 | 17.57 | 0.00 | 25.02 | Maranjian,G., et al. | 1966 |
| 2-Sunnis.Qatif and Hasa Oases | 323 | 0.00 | 38.46 | 0.00 | 1.63 | 16.72 | 17.93 | 0.00 | 25.26 | Maranjian,G., et al. | 1966 |
| 3-Western Saudi Arabia | 178 | 0.00 | 38.97 | 0.36 | 0.00 | 9.96 | 22.61 | 2.61 | 25.50 | Saha,N., et al. | 1980 |
| 4-Sunnis.Asir,Hejaz,Najran | 176 | 0.90 | 42.46 | 0.00 | 0.96 | 11.23 | 14.77 | 0.94 | 28.74 | Maranjian,G., et al. | 1966 |

Table 5.1.2.f VI

Rh blood groups distribution in Saudi Arabia tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$D^u$ sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | $CCD^ue$ | CCddee | CcDEE | CcDEe | $CcD^uEe$ | CcDee | $CcD^uee$ | CcddEe | Ccddee | ccDEE | $ccD^uEE$ | ccDEe | $ccD^uEe$ | ccDee | $ccD^uee$ | ccddEE | ccddEe | ccddee |
| 1-Sunnis.Qatif and Hasa Oases | 323 | - | - | 58 | - | - | - | 30 | - | 110 | - | - | 3 | 15 | - | 48 | - | 37 | 2 | - | - | 20 |
| 2-Sunnis.Asir,Hejaz , Najran | 176 | - | 2 | 37 | - | - | - | 17 | - | 60 | - | - | 1 | 5 | - | 16 | - | 19 | 2 | - | 1 | 16 |

Table 5.1.2. f VI (Cont.) Rh blood groups distribution in Saudi Arabia tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$D^u$ sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | $CD^ue$ | Cde | cDE | $cD^uE$ | cDe | $cD^ue$ | cdE | cde | |
| 1-Sunnis.Qatif and Hasa Oases | 323 | 0.00 | 38.46 | 0.00 | 1.63 | 16.72 | 0.00 | 16.78 | 1.15 | 0.00 | 25.62 | Maranjian, G., et al. 1966 |
| 2-Sunnis.Asir,Hejaz, Najran | 176 | 0.90 | 42.46 | 0.00 | 0.96 | 11.23 | 0.00 | 13.13 | 1.64 | 0.94 | 28.74 | Maranjian, G., et al. 1966 |

Table 5.1.2.j. VI

Rh blood groups distribution in Saudi Arabia tested with anti-C+$C^w$, anti-D,anti-E,anti-c,anti-e,anti-$C^w$,anti-V,and anti-$D^u$ sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | $CC^wDEe$ | CCDee | $CC^wDee$ | CCddee | CcDEE | CcDEev | CcDEe | $C^wcDEe$ | CcDeev | CcDee | $C^wcDeev$ | $C^wcDee$ | $CcD^ueev$ | $CcD^uee$ | CcddeeV | Ccddee | ccDEE | ccDEev | ccDEe | ccDeev | ccDee | $ccD^ueev$ | $ccD^uee$ | ccddeev | ccddee |
| 1-Saudi Arabians | 1167 | - | 2 | - | 224 | 1 | - | - | - | 136 | 1 | 41 | 368 | 1 | 3 | - | - | - | 7 | 32 | 17 | 112 | 56 | 85 | 2 | 5 | 1 | 73 |

Table 5.1.2.j. VI (cont.) Rh blood groups distribution in Saudi Arabia tested with anti-C+$C^w$, anti-D, anti-E, antic, anti-e, anti-$c^w$, anti-V, and anti-$D^u$ sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | $C^wDe$ | Cde | cDE | cDeV | cDe | $cD^uev$ | $cD^ue$ | cdev | cde | |
| 1-Saudi Arabians | 1167 | 0.13 | 41.80 | 0.26 | 1.13 | 14.09 | 4.81 | 11.67 | 0.27 | 0.82 | 0.13 | 24.89 | Maranjian, 1966 G., et al. |

Table 5.1.2. K VI Rh blood groups distribution in Saudi Arabia tested with anti-$C^w$ serum

| Population | Number Tested | $C^w$+ | $C^w$- | $C^w$+ % | | Authors | |
|---|---|---|---|---|---|---|---|
| Sunnis.Asir,Hejaz, Najran | 176 | 176 | | | CDEceD$^u$ | Maranjian,G.,et al. | 1966 |
| Sunnis,Qatif,Hasa Oases | 323 | 323 | | | CDEceD$^u$ | Maranjian,G.,et al. | 1966 |

Table 5.1.2.* VI Rh gene complex frequencies distribution in Saudi Arabia

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Saudi Arabians | 1167 | 0.13 | 42.06 | 0.00 | 1.13 | 14.09 | 17.57 | 0.00 | 25.02 | Maranjian,G., et al. 1966 |
| 2-Sunnis.Qatif & Hasa Oases | 323 | 0.00 | 38.46 | 0.00 | 1.63 | 16.72 | 17.93 | 0.00 | 25.26 | Maranjian,G., et al. 1966 |
| 3-Western Saudi Arabia | 178 | 0.00 | 38.97 | 0.36 | 0.00 | 9.96 | 22.61 | 2.61 | 25.50 | Saha,N., et al. 1980 |
| 4-Sunnis.Asir,Hejaz, Najran | 176 | 0.90 | 42.46 | 0.00 | 0.96 | 11.23 | 14.77 | 0.94 | 28.74 | Maranjian,G., et al. 1966 |

Table 5.1.2.c. VIII

Rh blood groups dsitribution in Pakistan tested with anti-C, anti-D, anti-E, and anti-c sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDee | CCddEE | CCddee | CcDEE | CcDee | CcddEE | Ccddee | ccDEE | ccDee | ccddEE | ccddee |
| 1-Bengalis.Dacca | 236 | 6 | 118 | - | 2 | 24 | 58 | - | 3 | 12 | 4 | - | 9 |
| 2-Punjabis.Lahore | 203 | 5 | 75 | - | - | 24 | 64 | - | 2 | 12 | 9 | - | 12 |
| 3-Moslems.Karachi | 150 | - | 62 | - | - | 20 | 48 | - | 1 | 8 | 1 | - | 10 |
| 4-Peshawar | 155 | 4 | 62 | - | - | 16 | 38 | - | 2 | 12 | 6 | 1 | 14 |
| 5-Parsis.Karachi | 103 | - | 44 | - | - | 12 | 31 | - | - | 4 | 1 | - | 11 |

Table 5.1.2.c. VIII (Cont.) Rh blood groups distribution in Pakistan tested with anti-C, anti-D, anti-E, and anti-c sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Bengalis.Dacca | 236 | 1.71 | 63.08 | 0.00 | 6.62 | 7.71 | 3.80 | 0.00 | 17.08 | Boyd,W.C., et al. 1954 |
| 2-Punjabis.Lahore | 203 | 2.04 | 57.39 | 0.00 | 2.17 | 8.64 | 7.21 | 0.00 | 22.55 | Boyd,W.C., et al. 1954 |
| 3-Moslems.Karachi | 150 | 0.00 | 63.03 | 0.00 | 1.31 | 9.76 | 1.20 | 0.00 | 24.70 | Moten,A.N., et al. 1956 |
| 4-Peshawar | 155 | 1.94 | 55.82 | 0.00 | 2.91 | 8.53 | 4.82 | 0.92 | 25.06 | Boyd,W.C., et al. 1954 |
| 5-Parsis.Karachi | 103 | 0.00 | 63.61 | 0.00 | 0.00 | 8.02 | 1.20 | 0.00 | 27.17 | Moten,A.N., et al. 1956 |

Table 5.1.2. d VIII Rh blood groups distribution in Pakistan tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddEE | CCddEe | CCddee | CcDEE | CcDEe | CcDee | CcddEE | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1-Baltis.Baltistan | 80 | - | - | 25 | - | - | - | - | 22 | 16 | - | - | - | 2 | 8 | 1 | - | - | 6 |
| 2-Moslems.Punjab & West | 101 | - | - | 46 | - | - | - | 1 | 10 | 33 | - | - | 1 | - | 5 | - | - | - | 5 |
| 3-Pathans.Swat.Saidu Sharif | 133 | - | 1 | 42 | - | - | - | - | 18 | 44 | - | - | - | 2 | 8 | 6 | - | - | 12 |
| 4-Hunza.Gilgit | 23 | - | - | 7 | - | - | - | - | 3 | 9 | - | - | - | 1 | 2 | - | - | - | 1 |

Table 5.1.2.d. VIII (Cont.) Rh blood groups distribution in Pakistan tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Baltis.Baltistan | 80 | 0.00 | 55.02 | 0.00 | 0.00 | 21.39 | 1.74 | 0.00 | 21.85 | Clegg,E.J., 1961 et al. |
| 2-Moslems.Punjab & West | 101 | 0.63 | 66.69 | 0.00 | 0.49 | 7.78 | 0.05 | 0.00 | 24.36 | Chaudhri,I. 1952 M.,et al. |
| 3-Pathans.Swat.Saidu Sharif | 133 | 0.59 | 55.08 | 0.00 | 0.00 | 11.08 | 6.09 | 0.00 | 27.16 | Alciati,G. 1968 |
| 4-Hunza.Gilgit | 23 | 0.00 | 56.52 | 0.00 | 0.00 | 15.22 | 0.00 | 0.00 | 28.26 | Ikin,Eliza- 1959 beth W.,et al. |

Table 5.1.2. e. VIII

Rh blood groups distribution in Pakistan tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$C^w$ sera

| Population | Number Tested | Phenotypes | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | $C^wCDEe$ | CCDee | $C^wCDee$ | $C^wC^wDee$ | CCddee | $C^wCddee$ | $C^wC^wddee$ | CcDEE | CcDEe | $C^wcDEe$ | CcDee | $C^wcDee$ | CcddEe | $C^wcddEe$ | Ccddee | $C^wcddee$ | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1-Moslems.Punjab & West | 101 | - | - | - | 46 | - | - | - | - | - | 1 | 10 | - | 33 | - | - | - | - | 1 | - | 5 | - | - | - | 5 |

Table 5.1.2.e. VIII (Cont.)

Rh blood groups distribution in Pakistan tested with anti-C, anti-D, anti-E, anti-c, anti-e, and anti-$C^w$ sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | $C^wDe$ | Cde | $C^wde$ | CDE | cDe | cdE | cde | |
| 1-Moslems.Punjab & West | 101 | 0.63 | 66.69 | 0.00 | 0.00 | 0.49 | 7.78 | 0.05 | 0.00 | 24.36 | Chaudhri,I.M., 1952 et al. |

Table 5.1.2. 1 VIII Rh blood groups distribution in Pakistan tested with anti-V serum

| Population | Number Tested | V+ | V- | V+% | Authors | |
|---|---|---|---|---|---|---|
| 1-Pathans | 139 | 139 | | | Vos,G.H., and Kirk,R.L. | 1961 |
| 2-Punjabis | 168 | 168 | | | Vos,G.H., and Kirk,R.L. | 1961 |

Table 5.1.2. * VIII Rh gene complex frequencies distribution in Pakistan

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Bengalis.Dacca | 236 | 1.71 | 63.08 | 0.00 | 6.62 | 7.71 | 3.80 | 0.00 | 17.08 | Boyd,W.C., et al. 1954 |
| 2-Baltis.Baltistan | 80 | 0.00 | 55.02 | 0.00 | 0.00 | 21.39 | 1.74 | 0.00 | 21.85 | Clegg,E., et al. 1961 |
| 3-Punjabis.Lahore | 203 | 2.04 | 57.39 | 0.00 | 2.17 | 8.64 | 7.21 | 0.00 | 22.55 | Boyd,W.C.,et al. 1954 |
| 4-Moslems.Punjab & West | 101 | 0.63 | 66.69 | 0.00 | 0.49 | 7.78 | 0.05 | 0.00 | 24.36 | Chaudhri,I.M., et al. 1952 |
| 5-Moslems.Karachi | 150 | 0.00 | 63.03 | 0.00 | 1.31 | 9.76 | 1.20 | 0.00 | 24.70 | Moten,A.N.,et al.1956 |
| 6-Peshawar | 155 | 1.94 | 55.82 | 0.00 | 2.91 | 8.53 | 4.82 | 0.92 | 25.06 | Boyd,W.C.,et al. 1954 |
| 7-Pathans.Swat. Saidu Sharif | 133 | 0.59 | 55.08 | 0.00 | 0.00 | 11.08 | 6.09 | 0.00 | 27.16 | Alciati,G. 1968 |
| 8-Parsis.Karachi | 103 | 0.00 | 63.61 | 0.00 | 0.00 | 8.02 | 1.20 | 0.00 | 27.17 | Moten,A.N.,et al.1956 |
| 9-Hunza.Gilgit | 23 | 0.00 | 56.52 | 0.00 | 0.00 | 15.22 | 0.00 | 0.00 | 28.26 | Ikin,Elizabeth W.,et al. 1959 |

Table 5.1.2.d*IX Rh blood groups distribution in Afghanistan tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Populations | Number tested | Phenotypes | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CCDEE | CCDEe | CCDee | CCddEE | CCddEe | CCddee | CcDEE | CcDEe | CcDee | CcddEE | CcddEe | Ccddee | ccDEE | ccDEe | ccDee | ccddEE | ccddEe | ccddee |
| 1-Timuri and related tribes | 118 | - | - | 38 | - | - | - | - | 43 | 24 | - | - | - | 6 | 2 | 1 | - | - | 4 |
| 2- Pushtus | 104 | - | - | 27 | - | - | - | - | 16 | 26 | - | - | - | 4 | 17 | 5 | - | - | 9 |
| 3- Daris | 179 | - | 1 | 60 | - | - | 1 | - | 24 | 43 | - | - | 1 | 3 | 20 | 6 | - | - | 20 |

Table 5.1.2.d*IX (Cont). Rh blood groups distribution in Afghanistan tested with anti-C, anti-D, anti-E, anti-c, and anti-e sera

| Population | Number Tested | Gene complex frequencies | | | | | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | CDE | CDe | CdE | Cde | cDE | cDe | cdE | cde | |
| 1-Timuri and related tribes | 118 | 0.00 | 60.61 | 0.00 | 0.00 | 24.15 | 1.61 | 0.00 | 13.63 | Woodd-Walker, 1967 R.B., et al. |
| 2-Pushtus | 104 | 0.00 | 46.10 | 0.00 | 0.00 | 19.70 | 6.80 | 0.00 | 27.40 | Papiha,S.S., 1977 et al. |
| 3-Daris | 179 | 0.40 | 52.40 | 0.00 | 0.80 | 13.80 | 4.00 | 0.00 | 28.60 | Papiha,S.S., 1977 et al. |

Table 5.1.2. K IX Rh blood groups distribution in Afghanistan tested with anti-$C^W$ serum

| Population | Number Tested | $C^W$+ | $C^W$- | $C^W$+% | | Authors |
|---|---|---|---|---|---|---|
| Timuri and related tribes | 118 | | 118 | | CDEce | Woodd-Walker,R.B.,et al. 1967 |

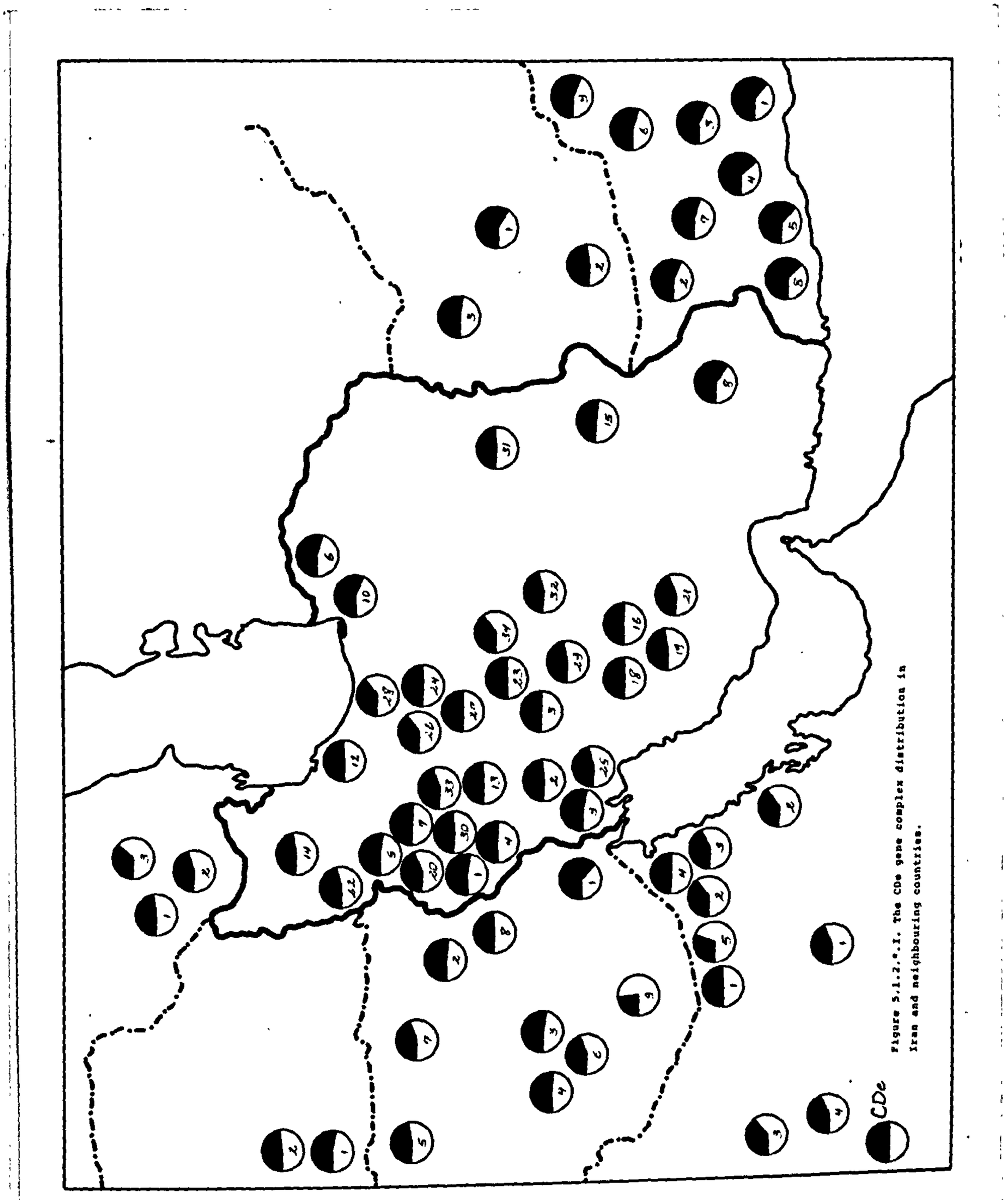



Figure 5.1.2.e.1. The CDe gene complex distribution in Iran and neighbouring countries.

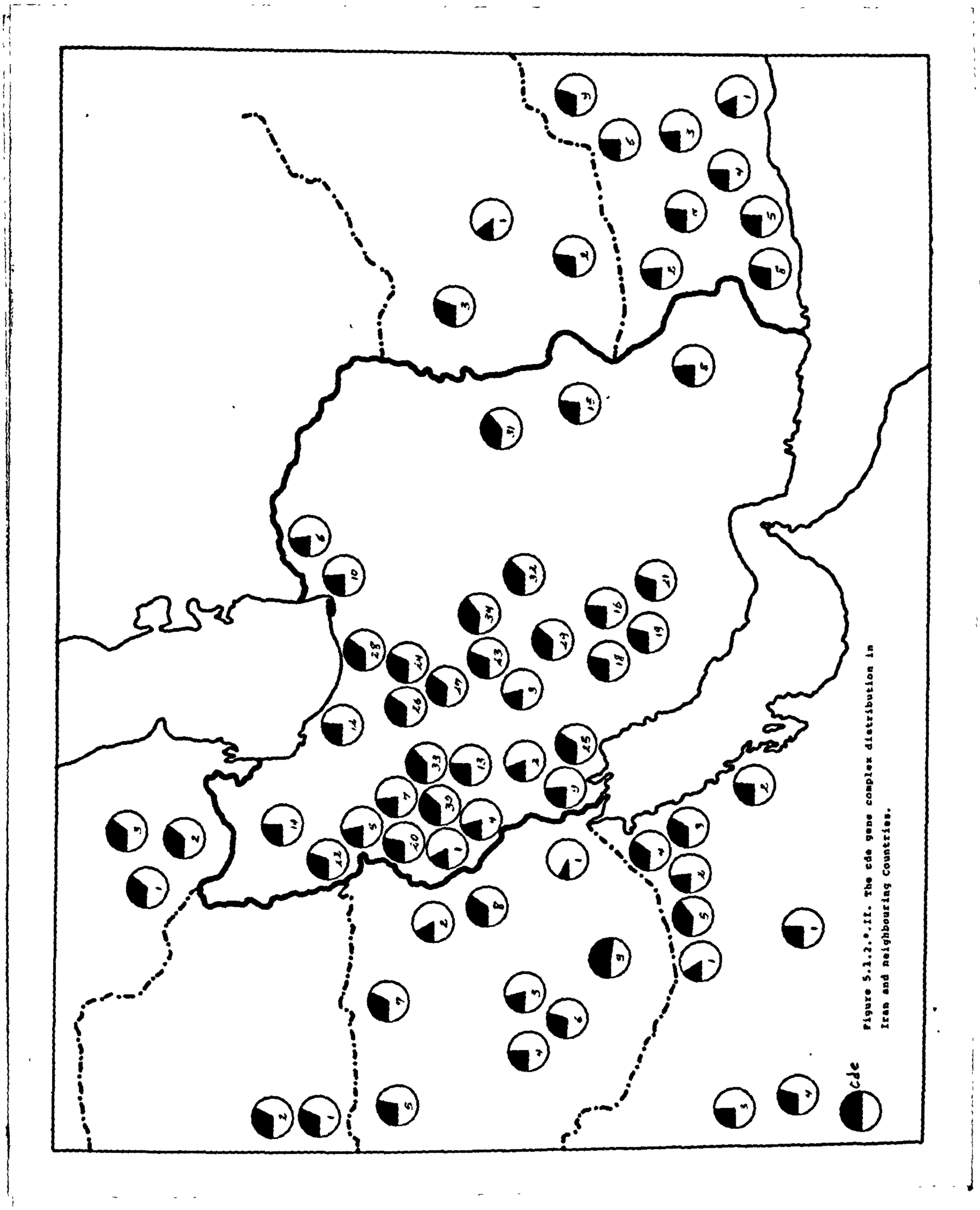


Figure 5.1.2.*.II. The cde gene complex distribution in Iran and neighbouring Countries.

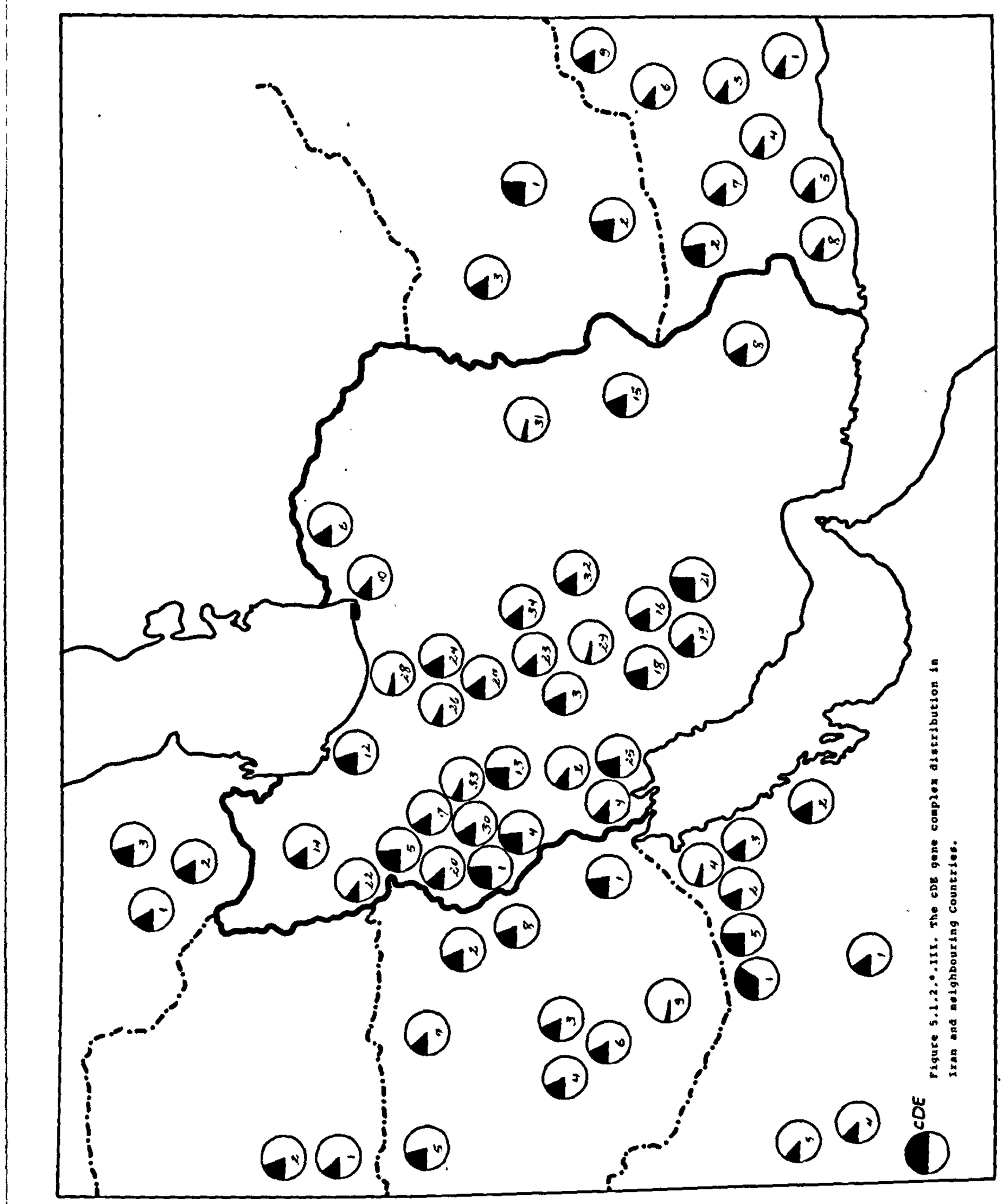



Figure 5.1.2.*.III. The cDE gene complex distribution in Iran and neighbouring Countries.

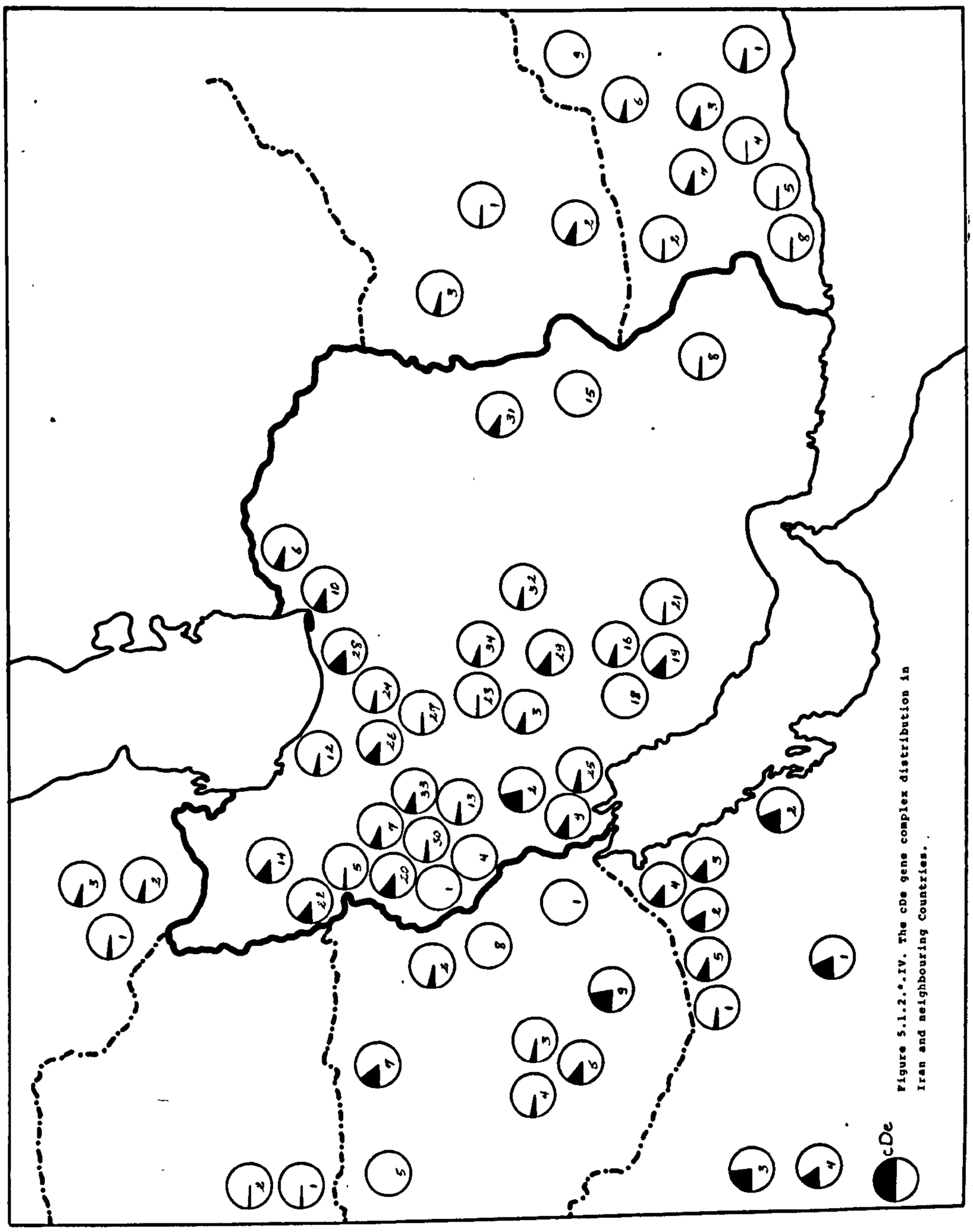



Figure 5.1.2.4.IV. The cDe gene complex distribution in Iran and neighbouring Countries.

## 5.1.3. The MNSs blood group system

The distribution of MN blood groups and respective gene frequencies in Iranian and neighbouring populations, expressed in term of three phenotypes after testing with two anti-sera, is shown in Tables 5.1.3.a.

The frequency of the M gene ranges from 49.59 to 75.23 percent in Iranians, being lowest in the Kurds of Rezaieh and highest in the Baluchis of Sistan and Baluchistan both obtained in the present investigation.

On the whole, with an average M gene frequency of 62.54 percent, the Iranian population appears to exhibit a much higher M frequency than that of around 55 percent found in most European populations(Mourant et al, 1976).

From the table, some differences in the distribution of the M gene frequencies seem to exist in Iran, as the M gene with an average frequency of 59.33 percent in western Iran appears to increases eastwards with an average frequency of 66.80 percent in eastern Iran.

The Kurds both in western Iran and in Iraq show an M gene frequency averaging 61.41 and 64.20 percent respectively which are near the average for Iran and for Iraq. It is, therefore,interesting that the Kurdish Jews both in Iran and in Iraq exhibit much lower M gene frequencies, averaging only 56.75 and 48.76 percent respectively,figures near those for Europeans. The non-Kurdis Jews of Iran and Iraq have an average M gene frequency of 62.27 and 55.24 percent respectively which are, on the whole,also rather low in relation to the regional level.

The Arabs of south western Iran, with an M gene frequency averaging 60.67 percent which is near the average for Iran, show no resemblance to the Arabs of Arabia with the extremely high M gene frequency averaging 74.17 percent.

Regarding neighbouring groups, the frequency of the M gene in the populations of the Caucasus ranges from 55.43 percent in the Azerbaijanians of Barda (Voronov,1973) to 71.00 percent in the Avars of Dagestan (Gadzhiev,1964), with an average M gene frequency of 63.80 percent, the population of the Caucasus appears to exhibit slightly higher M frequencies than those found in Iranians and higher than the European frequencies.

The M gene frequency in the Turkish populations varies between 49.07 percent in the Turks and 57.33 percent in the Eti-Turks (Aksoy et al, 1958). With an average M gene frequency of 55.49 percent, the population of Turkey shows more similarity to the populations of other east Mediterranean countries than to Iranians and other Asiatic populations generally, with their much higher M frequencies.

The frequency of the gene M in the populations of Iraq ranges from 35.19 percent in the Kurdish Jews(Godber et al, 1973) to 98.61 percent in the Karaite Jews (Goldschmidt et al,1976). With an average M gene frequency of 60.00 percent, the Iraqi population appears to exhibit a lower M frequency than that found in Iranians but higher than the European frequency.

The extremely high M gene frequency of 98.61 percent in the Karaite Jews clearly indicates the strong influence of isolation and genetic drift operating in this community.

The M gene frequency in the populations of Kuwait varies between 51.96 percent in the Suluba tribe and 70.19 percent in the Ajman tribe (Khaled et al, 1981). With an average M gene frequency of 59.16 percent, the Kuwaiti population seems to show a lower M gene frequency than that found in

Iranians but higher than that found in Europeans.

The frequency of the gene M in the populations of Saudi Arabia ranges from 63.07 percent in the Shia sample of Qatif and Hasa Oases (Maranjian et al, 1966) to 87.50 percent in western Saudi Arabians (Saha et al, 1980).With an average M gene frequency of 74.17 percent, the Arab population of Saudi Arabia appears to exhibit a much higher frequency of the gene M than that found in Iranians and ofcourse much higher than the European frequency.

High frequencies of the M gene characterize many of the indigenous populations of southern Asia, and from a peak in Arabia, the M gene frequency declines eastwards before rising again in the Indian subcontinent (Mourant et al, 1976).

The Abu-Dhabians of the United Arab Emirates with an M gene frequency of 61.00 percent (Kamel et al, 1980) seem to exhibit a much lower M frequency compared with that found in Saudi Arabians and even lower than the Iranian frequency but still higher than the frequency in Europeans.

The frequency of the M gene in the populations of Pakistan varies between 58.04 percent in the Bengalis (Boyd and Boyd, 1954) and 67.80 percent in the Pathans (Alciati, 1968). With an average M gene frequency of 63.01 percent, the Pakistani population appears to show a slightly higher M gene frequency than that found in Iranians and higher than the European frequency. The M gene frequency tends to be higher than this in most of the Indian region, varying between 65 and 70 percent (Mourant et al, 1976).

The M gene frequency in the Afghan populations ranges from 62.02 percent in the Pushtus (Papiha et al, 1977) to 66.-10 percent in the Timuri and related tribes (Woodd-Walker et

al, 1967). With an average M gene frequency of 64.22 percent, the population of Afghanistan seems to exhibit a higher M gene frequency than that found in Iranians and higher than the European frequency.

The distribution of the MNSs blood groups and respective gene combination frequencies in Iranian and neighbouring populations, expressed in terms of six and nine phenotypes after testing with three and four antisera, respectively, is set out in Tables 5.1.3. b and c.

The gene complex frequencies were combined in Tables 5.1.3* for comparative purposes.

The frequencies of the MNSs complexes in Iranian populations are as follows:

MS ranges from 10.24 percent in a sample from the north of Iran (Bajatzadeh and Walter, 1969) to 48.95 percent in the Baluchis of Sistan and Baluchistan (Present investigation).

Ms varies between 26.28 percent in the Baluchis of Sistan and Baluchistan (Present investigation ) and 51.92 percent in the northern sample of Bajatzadeh and Walter (1969).

NS ranges from 3.43 percent in the Ghashghai tribe (Nijenhuis, 1964) to 16.36 percent in the Kurdish Jews (Godber et al, 1973).

Ns varies between 17.69 percent in the Baluchis of Sistan and Baluchistan and 40.18 percent in the Kurds of Rezaieh (Present study).

The approximate MNSs-complex frequencies in European populations, calculated by the author on the basis of the data available in Mourant et al (1976), are as follows:

| Gene complex | % Frequency |
|---|---|
| MS | 25.33 |
| Ms | 32.55 |
| NS | 7.87 |
| Ns | 34.66 |

On the whole,with average MNSs-complex frequencies of:

| Gene complex | % Frequency |
|---|---|
| MS | 22.92 |
| Ms | 39.43 |
| NS | 9.60 |
| Ns | 28.05 |

the Iranian population appears to exhibit lower MS and Ns but higher Ms and NS-complex frequencies than those found in Europeans.

In Iranians about 37 percent of the M chromosomes carry S while the proportion of NS chromosomes is 25 percent,somewhat more than 1/6 given by Mourant et al (1976) a characteristic of Europeans.

The Kurdish Jews both in Iran and in Iraq appear to be characterized by low MS(average 15.44 and 17.13 percent respectively) and high NS(average 15.98 and 17.62 percent respectively)frequencies.

The Arabs of south western Iran with MNSs complex frequencies of MS 21.21 percent;Ms 36.52 percent; NS 7.14 percent;and Ns 35.13 percent,show no resemblance to the Arabs of Arabia with their extremely high MS(31.40 percent)and low Ns(19.66 percent) frequencies.

Regarding neighbouring populations,the frequencies of the MNSs complexes in the populations of the Caucasus are as follows: MS ranges from 31.02 percent in the Svanis of Georgia, Abkha-

zskaya ( Verbitsky et al, 1971) to 38.85 percent in the Georgians (Kherumian et al, 1954).

Ms varies between 27.08 percent in the Georgians (Kherumian et al, 1954) and 34.48 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1971).

NS ranges from 11.67 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1971) to 14.50 percent in the Georgians (Kherumian et al, 1954).

Ns varies between 19.57 percent in the Georgians (Kherumian et al, 1954) and 22.83 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1971). With average MNSs-complex frequencies of:

| Gene complex | % Frequency |
|---|---|
| MS | 34.94 |
| Ms | 30.78 |
| NS | 13.08 |
| Ns | 21.20 |

the population of the Caucasus appears to exhibit much higher MS and NS but lower Ms and Ns frequencies than those found in Iranians.

In the population of the Caucasus about half the M genes carry S and half s, while of the N genes about 38 percent carry S which is much higher than that in Iranians.

The frequencies of the MNSs complexes in the populations of Turkey (Aksoy et al, 1958) are as follows:

MS ranges from 19.04 percent in the Turks to 31.96 percent in the Eti-Turks.

Ms varies between 25.43 percent in the Eti-Turks and 30.11 percent in the Turks.

NS ranges from 8.52 percent in the Eti-Turks to 9.07 percent

in the Turks.

Ns varies between 34.09 percent in the Eti-Turks and 41.78 percent in the Turks. With average MNSs-complex frequencies of:

| Gene complex | % Frequency |
|---|---|
| MS | 25.50 |
| Ms | 27.77 |
| NS | 8.79 |
| Ns | 37.94 |

the population of Turkey seems to show higher MS and Ns but lower Ms and NS frequencies than those found in Iranians. In the population of Turkey, as in Europeans, about half the M genes carry S and half s, while of the N genes about one-sixth carry S and the rest s.

The frequencies of the MNSs complexes in the populations of Iraq are as follows:

MS ranges from 5.98 percent in the Kurdish Jews (Godber et al, 1973) to 36.10 percent in the Assyrians (Ikin et al, 1965).

Ms varies between 16.59 percent in the Kurdish Jews of the south east (Tills et al, 1977) and 80.60 percent in the Karaite Jews (Goldschmidt et al, 1976).

NS ranges from zero in the Karaite Jews (Goldschmidt et al, 1976) to 27.00 percent in the Kurdish series of Al- Khafaji et al (1976).

Ns varies between 1.40 percent in the Karaite Jews (Goldschmidt et al, 1976) and 41.24 percent in the Kurdish Jews of the south east (Tills et al, 1977).

With average MNSs-complex frequencies of :

| Gene complex | % Frequency |
|---|---|
| MS | 22.08 |
| Ms | 37.49 |
| NS | 15.60 |
| Ns | 24.83 |

the population of Iraq appears to exhibit higher NS but lower MS, Ms and Ns frequencies than those found in Iranians. In the Iraqi population about 37 percent of the M genes carry S, while of the N genes about 38 percent carry S which is more than ¼ a characteristic of Iranians.

The frequencies of the MNSs complexes in the populations of Kuwait are as follow:

MS ranges from 14.00 percent in the Suluba tribe to 38.00 percent in the Ajiman tribe (Khaled et al, 1981).

Ms varies between 30.00 percent in general population of Kuwait (Khaled et al, 1981) and 38.14 percent in the Kuwaiti Arabs (Sawhney, 1975).

NS ranges from 5.09 percent in the Kuwaiti Arabs (Sawhney, 1975) to 25.00 percent in the Suluba tribe (Khaled et al,1981).

Ns varies between 8.00 percent in the Ajiman tribe (Khaled et al, 1981) and 34.53 percent in the Kuwaiti Arabs (Sawhney, 1975).

With average MNSs-complex frequencies of:

| Gene complex | % Frequency |
|---|---|
| MS | 24.56 |
| Ms | 34.54 |
| NS | 18.02 |
| Ns | 22.88 |

the Population of Kuwait seems to show higher MS and NS but lower Ms and Ns frequencies than those found in Iranians.

In the Kuwaiti population about 41 percent of the M genes carry S, while the proportion of NS chromosomes is .44 percent, higher than ¼ in Iranians and much higher than 1/6 in Europeans.

The frequencies of the MNSs complexes in the populations of Saudi Arabia are as follows:

MS ranges from 25.61 percent in the Shia sample of Qatif and Hasa Oases to 36.50 percent in the Sunni sample of Najd(Maranjian et al, 1966).

Ms varies between 37.49 percent in the Shia sample of Qatif and Hasa Oases (Maranjian et al, 1966) and 57.06 percent in western Saudi Arabia (Saha et al, 1980).

NS ranges from 3.37 percent in western Saudi Arabia (Saha et al, 1980) to 9.63 percent in the Bedouins (Maranjian et al, 1966).

Ns varies between 9.13 percent in western Saudi Arabia (Saha et al, 1980) and 28.83 percent in the Shia of Qatif and Hasa Oases (Maranjian et al, 1966).

With average MNSs-complex frequencies of:

| Gene complex | % Frequency |
|---|---|
| MS | 31.40 |
| Ms | 42.80 |
| NS | 6.14 |
| Ns | 19.66 |

the population of Saudi Arabia appears to exhibit higher MS and Ms but lower NS and Ns frequencies than those found in Iranians.

In the Saudi Arabian population about 42 percent of the M genes carry S which is higher than that in Iranians, while of the N genes about 24 percent carry S, a figure similar to

that in Iranians.

The frequencies of the MNSs complexes in the Abu-Dhabians of the United Arab Emirates are as follows:

| Gene complex | % Frequency |
|---|---|
| MS | 24.70 |
| Ms | 36.30 |
| NS | 10.50 |
| Ns | 28.50 |

With the above mentioned MNSs - complex frequencies, the Abu-Dhabians seem to show higher MS, NS and Ns but lower Ms frequencies than those found in Iranians.

In the Abu-Dhabians of the United Arab Emirates about 40 percent of the M chromosomes carry S while the proportion of NS chromosomes is 27 percent, figures nearly similar to those is Saudi Arabians.

The frequencies of the MNSs complexes in the populations of Pakistan are as follows:

MS ranges from 17.71 percent in the Punjabis of Lahore to 32.16 percent in Peshawar (Body and Boyd, 1954).

Ms varies between 32.47 percent in the Gilgit, Hunza (Ikin et al, 1959 ) and 43.49 percent in the Pathans (Alciati, 1968).

NS ranges from 6.65 percent in Peshawar to 13.02 percent in the Bengali series of Dacca (Boyd and Boyd, 1954).

Ns varies between 19.79 percent in the Pathans (Alciati,1968) and 29.84 percent in the Gilgit, Hunza (Ikin et al, 1959).

With average MNSs-complex frequencies of:

| Gene complex | % Frequency |
|---|---|
| MS | 24.70 |
| Ms | 38.36 |
| NS | 11.05 |
| Ns | 25.89 |

the Pakistani population appears to exhibit slightly higher MS and NS but lower Ms and Ns frequencies than those found in Iranians.

In the population of Pakistan about 39 percent of the M genes carry S, while the proportion of NS chromosomes is about 30 percent, somewhat more than that in Iranians.

The MNSs-complex frequencies in the populations of Afghanistan are as follows:

MS ranges from 17.26 percent in the Timuri and related tribes (Woodd-Walker et al,1967)to 29.00 percent in the Pushtus (Papiha et al, 1977).

Ms varies between 33.00 percent in the Pushtus (Papiha et al 1977) and 48.84 percent in the Timuri and related tribes (Woodd-Walker et al, 1967).

NS ranges from 7.50 percent in the Pushtus (Papiha et al,1977) to 9.67 percent in the Timuri and related tribes(Woodd-Walker et al, 1967).

Ns varies between 24.23 percent in the Timuri and related tribes (Woodd-Walker et al,1967)and 30.50 percent in the Pushtus (Papiha et al,1977).

With average MNSs-complex frequencies of:

| Gene complex | % Frequency |
|---|---|
| MS | 23.99 |
| Ms | 40.21 |
| NS | 8.79 |
| Ns | 27.01 |

the Afghan population seems to show slightly higher MS and Ms but lower NS and Ns frequencies than those found in Iranians.

In the population of Afghanistan about 37 percent of the M gene carry S While of the N genes about 24 percent carry S, figures similar to that found in Iranians.

Conclusion

MN frequencies vary less, on the whole, than those of the ABO and Rh groups. Most of the populations tested show M gene frequencies between 50 and 60 percent. Such frequencies are found throughout most of Europe and Africa, and in eastern Asia.

Frequencies of M around the Mediterranean sea, being below 60 percent and mostly below 55 percent, are similar to those found in north western Europe, but eastwards the frequency of M rises steadily to values well above 60 percent in India ( Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the population of Turkey which shows more similarity to the European frequency, the frequency of the M gene, being 60 percent and over, is much higher than that in Europeans, and is characteristic of the 60-65 percent belt which sweeps across Finland to south east Asia, including the Caspian sea area and much of the Arabian Peninsula.

Saudi Arabia shows a very high local maximum with M frequencies above 70 percent.

Regarding the MNSs complexes, In the European populations about half the M genes carry S and halfss, while of theN genes about one-sixth carry S and the rest s (Mourant et al, 1976).

In Iranian and neighbouring populations, again with the

exception of the population of Turkey which shows more similarity to the Europeans, the proportion of MS chromosomes is less while that of NS chromosomes is more than ½ and 1/6 respectively given by Mourant et al (1976) as characteristics of the *European populations*.

The high frequency of 34.94 percent of the MS complex in the population of the Caucasus is noteworthy.

The Arabs of Saudi Arabia, as mentioned above, have an exceptionally high frequency of M.

Frequencies of M are high nearly everywhere in southern Asia, but Arabia represents a high Peak, separated by lower frequencies from the high ones of India and south east Asia. Moreover, M in Arabia is accompanied by exceptionally high frequencies of S, and it is here that the MS combination reaches its highest known frequency other than in American Indians.

Table 5.1.3.a. I MN blood groups distribution in Iran tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | | |
| 1-Kurds Rezaieh | 124 | 31 | 61 | 32 | 49.59 | 50.41 | Present study. | |
| 2-Turks.Azerbaijan | 1043 | 312 | 489 | 242 | 53.36 | 46.64 | Bidar,A. | 1956 |
| 3-Armenians | 78 | 27 | 34 | 17 | 56.41 | 43.59 | Nijenhuis, L.E. | 1964 |
| 4-Kurdish Jews | 106 | 30 | 60 | 16 | 56.60 | 43.40 | Tills, D., et al. | 1977 |
| 5-Kurdish Jews | 94 | 27 | 53 | 14 | 56.91 | 43.09 | Godber,Marilyn J., et al. | 1973 |
| 6-Arabs.Khuzistan | 97 | | | | 57.73 | 42.27 | Marzban, M. | 1978 |
| 7-Turks.Rezaieh | 139 | 49 | 65 | 25 | 58.63 | 41.37 | Present study. | |
| 8-Iranians | 56 | 19 | 28 | 9 | 58.93 | 41.07 | Nijenhuis, L.E. | 1964 |
| 9-Jews | 200 | 63 | 111 | 26 | 59.25 | 40.75 | Gurevitch, J.,et al. | 1956 |
| 10-Tehran | 113 | 43 | 49 | 21 | 59.73 | 40.27 | Bajatzadeh,M., & Walter, H. | 1969 |
| 11-KUrds.Kermanshah | 127 | 47 | 58 | 22 | 59.84 | 40.16 | Nijenhuis, L.E. | 1964 |
| 12-Kurds.Sanandaj | 107 | 44 | 42 | 21 | 60.75 | 39.25 | Lehmann,H.,et al. | 1973 |

Table 5.1.3.a.I(Cont.) MN blood groups distribution in Iran tested with anti-M and anti- N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | | |
| 13-Zoroastrians | 74 | 27 | 36 | 11 | 60.81 | 39.19 | Present study. | |
| 14-North | 58 | 24 | 24 | 10 | 62.07 | 37.93 | Bajatzadeh,M., & Walter, H. | 1969 |
| 15-Assyrians.Abadan | 32 | 12 | 16 | 4 | 62.50 | 37.50 | Nijenhuis, L.E. | 1964 |
| 16-Shi'a Yazd | 151 | 57 | 75 | 19 | 62.58 | 37.42 | Sunderland,E., & Smith, H.M. | 1966 |
| 17-Turks.Mainly south east | 348 | 128 | 181 | 39 | 62.79 | 37.21 | Nijenhuis,L.E. | 1964 |
| 18-Central and South | 109 | 48 | 41 | 20 | 62.85 | 37.15 | Bajatzadeh,M., & Walter, H. | 1969 |
| 19-Moslems.Tehran | 256 | 104 | 117 | 35 | 63.48 | 36.52 | Boue' and Boue'. | 1956 |
| 20-Arabs. Abadan | 158 | 62 | 77 | 19 | 63.61 | 36.39 | Nijenhuis,L.E. | 1964 |
| 21-Kurds.Baneh , Marivan | 77 | 33 | 32 | 12 | 63.64 | 36.36 | Lehmann,H., et al. | 1973 |
| 22-East | 67 | 28 | 30 | 9 | 64.18 | 35.82 | Bajatzadeh,M., & Walter, H. | 1969 |
| 23-Jews | 108 | 54 | 33 | 21 | 65.30 | 34.70 | Tabatabai, H. | 1977 |

Table 5.1.3.a. I (Cont) MN blood groups distribution in Iran tested with anti-M and Anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | | |
| 24-Zabolis.Sistan & Baluchistan | 114 | 52 | 45 | 17 | 65.35 | 34.65 | Present study. | |
| 25-West | 107 | 48 | 44 | 15 | 65.42 | 34.58 | Bajatzadeh,M., & Walter, H. | 1969 |
| 26-North west | 66 | 31 | 25 | 10 | 65.91 | 34.09 | Bajatzadeh,M., & Walter, H. | 1969 |
| 27-Ghashghais | 66 | 27 | 33 | 6 | 65.91 | 34.09 | Nijenhuis,L.E. | 1964 |
| 28-Tehran | 137 | 60 | 62 | 15 | 66.42 | 33.58 | Sawhney, K.S. | 1975 |
| 29-Bakhtiaris | 138 | 62 | 60 | 16 | 66.67 | 33.33 | Nijenhuis, L.E. | 1964 |
| 30-Esfahan | 111 | 56 | 40 | 15 | 68.47 | 31.53 | Sawhney, K.S. | 1975 |
| 31-Armenians | 145 | 80 | 46 | 19 | 71.10 | 28.90 | Tabatabai, H. | 1977 |
| 32-Lurs.Luristan | 153 | 83 | 54 | 16 | 71.90 | 28.10 | Present study. | |
| 33-Baluchis.Sistan & Baluchistan | 111 | 63 | 41 | 7 | 75.23 | 24.77 | Present study. | |

Table 5.1.3.a. II MN blood groups distribution in the Caucasus tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | | |
| 1-Azerbaijanians.Barda | 147 | 38 | 87 | 22 | 55.43 | 44.57 | Voronov, A.A. | 1973 |
| 2-Dagestan.Various tribes | 161 | 75 | 39 | 47 | 58.70 | 41.30 | Kherumian, R. | 1948 |
| 3-Caucasus(different regions) | 641 | 218 | 321 | 102 | 59.05 | 40.95 | Voronov, A.A.,et al. | 1967 |
| 4-Azerbaijanians. Shemakha | 180 | 70 | 74 | 36 | 59.44 | 40.56 | Voronov, A.A. | 1973 |
| 5-Gurians.Lanchkhut | 98 | | | | 61.23 | 38.77 | Voronov ,A.A. | 1973 |
| 6-Georgia,Gori | 428 | 164 | 204 | 60 | 62.15 | 37.85 | Solovyeva, T.G. | 1967 |
| 7- Kakhetians ,Gurdjaani | 196 | 73 | 99 | 24 | 62.50 | 37.50 | Voronov, A.A. | 1973 |
| 8-Georgia,Tbilisi | 500 | 198 | 230 | 72 | 62.60 | 37.40 | Boyd,W.C.,et al. | 1937 |
| 9-Dagestan.Kumky | 178 | 79 | 66 | 33 | 62.92 | 37.08 | Gadzhiev, A.G. | 1964 |
| 10-Azerbaijanians. Nukha | 248 | 104 | 105 | 39 | 63.15 | 36.85 | Voronov, A.A. | 1973 |
| 11-Megrelians.Abashi | 192 | 85 | 75 | 32 | 63.85 | 36.15 | Voronov, A.A. | 1973 |
| 12-Georgia,Abkhazskaya. Svanis | 655 | 275 | 308 | 72 | 65.50 | 34.50 | Verbitsky,M.Sh., et al. | 1971 |

Table 5.1.3.a. II (Cont.) MN blood groups distribution in the Caucasus tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | | |
| 13-Georgians abroad . Autochthonous | 41 | 20 | 14 | 7 | 65.85 | 34.15 | Kherumian,R., et al. | 1954 |
| 14-Dagestan.Darghian | 179 | 82 | 77 | 20 | 67.32 | 32.68 | Gadzhiev, A.G. | 1964 |
| 15-Dagestan. Lezgian | 196 | 98 | 72 | 26 | 68.37 | 31.63 | Gadzhiev, A.G. | 1964 |
| 16-Dagestan.Lakts | 151 | 76 | 55 | 20 | 68.54 | 31.46 | Gadzhiev, A.G. | 1964 |
| 17-Abkhazians.Ochamchiri | 91 | | | | 70.88 | 29.12 | Voronov, A.A. | 1973 |
| 18-Dagestan.Avar | 200 | 100 | 84 | 16 | 71.00 | 29.00 | Gadzhiev, A.G. | 1964 |

Table 5.1.3.a. III MN blood groups distribution in Turkey tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | | |
| 1-Turks.Asia Minor, Mersin | 108 | 27 | 52 | 29 | 49.07 | 50.93 | Aksoy,M., et al.. | 1958 |
| 2-Turkish | 1266 | 411 | 619 | 236 | 56.91 | 43.09 | Baecher. | 1935 |
| 3-Kurds | | | | | 57.00 | 43.00 | Richard, P. | 1976 |
| 4-Turkish Donors | 2361 | 763 | 1172 | 426 | 57.14 | 42.86 | Mizan, N., et al. | 1965 |
| 5-Eti- Turks. Asia Minor near Mersin | 116 | 43 | 47 | 26 | 57.33 | 42.67 | Aksoy,M., et al. | 1958 |

Table 5.1.3.a. IV MN blood groups distribution in Iraq tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | | |
| 1-Kurdish Jews | 27 | 4 | 11 | 12 | 35.19 | 64.81 | Godber,Marilyn J., et al. | 1973 |
| 2-Kurdish Jews.South east | 50 | 11 | 21 | 18 | 43.00 | 57.00 | Tills, D., et al. | 1977 |
| 3-Jews.Baghdad | 215 | 57 | 101 | 57 | 50.00 | 50.00 | Boyd,W.C., & Boyd, Lyle G. | 1941 |
| 4-Jews. Kurdistan | 120 | 37 | 53 | 30 | 52.92 | 47.08 | Gurevitch,J., & Margolis, E. | 1955 |
| 5-Kurds | | | | | 56.00 | 44.00 | Al-Khafagi,S.D., et al. | 1976 |
| 6-Bedouins | 139 | 47 | 64 | 28 | 56.83 | 43.17 | Kayssi,A.I.,et al. | 1938 |
| 7-Moslems.Baghdad | 387 | 143 | 182 | 62 | 60.47 | 39.53 | Boyd,W.C., & Boyd,Lyle G. | 1941 |
| 8-Jews. Baghdad | 162 | 66 | 64 | 32 | 60.49 | 39.51 | Gurevitch,J., & Margolis, E. | 1955 |
| 9-Assyrians | 99 | 37 | 46 | 16 | 60.61 | 39.39 | Ikin,Elizabeth W., et al. | 1956 |

Table 5.1.3.a. IV (Cont.) MN blood groups distribution in Iraq tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | | |
| 10-Bedouins.Mosul | 206 | 76 | 102 | 28 | 61.65 | 38.35 | Boyd, W.C. | 1939 |
| 11-Christians.Baghdad | 63 | 24 | 30 | 9 | 61.90 | 38.10 | Boyd,W.C., and Boyd, Lyle.G. | 1941 |
| 12-Kurdish Jews.North west | 61 | 29 | 20 | 12 | 63.93 | 36.07 | Tills.D., et al. | 1977 |
| 13-Bedouins | 199 | 82 | 99 | 18 | 66.08 | 33.92 | Kayssi,A.I.,et al. | 1938 |
| 14-Kurds.Baghdad | 29 | 17 | 8 | 4 | 72.41 | 27.59 | Boyd,W.C., and Boyd, Lyle,G. | 1941 |
| 15-Karaite Jews | 72 | 70 | 2 | - | 98.61 | 1.39 | Goldschmidt,E., et al. | 1976 |

Table 5.1.3.a. V MN blood groups distribution in Kuwait tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | | |
| 1-Suluba tribe | 51 | 8 | 37 | 6 | 51.96 | 48.04 | Khaled, E., et al. | 1981 |
| 2-General population | 73 | 16 | 47 | 10 | 54.11 | 45.89 | Khaled, E., et al. | 1981 |
| 3-Kuwaiti Arabs | 159 | 52 | 88 | 19 | 60.38 | 39.62 | Sawhney,K.S. | 1975 |
| 4-Ajman tribe | 52 | 22 | 29 | 1 | 70.19 | 29.81 | Khaled,E., et al. | 1981 |

Table 5.1.3.a. VI MN blood groups distribution in Saudi Arabia tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | |
| 1-Shias.Qatif and Hasa Oases | 463 | 188 | 208 | 67 | 63.07 | 36.93 | Maranjian,G.,et al. 1966 |
| 2-Sunnis.Qatif and Hasa Oases | 323 | 151 | 134 | 38 | 67.49 | 32.51 | Maranjian,G.,et al. 1966 |
| 3-Sunnis.Hejaz,Asir,Najran | 176 | 100 | 61 | 15 | 74.15 | 25.85 | Maranjian,G.,et al. 1966 |
| 4-Sunnis. Najd | 180 | 104 | 62 | 14 | 75.00 | 25.00 | Maranjian,G.,et al. 1966 |
| 5-Bedouins | 178 | 109 | 59 | 10 | 77.81 | 22.19 | Maranjian,G.,et al. 1966 |
| 6-Western Saudi Arabia | 176 | 145 | 18 | 13 | 87.50 | 12.50 | Saha, N.,et al. 1980 |

Table 5.1.3.a. VII MN blood groups distribution in the United Arab Emirates tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | |
| 1-Abu-Dhabians | 100 | 39 | 44 | 17 | 61.00 | 39.00 | Kamel,K.,et al. 1980 |

Table 5.1.3.a VIII MN blood groups distribution in Pakistan tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | | |
| 1-Bengalis. Dacca | 230 | 79 | 109 | 42 | 58.04 | 41.96 | Boyd,W.C., & Boyd,L.G. | 1954 |
| 2-Punjabis.Lahore | 202 | 69 | 97 | 36 | 58.17 | 41.83 | Boyd,W.C., & Boyd,L.G. | 1954 |
| 3-Gilgit.Hunza | 23 | 10 | 8 | 5 | 60.87 | 39.13 | Ikin,Elizabeth W., et al. | 1959 |
| 4-Peshawar | 153 | 64 | 73 | 16 | 65.69 | 34.31 | Boyd,W.C., & Boyd,L.G. | 1954 |
| 5-Baltis.Baltistan | 80 | 38 | 32 | 10 | 67.50 | 32.50 | Clegg,E.J.,et al. | 1961 |
| 6-Pathans.Swat.Saidu Sharif | 132 | 65 | 49 | 18 | 67.80 | 32.20 | Alciati, G. | 1968 |

Table 5.1.3.a IX MN blood groups distribution in Afghanistan tested with anti-M and anti-N sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | MM | MN | NN | M | N | |
| 1-Pushtus | 104 | 39 | 51 | 14 | 62.02 | 37.98 | Papiha, S.S.,et al. 1977 |
| 2-Daris | 179 | 82 | 67 | 30 | 64.53 | 35.47 | Papiha, S.S.,et al. 1977 |
| 3-Timuri and related tribes | 118 | 52 | 52 | 14 | 66.10 | 33.90 | Woodd-Walker,R.B., 1967 et al. |

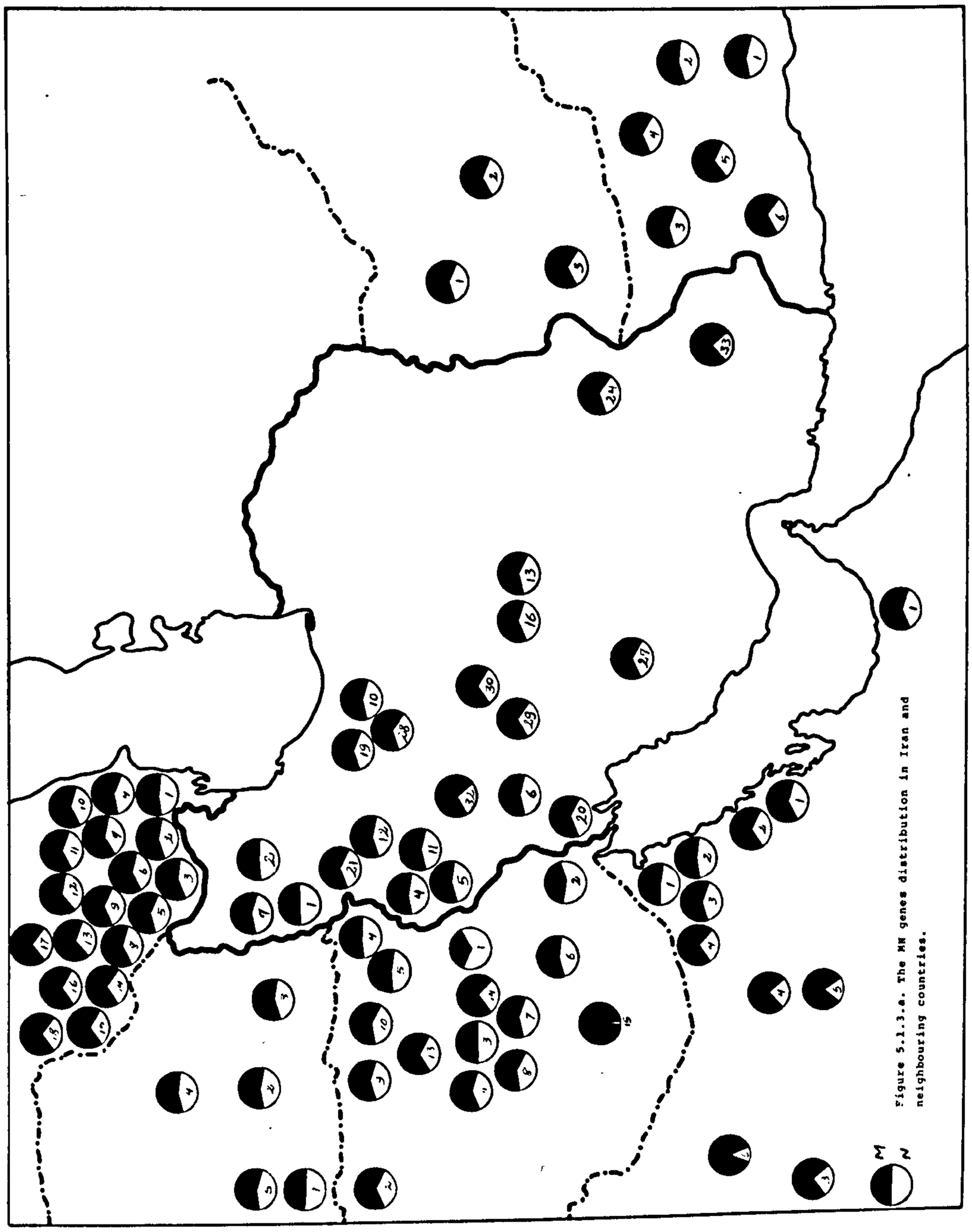


Figure 5.1.3.4. The MM genes distribution in Iran and neighbouring countries.

Table 5.1.3.b. I MNSs blood groups distribution in Iran tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | MMSS | MMss | MNSS | MNss | NNSS | NNss |
| 1-North | 58 | 5 | 19 | 13 | 11 | 1 | 9 |
| 2-Kurdish Jews | 94 | 12 | 15 | 30 | 23 | 8 | 6 |
| 3-Tehran | 113 | 20 | 23 | 20 | 29 | 10 | 11 |
| 4-Kurdish Jews | 106 | 13 | 17 | 35 | 25 | 8 | 8 |
| 5-East | 67 | 13 | 15 | 10 | 20 | 3 | 6 |
| 6-Iranians | 56 | 13 | 6 | 10 | 18 | 6 | 3 |
| 7-Central and South | 109 | 25 | 23 | 21 | 20 | 8 | 12 |
| 8-North West | 66 | 15 | 16 | 12 | 13 | 2 | 8 |
| 9-West | 107 | 24 | 24 | 24 | 20 | 5 | 10 |
| 10-Ghashghais | 37 | 12 | 2 | 9 | 10 | 1 | 3 |

Table 5.1.3.b.I.(Cont). MNSs blood groups distribution in Iran tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Gene complex frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | | |
| 1-North | 58 | 10.24 | 51.92 | 7.80 | 30.04 | Bajatzadeh,M., & Walter, H. | 1969 |
| 2-Kurdish Jews | 94 | 15.18 | 41.73 | 16.36 | 26.73 | Godber,Marilyn.J., et al. | 1973 |
| 3-Tehran | 113 | 15.32 | 44.47 | 10.05 | 30.16 | Bajatzadeh,M., & Walter, H. | 1969 |
| 4-Kurdish Jews | 106 | 15.70 | 40.90 | 15.60 | 27.80 | Tills,D., et al. | 1977 |
| 5-East | 67 | 16.34 | 47.93 | 5.55 | 30.18 | Bajatzadeh,M., & Walter, H. | 1969 |
| 6-Iranians | 56 | 19.90 | 39.08 | 10.73 | 30.29 | Nijenhuis, L.E. | 1964 |
| 7-Central and South | 109 | 20.03 | 42.87 | 8.99 | 28.11 | Bajatzadeh,M., & Walter, H. | 1969 |
| 8-North West | 66 | 20.44 | 45.52 | 4.74 | 29.30 | Bajatzadeh,M.,& Walter, H. | 1969 |
| 9-West | 107 | 20.89 | 44.61 | 8.15 | 26.35 | Bajatzadeh,M., & Walter, H. | 1969 |
| 10-Ghashghais | 37 | 33.18 | 30.40 | 3.43 | 32.99 | Nijenhuis,L.E. | 1964 |

Table 5.1.3. C.I MNSs blood groups distribution in Iran tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | MMSS | MMSs | MMss | MNSS | MNSs | MNss | NNSS | NNSs | NNss |
| 1-Kurds.Rezaieh | 124 | 2 | 11 | 18 | 2 | 19 | 40 | 2 | 9 | 21 |
| 2-Turks.Rezaieh | 139 | 4 | 19 | 26 | 7 | 28 | 30 | 4 | 11 | 10 |
| 3-Zoroastrians | 74 | 2 | 12 | 13 | 4 | 16 | 16 | 2 | 4 | 5 |
| 4-Arabs.Khuzistan | 97 | | | | | | | | | |
| 5-Shi'a.Yazd | 151 | 10 | 25 | 22 | 10 | 30 | 35 | 3 | 4 | 12 |
| 6-Zabolis.Sistan & Baluchistan | 114 | 6 | 28 | 18 | 4 | 18 | 23 | 2 | 4 | 11 |
| 7-Kurds.Sanandaj | 107 | 11 | 22 | 11 | 4 | 17 | 21 | 3 | 7 | 11 |
| 8-Kurds.Baneh,Marivan | 77 | 11 | 13 | 9 | 7 | 10 | 15 | 2 | 3 | 7 |
| 9-Tehran | 137 | 18 | 19 | 23 | 9 | 32 | 21 | 2 | 4 | 9 |
| 10-Lurs.Luristan | 153 | 22 | 41 | 20 | 9 | 30 | 15 | 1 | 6 | 9 |
| 11-Baluchis.Sistan & Baluchistan | 111 | 27 | 28 | 8 | 10 | 22 | 9 | 1 | 2 | 4 |

Table 5.1.3.C.I.(Cont.) MNSs blood groups distribution in Iran tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Gene complex frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | | |
| 1-Kurds.Rezaieh | 124 | 11.99 | 37.60 | 10.23 | 40.18 | Present study. | |
| 2-Turks.Rezaieh | 139 | 16.15 | 42.48 | 15.73 | 25.64 | Present study. | |
| 3-Zoroastrians | 74 | 18.01 | 42.80 | 14.24 | 24.95 | Present study. | |
| 4-Arabs.Khuzistan | 97 | 21.21 | 36.52 | 7.14 | 35.13 | Marzban,M. | 1978 |
| 5-Shi'a,Yazd | 151 | 24.62 | 38.04 | 10.29 | 27.05 | Sunderland,E.,& Smith, H.M. | 1966 |
| 6-Zabolis.Sistan & Baluchistan | 114 | 24.62 | 40.73 | 7.84 | 26.81 | Present study . | |
| 7-Kurds.Sanandaj | 107 | 28.06 | 32.69 | 10.26 | 28.99 | Lehmann, H., et al. | 1973 |
| 8-Kurds.Baneh,Marivan | 77 | 31.79 | 31.85 | 11.06 | 25.30 | Lehmann, H., et al. | 1973 |
| 9-Tehran | 137 | 31.87 | 34.55 | 9.38 | 24.20 | Sawhney, K.S. | 1975 |
| 10-Lurs.Luristan | 153 | 36.81 | 35.08 | 7.02 | 21.09 | Present study. | |
| 11-Baluchis.Sistan & Baluchistan | 111 | 48.95 | 26.28 | 7.08 | 17.69 | Present study. | |

Table 5.1.3.d.I. MN blood groups distribution in Iran tested with anti-He ( Henshaw ) serum

| Population | Number Tested | He+ | He- | He+% | Authors | |
|---|---|---|---|---|---|---|
| 1-Kurds.Kermanshah | 122 | - | 122 | | Nijenhuis,L.E. | 1964 |
| 2-Ghashghai.Fars.South of Shiraz | 8 | - | 8 | | Nijenhuis,L.E. | 1964 |
| 3-Bakhtiari.Zagros Mountain | 131 | - | 131 | | Nijenhuis,L.E. | 1964 |
| 4-Iranians | 76 | - | 76 | | Nijenhuis,L.E. | 1964 |
| 5-Assyrians | 14 | - | 14 | | Nijenhuis,L.E. | 1964 |
| 6-Armenians | 9 | - | 9 | | Nijenhuis,L.E. | 1964 |
| 7-Kurdish Jews | 121 | - | 121 | | Godber,Marilyn,J., et al. | 1973 |
| 8-Kurds.Sanandaj | 107 | - | 107 | | Lehmann,H.,et al. | 1973 |
| 9-Kurds.Baneh,Marivan | 77 | - | 77 | | Lehmann,H.,et al. | 1973 |
| 10-Kurdish Jews | 106 | - | 106 | | Tills,D., et al. | 1977 |

Table 5.1.3. * I MNSs-complex frequencies distribution in Iran

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-North | 58 | 10.24 | 51.92 | 7.80 | 30.04 | Bajatzadeh,M.,& Walter, H. 1969 |
| 2-Kurds.Rezaieh | 124 | 11.99 | 37.60 | 10.23 | 40.18 | Present study. |
| 3-Kurdish Jews | 94 | 15.18 | 41.73 | 16.36 | 26.73 | Godber,Marilyn J., et al. 1973 |
| 4-Tehran | 113 | 15.32 | 44.47 | 10.05 | 30.16 | Bajatzadeh,M., & Walter, H. 1969 |
| 5-Kurdish Jews | 106 | 15.70 | 40.90 | 15.60 | 27.80 | Tills, D., et al. 1977 |
| 6-Turks.Rezaieh | 139 | 16.15 | 42.48 | 15.73 | 25.64 | Present study. |
| 7-East | 67 | 16.34 | 47.93 | 5.55 | 30.18 | Bajatzadeh,M., & Walter, H. 1969 |
| 8-Zoroastrians | 74 | 18.01 | 42.80 | 14.24 | 24.95 | Present study. |
| 9-Iranians | 56 | 19.90 | 39.08 | 10.73 | 30.29 | Nijenhuis,L.E. 1964 |
| 10-Central & South | 109 | 20.03 | 42.87 | 8.99 | 28.11 | Bajatzadeh,M.,& Walter, H. 1969 |
| 11-North west | 66 | 20.44 | 45.52 | 4.74 | 29.30 | Bajatzadeh,M., & Walter,H. 1969 |

Table 5.1.3. * I (Cont.) MNSs-complex frequencies distribution in Iran

| Population | Number Tested | Gene complex frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | | |
| 12-West | 107 | 20.89 | 44.61 | 8.15 | 26.35 | Bajatzadeh,M., & Walter, H. | 1969 |
| 13-Arabs.Khuzistan | 97 | 21.21 | 36.52 | 7.14 | 35.13 | Marzban, M. | 1978 |
| 14-Shi'a . Yazd | 151 | 24.62 | 38.04 | 10.29 | 27.05 | Sunderland,E.,& Smith, H.M. | 1966 |
| 15-Zabolis. Sistan & Baluchistan | 114 | 24.62 | 40.73 | 7.84 | 26.81 | Present study. | |
| 16-Kurds. Sanandaj | 107 | 28.06 | 32.69 | 10.26 | 28.99 | Lehmann, H.,et al. | 1973 |
| 17-Kurds.Baneh, Marivan | 77 | 31.79 | 31.85 | 11.06 | 25.30 | Lehmann, H.,et al. | 1973 |
| 18-Tehran | 137 | 31.87 | 34.55 | 9.38 | 24.20 | Sawhney, K.S. | 1975 |
| 19-Ghashghais | 37 | 33.18 | 30.40 | 3.43 | 32.99 | Nijenhuis, L.E. | 1964 |
| 20-Lurs.Luristan | 153 | 36.81 | 35.08 | 7.02 | 21.09 | Present study. | |
| 21-Baluchis.Sistan & Baluchistan | 111 | 48.95 | 26.28 | 7.08 | 17.69 | Present study. | |

Table 5.1.3.b.* II MNSs blood groups distribution in the Caucasus tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | MMSS | MMss | MNSS | MNss | NNSS | NNss |
| 1-Svanis.. Georgia.Abkhazskaya | 655 | 196 | 79 | 206 | 102 | 38 | 34 |
| 2-Georgians | 41 | 17 | 3 | 10 | 4 | 5 | 2 |

Table 5.1.3.b.*II (Cont.). MNSs blood groups distribution in the Caucasus tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Svanis . Georgia.Abkhazskaya | 655 | 31.02 | 34.48 | 11.67 | 22.83 | Verbitsky,M.Sh.,et al.1971 |
| 2-Georgians | 41 | 38.85 | 27.08 | 14.50 | 19.57 | Kherumian,R.,et al. 1954 |

Table 5.1.3.b.* III MNSs blood groups distribution in Turkey tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | MMSS | MMss | MNSS | MNss | NNSS | NNss |
| 1-Turks.Asia Minor, Mersin | 108 | 18 | 9 | 24 | 28 | 10 | 19 |
| 2-Eti-Turks. Asia Minor, near Mersin | 116 | 36 | 7 | 28 | 19 | 10 | 16 |

Table 5.1.3.b.*III (Cont.) MNSs blood groups distribution in Turkey tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Gene complex frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | | |
| 1-Turks. Asia Minor, Mersin | 108 | 19.04 | 30.11 | 9.07 | 41.78 | Aksoy,M.,et al. | 1958 |
| 2-Eti-Turks.Asia Minor, near Mersin | 116 | 31.96 | 25.43 | 8.52 | 34.09 | Aksoy,M.,et al. | 1958 |

Table 5.1.3.b. IV MNSs blood groups distribution in Iraq tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | MMSS | MMss | MNSS | MNss | NNSS | NNss |
| 1-Kurdish Jews | 27 | 2 | 2 | 3 | 8 | 9 | 3 |
| 2-Kurdish Jews. North west | 61 | 15 | 14 | 10 | 10 | 7 | 5 |
| 3-Kurdish Jews; South east | 50 | 10 | 1 | 14 | 7 | 9 | 9 |
| 4-Kurds | | | | | | | |
| 5-Assyrians | 99 | 32 | 5 | 32 | 14 | 10 | 6 |

Table 5.1.3.b. IV (Cont.) MNSs blood groups distribution in Iraq tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Kurdish Jews | 27 | 5.98 | 29.21 | 25.02 | 39.79 | Godber,Marilyn.J., et al. 1973 |
| 2-Kurdish Jews.North west | 61 | 18.99 | 44.94 | 12.07 | 24.00 | Tills,D., et al. 1977 |
| 3-Kurdish Jews. South east | 50 | 26.41 | 16.59 | 15.76 | 41.24 | Tills,D., et al. 1977 |
| 4-Kurds | | 27.00 | 29.00 | 27.00 | 17.00 | Al-Khafaji,S.D.,et al.1976 |
| 5-Assyrians | 99 | 36.10 | 24.59 | 13.78 | 25.53 | Ikin,Elizabeth W., et al. 1965 |

Table 5.1.3.C. IV MNSs blood groups distribution in Iraq tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | MMSS | MMSs | MMss | MNSS | MNSs | MNss | NNSS | NNSs | NNss |
| 1-Karaite Jews | 72 | 2 | 22 | 46 | - | - | 2 | - | - | - |

Table 5.1.3.C.IV (Cont.) MNSs blood groups distribution in Iraq tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Karaite Jews | 72 | 18.00 | 80.60 | 0.00 | 1.40 | Goldschmidt,Elizabeth., 1976 et al. |

Table 5.1.3.d. IV MN blood groups distribution in Iraq tested with anti-He ( Henshaw ) serum

| Population | Number Tested | He+ | He- | He+% | Authors |
|---|---|---|---|---|---|
| 1-Kurdish Jews..South east | 50 | - | 50 | | Tills,D., et al. 1977 |
| 2-Kurdish Jews..North west | 61 | - | 61 | | Tills,D., et al. 1977 |

Table 5.1.3. * IV. MNSs - complex frequencies distribution in Iraq

| Population | Number Tested | Gene complex frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | | |
| 1-Kurdish Jews | 27 | 5.98 | 29.21 | 25.02 | 39.79 | Godber,Marilyn J.,et al. | 1973 |
| 2-Karaite Jews | 72 | 18.00 | 80.60 | 0.00 | 1.40 | Goldschmidt,Elizabeth., et al. | 1976 |
| 3-Kurdish Jews. North west | 61 | 18.99 | 44.94 | 12.07 | 24.00 | Tills,D., et al. | 1977 |
| 4-Kurdish Jews. South east | 50 | 26.41 | 16.59 | 15.76 | 41.24 | Tills,D., et al. | 1977 |
| 5-Kurds | | 27.00 | 29.00 | 27.00 | 17.00 | Al-Khafaji,S.D.,et al. | 1976 |
| 6-Assyrians | 99 | 36.10 | 24.59 | 13.78 | 25.53 | Ikin,Elizabeth W.,et al. | 1965 |

Table 5.1.3.b. V MNSs blood groups distribution in Kuwait tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | MMSS | MMss | MNSS | MNss | NNSS | NNss |
| 1- Kuwaiti Arabs | 159 | 29 | 23 | 43 | 45 | 3 | 16 |

Table 5.1.3.b. V (Cont.). MNSs blood groups distribution in Kuwait tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Kuwaiti Arabs | 159 | 22.24 | 38.14 | 5.09 | 34.53 | Sawhney, K.S. 1975 |

Table 5.1.3.C.V MNSs blood groups distribution in Kuwait tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | MMSS | MMSs | MMss | MNSS | MNSs | MNss | NNSS | NNSs | NNss |
| 1-Suluba tribe | 51 | 1 | 3 | 4 | 4 | 21 | 12 | 2 | 2 | 2 |
| 2-General population | 73 | 5 | 3 | 8 | 13 | 18 | 16 | 1 | 5 | 4 |
| 3-Ajman tribe | 52 | 7 | 8 | 7 | 14 | 11 | 4 | - | 1 | - |

Table 5.1.3.C.V (Cont.) MNSs blood groups distribution in Kuwait tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Gene complex frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | | |
| 1-Suluba tribe | 51 | 14.00 | 38.00 | 25.00 | 23.00 | Khaled,E., et al. | 1981 |
| 2-General population | 73 | 24.00 | 30.00 | 20.00 | 26.00 | Khaled,E., et al. | 1981 |
| 3-Ajman tribe | 52 | 38.00 | 32.00 | 22.00 | 8.00 | Khaled,E., et al. | 1981 |

Table 5.1.3. * V MNSs- complex frequencies distribution in Kuwait

| Population | Number Tested | Gene complex frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | | |
| 1-Suluba tribe | 51 | 14.00 | 38.00 | 25.00 | 23.00 | Khaled,E., et al. | 1981 |
| 2-Kuwaiti Arabs | 159 | 22.24 | 38.14 | 5.09 | 34.53 | Sawhney, K.S. | 1975 |
| 3-General population | 73 | 24.00 | 30.00 | 20.00 | 26.00 | Khaled, E., et al. | 1981 |
| 4-Ajman tribe | 52 | 38.00 | 32.00 | 22.00 | 8.00 | Khaled, E., et al. | 1981 |

Table 5.1.3.b. VI MNSs blood groups distribution in Saudi Arabia tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | MMSS | MMss | MNSS | MNss | NNSS | NNss |
| 1-Shias.Qatif & Haza Oases | 463 | 123 | 65 | 109 | 99 | 27 | 40 |
| 2-Sunnis.Qatif & Haza Oases | 323 | 92 | 59 | 75 | 59 | 8 | 30 |
| 3-Sunnis.Hejaz,Asir, Najran | 176 | 69 | 31 | 40 | 21 | 6 | 9 |
| 4-Bedouins | 178 | 73 | 36 | 44 | 15 | 5 | 5 |
| 5-Sunnis.Najd | 180 | 74 | 30 | 39 | 23 | 3 | 11 |

Table 5.1.3.b. VI (Cont.) MNSs blood groups distribution in Saudi Arabia tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Shias.Qatif & Haza Oases | 463 | 25.61 | 37.49 | 8.07 | 28.83 | Maranjian,G.,et al. 1966 |
| 2-Sunnis. Qatif & Haza Oases | 323 | 27.45 | 40.09 | 4.81 | 27.65 | Maranjian,G.,et al. 1966 |
| 3-Sunnis.Hejaz,Asir,Najran | 176 | 34.07 | 40.111 | 7.11 | 18.71 | Maranjian,G.,et al. 1966 |
| 4-Bedouins | 178 | 34.31 | 43.53 | 9.63 | 12.53 | Maranjian,G.,et al. 1966 |
| 5-Sunnis.Najd | 180 | 36.50 | 38.52 | 3.85 | 21.13 | Maranjian,G.,et al. 1966 |

Table 5.1.3.C. VI MNSs blood groups distribution in Saudi Arabia tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | MMSS | MMSs | MMss | MNSS | MNSs | MNss | NNSS | NNSs | NNss |
| 1-Western Saudi Arabia | 176 | 38 | 26 | 81 | 1 | 7 | 10 | 3 | 2 | 8 |

Table 5.1.3.C.VI (Cont.) MNSs blood groups distribution in Saudi Arabia tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Western Saudi Arabia | 176 | 30.44 | 57.06 | 3.37 | 9.13 | Saha, N., et al. 1980 |

Table 5.1.3.d. VI MN blood groups distribution in Saudi Arabia tested with anti-He (Henshaw ) serum

| Population | Number Tested | He+ | He- | He+% | Authors | |
|---|---|---|---|---|---|---|
| 1-Bedouins | 156 | - | 156 | | Maranjian,G.,et al. | 1966 |
| 2-Sunnis.Najd | 180 | - | 180 | | Maranjian,G.,et al. | 1966 |
| 3-Sunnis.Asir,Hejaz, Najran | 168 | - | 168 | | Maranjian,G.,et al. | 1966 |
| 4-Shias.Qatif & Hasa Oases | 412 | 4 | 408 | 0.97 | Maranjian,G.,et al. | 1966 |
| 5-Sunnis. Qatif & Hasa Oases | 287 | 4 | 283 | 1.39 | Maranjian,G.,et al. | 1966 |

Table 5.1.3. *. VI MNSs - complex frequencies distribution in Saudi Arabia

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Shias.Qatif & Hasa Oases | 463 | 25.61 | 37.49 | 8.07 | 28.83 | Maranjian,G.,et al. 1966 |
| 2-Sunnis.Qatif & Hasa Oases | 323 | 27.45 | 40.09 | 4.81 | 27.65 | Maranjian,G.,et al. 1966 |
| 3-Western Saudi Arabia | 176 | 30.44 | 57.06 | 3.37 | 9.13 | Saha,N.,et al. 1980 |
| 4-Sunnis,Hejaz,Asir,Najran | 176 | 34.07 | 40.11 | 7.11 | 18.71 | Maranjian,G.,et al. 1966 |
| 5-Bedouins | 178 | 34.31 | 43.53 | 9.63 | 12.53 | Maranjian,G.,et al. 1966 |
| 6-Sunnis.Najd | 180 | 36.50 | 38.52 | 3.85 | 21.13 | Maranjian,G.,et al. 1966 |

Table 5.1.3.b.*VII MNSs blood groups distribution in the United Arab Emirates tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | MMSS | MMss | MNSS | MNss | NNSS | NNss |
| 1-Abu-Dhabians | 100 | 25 | 14 | 25 | 19 | 8 | 9 |

Table 5.1.3.b.*VII (Cont.) MNSs blood groups distribution in the United Arab Emirates tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Abu-Dhabians | 100 | 24.70 | 36.30 | 10.50 | 28.50 | Kamel,K.,et al. 1980 |

Table 5.1.3.b.*VIII MNSs blood groups distribution in Pakistan tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | MMSS | MMss | MNSS | MNss | NNSS | NNss |
| 1-Punjabis.Lahore | 202 | 34 | 35 | 53 | 44 | 17 | 19 |
| 2-Bengalis.Dacca | 230 | 44 | 35 | 62 | 47 | 21 | 21 |
| 3-Pathans.Swat,Saidu Sharif | 132 | 38 | 27 | 30 | 19 | 11 | 7 |
| 4-Baltis.Baltistan | 80 | 24 | 14 | 18 | 14 | 7 | 3 |
| 5-Gilgit.Hunza | 23 | 7 | 3 | 5 | 3 | 2 | 3 |
| 6-Peshawar | 153 | 46 | 18 | 45 | 28 | 5 | 11 |

Table 5.1.3.b*.VIII (Cont.) MNSs blood groups distribution in Pakistan tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Gene complex frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | | |
| 1-Punjabis.Lahore | 202 | 17.71 | 40.51 | 12.67 | 29.11 | Boyd,W.C., & Boyd, Lyle.G. | 1954 |
| 2-Bengalis.Dacca | 230 | 20.09 | 38.00 | 13.02 | 28.89 | Boyd,W.C., & Boyd, Lyle.G. | 1954 |
| 3-Pathans.Swat,Saidu Sharif | 132 | 24.38 | 43.49 | 12.34 | 19.79 | Alciati,G. | 1968 |
| 4-Baltis.Baltistan | 80 | 25.48 | 42.09 | 12.35 | 20.08 | Clegg,E.J.,et al. | 1961 |
| 5-Gilgit.Hunza | 23 | 28.41 | 32.47 | 9.28 | 29.84 | Ikin,Elizabeth W., et al. | 1959 |
| 6-Peshawar | 153 | 32.16 | 33.58 | 6.65 | 27.61 | Boyd,W.C., & Boyd, Lyle.G. | 1954 |

Table 5.1.3.b. IX MNSs blood groups distribution in Afghanistan tested with anti-M, anti-N, and anti-S sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | MMSS | MMss | MNSS | MNss | NNSS | NNss |
| 1-Timuri & related tribes | 118 | 27 | 25 | 20 | 32 | 8 | 6 |

Table 5.1.3.b. IX (Cont). MNSs blood groups distribution in Afghanistan tested with anti-M , anti-N, and anti-S sera

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Timuri & related tribes | 118 | 17.26 | 48.84 | 9.67 | 24.23 | Woodd-Walker,R.B., 1967 et al. |

Table 5.1.3.C. IX MNSs blood groups distribution in Afghanistan tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | MMSS | MMSs | MMss | MNSS | MNSs | MNss | NNSS | NNSs | NNss |
| 1-Daris | 179 | 17 | 37 | 28 | 24 | 29 | 34 | 2 | 13 | 15 |
| 2-Pushtus | 104 | 6 | 21 | 12 | 5 | 28 | 18 | 1 | 3 | 10 |

Table 5.1.3.C. IX (Cont.) MNSs blood groups distribution in Afghanistan tested with anti-M, anti-N, anti-S, and anti-s sera

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Daris | 179 | 25.70 | 38.80 | 9.20 | 26.30 | Papiha,S.S.,et al. 1977 |
| 2-Pushtus | 104 | 29.00 | 33.00 | 7.50 | 30.50 | Papiha,S.S.,et al. 1977 |

Table 5.1.3. * IX MNSs - complex frequencies distribution in Afghanistan

| Population | Number Tested | Gene complex frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | MS | Ms | NS | Ns | |
| 1-Timuri & related tribes | 118 | 17.26 | 48.84 | 9.67 | 24.23 | Woodd-Walker,R.B.,et al.1967 |
| 2-Daris | 179 | 25.70 | 38.80 | 9.20 | 26.30 | Papiha,S.S.,et al. 1977 |
| 3-Pushtus | 104 | 29.00 | 33.00 | 7.50 | 30.50 | Papiha,S.S.,et al. 1977 |

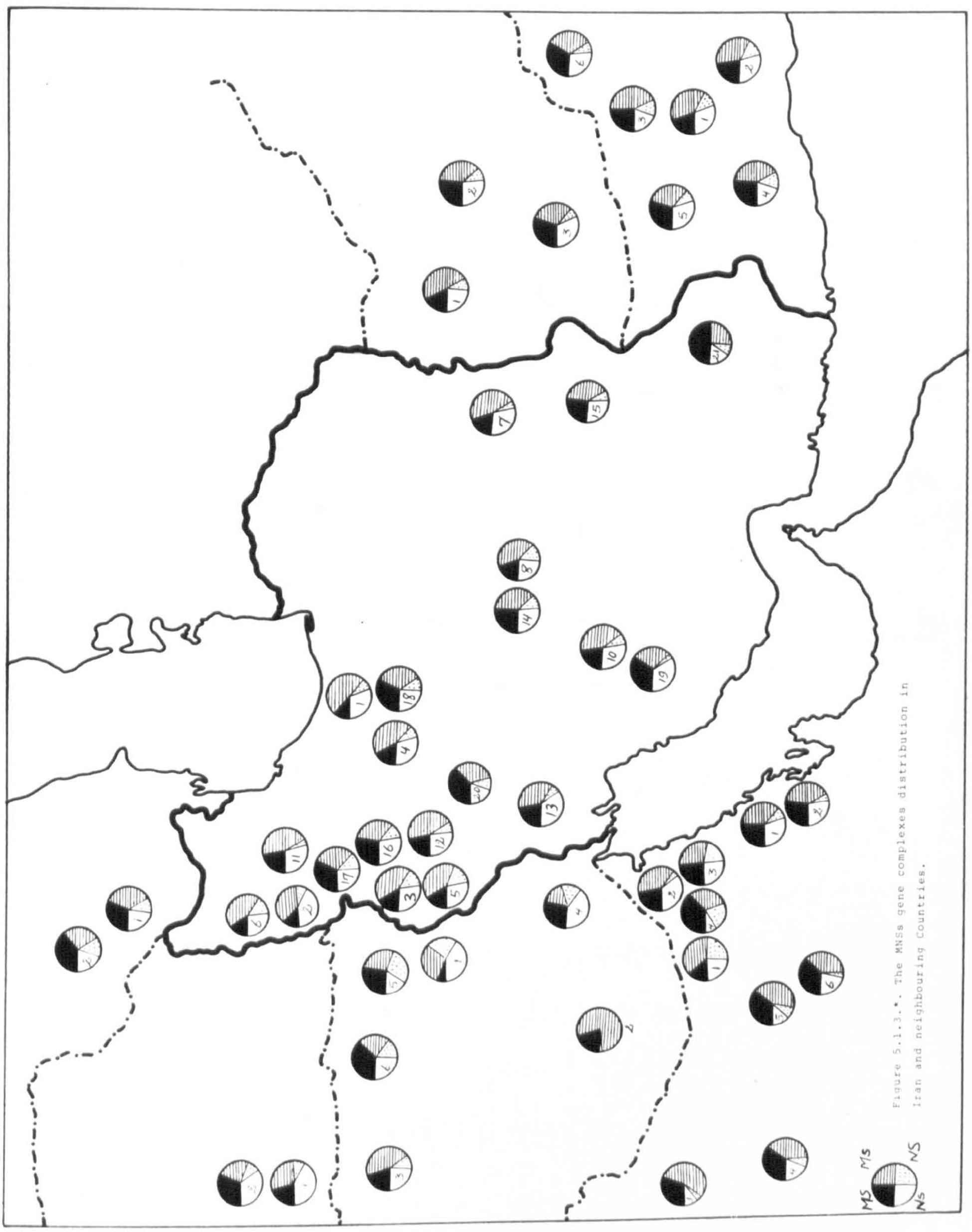



Figure 5.1.3.*. The MNSs gene complexes distribution in Iran and neighbouring Countries.

## 5.1.4. The P blood group system

The distribution of the P blood groups and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.1.4.

The frequency of the $P_1$ gene ranges from 23.27 to 53.22 percent in Iranians, being lowest in the Turks of Rezaieh (present investigation) and highest in the Assyrians( Nijenhuis, 1964). With the exception of the Turks of Rezaieh, values obtained in the present investigation are within the range of variation.

On the whole, with an average $P_1$ frequency of 42.62 percent, the Iranian population appears to exhibit a lower $P_1$ gene frequency than that of over 50 percent found in Europeans (Mourant et al, 1976).

In general, the allele $P_1$ does not show much variability within Iran.

Regarding neighbouring areas, the frequency of the gene $P_1$ in the populations of the Caucasus varies between 46.79 percent in the Svanis of Georgia, Abkhazskaya ( Verbitsky et al, 1971) and 53.15 percent in the Georgians (Kherumian et al, 1954). With an average $P_1$ frequency of 49.97 percent, the population of the Caucasus seems to show a higher $P_1$ frequency than that found in Iranians. The $P_1$ frequency in the Caucasus is similar to that in Europe.

The $P_1$ gene frequency of 35.00 percent in the Kurds of Turkey (Richard, 1976) is much lower than the values ranging from 47.50 to 51.65 percent in the Kurds of western Iran (Nijenhuis, 1964; and Lehmann et al, 1973).

The frequency of the $P_1$ gene in the populations of Iraq

ranges from 21.26 percent in the Kurdish Jews of the south east( Tills et al, 1977) to 41.40 percent in the Assyrians (Ikin et al, 1965).

With an average $P_1$ frequency of 30.34 percent, the Iraqi population appears to exhibit a much lower frequency of the $P_1$ gene than that found in Iranians. The Iraqi Kurds and Kurdish Jews seem to show much lower $P_1$ frequencies than those found in the same two population groups in Iran.

The $P_1$ gene frequency in the Kuwaiti populations varies between 45.00 percent in the Ajman tribe and 61.00 percent in the Suluba tribe (Khaled et al, 1981). With an average $P_1$ frequency of 52.67 percent, the population of Kuwait has a higher $P_1$ frequency than that found in Iranians and is more similar to the European frequency.

The frequency of the $P_1$ gene in the populations of Saudi Arabia ranges from 42.42 percent in the Bedouins to 57.18 percent in the Sunni sample of Najd(Maranjian et al, 1966). With an average $P_1$ frequency of 50.78 percent, the population of Saudi Arabia, like that of Kuwait, appears to exhibit a higher $P_1$ frequency than that found in Iranians and more similar to the European frequency.

The $P_1$ frequency of 46.10 percent in the Abu-Dhabians of the United Arab Emirates (Kamel et al, 1980) is also higher than in Iran but lower than the European frequency.

The frequency of the $P_1$ gene in the populations of Pakistan varies between 35.77 percent in the Baltis(Clegg et al, 1961) and 48.92 percent in the Gilgit, Hunza (Ikin et al, 1959). With an average $P_1$ frequency of 40.52 percent, the Pakistani population seems to show a slightly

lower $P_1$ frequency than that found in Iranians and lower than the European frequency.

The $P_1$ gene frequency in the populations of Afghanistan ranges from 38.93 percent in the Timuri and related tribes (Woodd-Walker et al, 1967) to 50.40 percent in the Daris (Papiha et al, 1977). With an average $P_1$ frequency of 42.98 percent, the Afghan population appears to exhibit a similar $P_1$ frequency to that found in Iranians and lower than the frequency in Europeans.

Conclusion

In the European populations the gene $P_1$ has a frequency of slightly over 50 percent. In Negroes the frequency is much higher, around 80 percent. In India and south east Asia frequencies are mostly similar to those found in Europe but in eastern Asia is considerably lower (Mourant et al,1976).

In the Iranian and neighbouring populations, with the exception of the Arab populations of Kuwait and Saudi Arabia with relatively higher values, the frequency of the $P_1$ gene is below the typical European level, though tending rather towards the low values found in Asia than to the high ones in Africa. The low frequency of $P_1$ is consistent with the suggestion of Mongoloid contribution to the gene pool.

Table 5.1.4. I P blood groups distribution in Iran tested with anti-$P_1$ serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2$+ P | |
| 1-Turks.Rezaieh | 141 | 58 | 83 | 23.27 | 76.73 | Present study. |
| 2-Jews | 108 | 51 | 57 | 27.35 | 72.65 | Tabatabai,H. 1977 |
| 3-Zoroastrians | 86 | 41 | 45 | 27.66 | 72.34 | Present study. |
| 4-Kurds.Rezaieh | 138 | 74 | 64 | 31.90 | 68.10 | Present study. |
| 5-North | 58 | 35 | 23 | 37.02 | 62.98 | Bajatzadeh,M.,& Walter, H. 1969 |
| 6-Tehran | 112 | 71 | 41 | 39.49 | 60.51 | Bajatzadeh,M.,& Walter, H. 1969 |
| 7-Kurdish Jews | 106 | 71 | 35 | 42.54 | 57.46 | Tills,D.,et al. 1977 |
| 8-Ghashghais.Fars | 66 | 45 | 21 | 43.59 | 56.41 | Nijenhuis,L.E. 1964 |
| 9-Armenians | 143 | 98 | 45 | 43.90 | 56.10 | Tabatabai,H. 1977 |
| 10-Bakhtiaris | 137 | 94 | 43 | 43.97 | 56.03 | Nijenhuis,L.E. 1964 |
| 11-Central and South | 106 | 73 | 33 | 44.21 | 55.79 | Bajatzadeh,M.,& Walter, H. 1969 |
| 12-West | 106 | 73 | 33 | 44.21 | 55.79 | Bajatzadeh,M.,& Walter, H. 1969 |

Table 5.1.4.I (Cont.) P blood groups distribution in Iran tested with anti-$P_1$ serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2+P$ | | |
| 13-Kurdish Jews | 94 | 65 | 29 | 44.46 | 55.54 | Godber,Marilyn. J., et al. | 1973 |
| 14-East | 66 | 46 | 20 | 44.95 | 55.05 | Bajatzadeh,M.,& Walter, H. | 1969 |
| 15-North west | 65 | 46 | 19 | 45.94 | 54.06 | Bajatzadeh,M.,& Walter,H. | 1969 |
| 16-Kurds.Kermanshah | 127 | 92 | 35 | 47.50 | 52.50 | Nijenhuis,L.E. | 1964 |
| 17-Turks.Mainly south East | 348 | 256 | 92 | 48.58 | 51.42 | Nijenhuis,L.E. | 1964 |
| 18-Kurds.Sanandaj | 106 | 78 | 28 | 48.60 | 51.40 | Lehmann,H.,et al. | 1973 |
| 19-Armenians | 78 | 59 | 19 | 50.64 | 49.36 | Nijenhuis,L.E. | 1964 |
| 20-Kurds.Baneh,Marivan | 77 | 59 | 18 | 51.65 | 48.35 | Lehmann,H.,et al. | 1973 |
| 21-Arabs.Abadan | 158 | 123 | 35 | 52.94 | 47.06 | Nijenhuis,L.E. | 1964 |
| 22-Assyrians | 32 | 25 | 7 | 53.22 | 46.78 | Nijenhuis,L.E. | 1964 |

Table 5.1.4.II P blood groups distribution in the Caucasus tested with anti-$P_1$ serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2+P$ | | |
| 1-Svanis.Georgia, Abkhazskaya | 650 | 466 | 184 | 46.79 | 53.21 | Verbitsky,M.Sh., et al. | 1971 |
| 2-Georgians | 41 | 32 | 9 | 53.15 | 46.85 | Kherumian,R.,et al. | 1954 |

Table 5.1.4. III P blood groups distribution in Turkey tested with anti-$P_1$ serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2+P$ | |
| 1-Kurds | | | | 35.00 | 65.00 | Richard, P. 1976 |

Table 5.1.4. IV P blood groups distribution in Iraq tested with anti-$P_1$ serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2+P$ | |
| 1-Kurdish Jews.south east | 50 | 19 | 31 | 21.26 | 78.74 | Tills,D.,et al. 1977 |
| 2-Kurds | | | | 27.00 | 73.00 | Al-Khafaji,S.D.,et al. 1976 |
| 3-Kurdish Jews. North west | 61 | 30 | 31 | 28.71 | 71.29 | Tills,D.,et al. 1977 |
| 4-Kurdish Jews | 27 | 15 | 12 | 33.34 | 66.66 | Godber,Marilyn J.,et al.1973 |
| 5-Assyrians | 99 | 65 | 34 | 41.40 | 58.60 | Ikin,Elizabeth W.,et al.1965 |

Table 5.1.4.V P blood groups distribution in Kuwait tested with anti-$P_1$ serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2$+P | |
| 1-Ajman tribe | 52 | 36 | 16 | 45.00 | 55.00 | Khaled,E.,et al.1981 |
| 2-General population | 74 | 57 | 17 | 52.00 | 48.00 | Khaled,E.,et al.1981 |
| 3-Suluba tribe | 52 | 44 | 8 | 61.00 | 39.00 | Khaled,E.,et al.1981 |

Table 5.1.4. VI P blood groups distribution in Saudi Arabia tested with anti-$P_1$ serum

| Population | Number Tested | Phenotypes | | Gene fruequencies | | Authors |
|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2$+P | |
| 1-Bedouins | 178 | 119 | 59 | 42.42 | 57.58 | Maranjian,G.,et al. 1966 |
| 2-Shias.Qatif & Hasa Oases | 465 | 320 | 145 | 44.16 | 55.84 | Maranjian,G.,et al. 1966 |
| 3-Sunnis.Qatif & Hasa Oases | 323 | 253 | 70 | 53.45 | 46.55 | Maranjian,G.,et al. 1966 |
| 4-Sunnis.Asir,Hejaz, Najran | 176 | 143 | 33 | 56.70 | 43.30 | Maranjian,G.,et al. 1966 |
| 5-Sunnis. Najd | 180 | 147 | 33 | 57.18 | 42.82 | Maranjian,G.,et al. 1966 |

Table 5.1.4. VII P blood groups distribution in the United Arab Emirates tested with anti- $P_1$ serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2+P$ | |
| 1-Abu-Dhabians | 100 | 71 | 29 | 46.10 | 53.90 | Kamel,K.,et al. 1980 |

Table 5.1.4. VIII

P blood groups distribution in Pakistan tested with anti-$P_1$ serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2+P$ | |
| 1- Baltis.Baltistan | 80 | 47 | 33 | 35.77 | 64.23 | Clegg,E.J.,et al. 1961 |
| 2-Pathans.Swat,Saidu Sharif | 133 | 80 | 53 | 36.87 | 63.13 | Alciati,G. 1968 |
| 3-Gilgit,Hunza | 23 | 17 | 6 | 48.92 | 51.08 | Ikin,Elizabeth W., et al. 1959 |

Table 5.1.4. IX P blood groups distribution in Afghanistan tested with anti- $P_1$ serum

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | $P_1$ | $P_2$ | $P_1$ | $P_2+P$ | | |
| 1-Timuri and related tribes | 118 | 74 | 44 | 38.93 | 61.07 | Woodd-Walker,R.B.,et al. | 1967 |
| 2-Pushtus | 104 | 66 | 38 | 39.60 | 60.40 | Papiha,S.S.,et al. | 1977 |
| 3-Daris | 179 | 135 | 44 | 50.40 | 49.60 | Papiha,S.S.,et al. | 1977 |

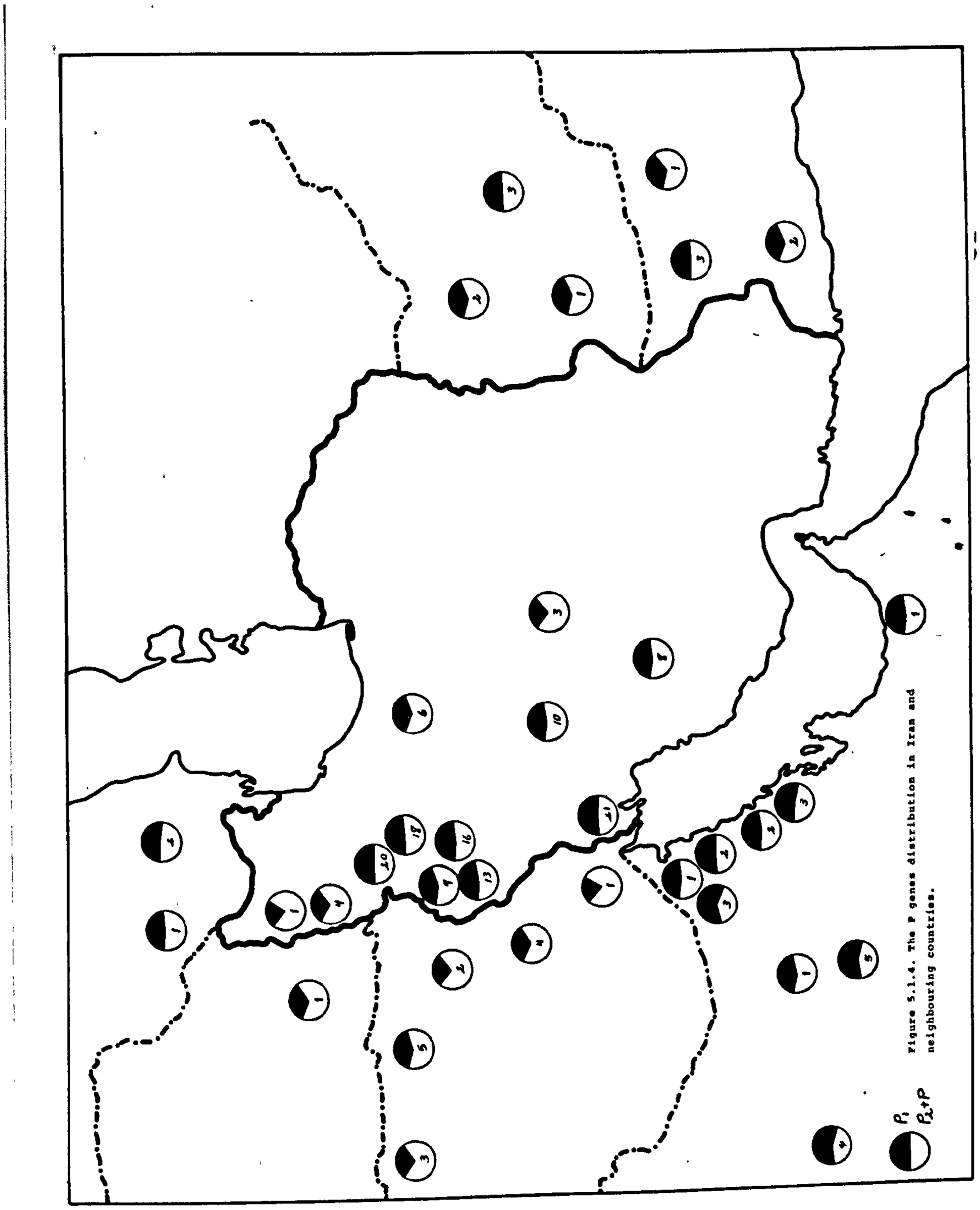



Figure 5.1.4. The P genes distribution in Iran and neighbouring countries.

## 5.1.5. The Kell blood group system

The distribution of the Kell blood groups and respective gene frequencies in Iranian and neighbouring populations, expresssed in terms of 2, 3 and 6 phenotypes after testing with 1, 2 and 3 antisera respectively, is set out in Tables 5.1.5. a, b and c.

Data summarized in the tables (5.1.5.a) show that the frequency of the gene K varies between zero and 5.67 percent in Iranians, being lowest in the Assyrians and highest in the Kurds of Kermanshah (Nijenhnis, 1964). Values obtained in the present investigation are within this range of variation.

On the whole, with an average K gene frequency of 2.46 percent, the Iranian population appears to exhibit a K frequency similar to that varying between 1.54 and 5.82 percent in the European populations( Mourant et al, 1976).

From the table, some differences in the distribution of the K gene frequencies seem to exist in Iran, as the K values ranging from 0.46 to 3.20 percent in eastern Iran (average 1.43 percent) appear to be lower than those varying between 0.56 and 5.67 percent (average 2.55 percent ) in western Iran.

The Arabs of south western Iran, with a K gene frequency of 3.66 percent which is near the average for Iran, show no resemblance to the Arabs of Arabia with their much higher K frequency of above 6 percent.

The Zoroastrians with a K gene frequency of 0.58 percent, which is one of the lowest frequencies within Iran, differ strongly from the Shia from Yazd in the same region, with a much higher K gene frequency of 4.69 percent.

Regarding neighbouring groups , the frequency of the K gene in the populations of the Caucasus ranges from 2.53 percent in the Georgians(Kherumian et al, 1954) to 2.88 percent in the Svanis of Georgia, Abkhazskaya ( Verbitsky et al 1972). With an average K frequency of 2.70 percent, the population of the Caucasus appears to exhibit a K gene grequency similar to that found in Iranians and comparable with the European frequency.

The frequency of the K gene in the Turkish populations varies between 1.20 percent in the Kurds (Richard, 1976) and 5.31 percent in the Eti-Turks (Aksoy et al, 1958). With an average K gene frequency of 2.80 percent, the population of Turkey also seems to show a K gene frequency comparable with that found in Iranians and similar to the European frequency.

The frequency of the gene K in the populations of Iraq ranges from zero in the Kurdish Jews (Godber et al, 1973) to 9.48 percent in the Karaite Jews (Goldschmidt et al, 1976 ). With an average K gene frequency of 3.1 percent, the Iraqi population appears to exhibit a K gene frequency similar to that found in Iranians and comparable with the frequency in Europe.The very high K gene frequency of 9.48 percent in the Karaite Jews is remarkable and clearly indicates the strong influence of isolation and genetic drift operating in this community.

The freqeuncy of the K gene in the Kuwaiti populations varies between 1.87 percent in the Kuwaiti Arabs( Sawhney, 1975) and 12.00 percent in the Ajman tribe (Khaled et al, 1981). With an average K gene frequency of 6.47 percent, the population of Kuwait seems to show a much higher K frequency than that found in Iranians and in Europeans.

The K gene frequency in the Saudi Arabian populations ranges from 3.17 percent in the Shia sample of Qatif and Hasa Oases to 10.56 percent in the Bedouins (Maranjian et al, 1966), With an average K gene frequency of 6.38 percent, the population of Saudi Arabia, like that of Kuwait, appears to exhibit a much higher K frequency than that found in Iranians and in Europeans.

The K gene frequency of 5.00 percent in the Abu-Dhabians of the United Arab Emirates (Kamel et al, 1980), though lower than the frequencies in the Arab populations of Kuwait and Saudi Arabia, is still higher than the Iranian and the European frequencies.

The few studies on the inhabitants of the Arabian Peninsula have already shown that the K gene of the Kell system reaches its highest frequency there. The average K frequency of around 6 percent in the Arabs of Kuwait and Saudi Arabia seems to be suggestive of African admixture.

The frequency of the K gene in the populations of Pakistan varies between zero in the Baltis (Clegg et al, 1961) and 8.40 percent in the Punjabis (Boyd and Boyd, 1954). With an average K gene frequency of 4.15 percent, the Pakistani population seems to show a higher K frequency than that found in Iranians and higher than the European frequency.

The K gene frequency in the Afghan populations ranges from 1.40 percent in the Pushtus to 2.00 percent in the Daris(Papiha et al, 1977). With an average K gene frequency of 1.70 percent, the population of Afghanistan appears to exhibit a lower K frequency than that in Iranians and other neighbouring populations and in almost all Europeans.

In north west India, and in some Himalayan populations there

appears to be a zone of low K frequency, into which the Afghan frequency appears to fit.

Conclusion

The frequency of the K gene is relatively uniform throughout Europe, ranging usually between 1.54 and 5.82 percent (Mourant et al, 1976).

In general, the Kell blood groups do not vary much on a geographical basis. The K gene frequencies in Iranians are in line with studies in neighbouring populations, showing K gene frequencies similar to those found in Europeans. The only exception is among the Arabs of Arabia in whom the highest frequencies of the K gene, exceeding 12 percent, are found. The very high frequencies of the K gene in the Arab populations appear to be suggestive of African admixture.

Table 5.1.5.a. I Kell blood groups distribution in Iran tested with anti-K serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | K(+) | K(-) | K | k | | |
| 1-Assyrians | 27 | - | 27 | 0.00 | 100.00 | Nijenhuis,L.E. | 1964 |
| 2-Baluchis.Sistan & Baluchistan | 110 | 1 | 109 | 0.46 | 99.54 | Present study. | |
| 3-Zoroastrians | 86 | 1 | 85 | 0.58 | 99.42 | Present study. | |
| 4-Kurds.Baneh,Marivan | 77 | 1 | 76 | 0.65 | 99.35 | Lehmann,H.,et al. | 1973 |
| 5-East | 66 | 1 | 65 | 0.76 | 99.24 | Bajatzadeh,M., & Walter,H. | 1969 |
| 6-West | 103 | 2 | 101 | 0.97 | 99.03 | Bajatzadeh,M., & Walter, H. | 1969 |
| 7-Kurds.Rezaieh | 138 | 3 | 135 | 1.09 | 98.91 | Present study. | |
| 8-Zabolis.Sistan & Baluchistan | 115 | 3 | 112 | 1.31 | 98.69 | Present study. | |
| 9-Kurds.Sanandaj | 107 | 3 | 104 | 1.40 | 98.60 | Lehmann,H.,et al. | 1973 |
| 10-Armenians | 67 | 2 | 65 | 1.51 | 98.49 | Nijenhuis,L.E. | 1964 |
| 11-Tehran | 195 | 6 | 189 | 1.55 | 98.45 | Sawhney, K.S. | 1975 |
| 12-Kurdish Jews | 94 | 3 | 91 | 1.60 | 98.40 | Godber,Marilyn J.,et al. | 1973 |

Table 5.1.5.a. I(Cont.) Kell blood groups distribution in Iran tested with anti-K serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | K(+) | K(-) | K | k | | |
| 13- Bakhtiaris | 138 | 6 | 132 | 2.20 | 97.80 | Nijenhuis,L.E. | 1964 |
| 14-Kurdish Jews | 106 | 5 | 101 | 2.36 | 97.64 | Tills,D., et al. | 1977 |
| 15-Turks.Rezaieh | 141 | 7 | 134 | 2.51 | 97.49 | Present study. | |
| 16-Ghashghais | 66 | 4 | 62 | 3.08 | 96.92 | Nijenhuis,L.E. | 1964 |
| 17-Esfahan | 112 | 7 | 105 | 3.18 | 96.82 | Sawhney,K.S. | 1975 |
| 18-South-East | 302 | 19 | 283 | 3.20 | 96.80 | Nijenhuis,L.E. | 1964 |
| 19-Lurs.Luristan | 149 | 10 | 139 | 3.41 | 96.59 | Present study. | |
| 20-North | 58 | 4 | 54 | 3.51 | 96.49 | Bajatzadeh,M., & Walter, H. | 1969 |
| 21-Arabs.Abadan | 153 | 11 | 142 | 3.66 | 96.34 | Nijenhuis,L.E. | 1964 |
| 22-Central and South | 106 | 9 | 97 | 4.34 | 95.66 | Bajatzadeh,M., & Walter, H. | 1969 |
| 23-Tehran | 110 | 10 | 100 | 4.65 | 95.35 | Bajatzadeh,M.,& Walter, H. | 1969 |
| 24-Shi'a.Yazd | 142 | 13 | 129 | 4.69 | 95.31 | Sunderland,E., & Smith,H.M. | 1966 |

Table 5.1.5.a.I (Cont.) Kell blood groups distribution in Iran tested with anti-K serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | K(+) | K(-) | K | k | | |
| 25-North west | 64 | 7 | 57 | 5.63 | 94.37 | Bajatzadeh,M.,& Walter, H. | 1969 |
| 26-Kurds.Kermanshah | 127 | 14 | 113 | 5.67 | 94.33 | Nijenhuis, L.E. | 1964 |

Table . 5.1.5.C. I

Kell blood groups distribution in Iran tested with anti-K, anti-k, and anti-$Js^a$ sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | KKJs(a+) | KKJs(a-) | KkJs(a+) | KkJs(a-) | kkJs(a+) | kkJs(a-) |
| 1-Kurds.Baneh, Marivan | 77 | - | - | - | 1 | - | 76 |
| 2-Kurds.Sanandaj | 107 | - | - | - | 3 | - | 104 |
| 3-Kurdish Jews | 94 | - | - | - | 3 | - | 91 |

Table 5.1.5.C.I (Cont.) Kell blood groups distribution in Iran tested with anti-K, anti-k, and anti-Js$^{a}$ sera

| Population | Number Tested | Gene frequencies | | | Authors |
|---|---|---|---|---|---|
| | | KJs$^{b}$ | kJs$^{a}$ | kJs$^{b}$ | |
| 1-Kurds.Baneh, Marivan | 77 | 0.65 | 0.00 | 99.35 | Lehmann,H.,et al. 1973 |
| 2-Kurds.Sanandaj | 107 | 1.40 | 0.00 | 98.60 | Lehmann,H.,et al. 1973 |
| 3-Kurdish Jews | 94 | 1.60 | 0.00 | 98.40 | Godber,Marilyn J., et al. 1973 |

Table 5.1.5. d I. Kell blood groups distribution in Iran tested with anti-$Js^a$( Sutter) serum

| Population | Number Tested | Js(a+) | Js(a-) | Authors | |
|---|---|---|---|---|---|
| Kurdish Jews | 87 | - | 87 | Tills,D.,et al. | 1977 |

Table 5.1.5.a.II Kell blood groups distribution in the Caucasus tested with anti-K serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | K(+) | K(-) | K | k | |
| 1-Georgians.Autochthonous | 40 | 2 | 38 | 2.53 | 97.47 | Kherumian,R.,et al. 1954 |
| 2-Svanis.Georgia,Abkhazskaya | 659 | 38 | 621 | 2.88 | 97.12 | Verbitsky,M.Sh.,et al. 1972 |

Table 5.1.5.b. II Kell blood groups distribution in the Caucasus tested with anti-K and anti-k sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | KK | Kk | kk | K | k | |
| 1-Svanis.. Georgia,Abkhazskaya | 659 | - | 38 | 621 | 2.88 | 97.12 | Verbitsky,M.Sh., et al. 1972 |

Table 5.1.5.a. III Kell blood groups distribution in Turkey tested with anti-K serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | K(+) | K(-) | K | k | | |
| I- Kurds | | | | 1.20 | 98.80 | Richard, P. | 1976 |
| 2-Turks.Asia Minor,Mersin | 107 | 4 | 103 | 1.89 | 98.11 | Aksoy,M., et al. | 1958 |
| 3-Eti-Turks. Asia Minor. ,near Mersin | 116 | 12 | 104 | 5.31 | 94.69 | Aksoy,M., et al. | 1958 |

Table 5.1.5. a. IV Kell blood groups distribution in Iraq tested with anti- K serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | K(+) | K(-) | K | k | |
| 1- Kurdish Jews | 27 | - | 27 | 0.00 | 100.00 | Godber,Marilyn.J., et al. 1973 |
| 2-Kurdish Jews.South east | 50 | 1 | 49 | 1.00 | 99.00 | Tills,D., et al. 1977 |
| 3-Assyrians | 99 | 3 | 96 | 1.53 | 98.47 | Ikin,Elizabeth W., et al. 1965 |
| 4-Kurdish Jews.North west | 61 | 4 | 57 | 3.28 | 96.72 | Tills,D., et al. 1977 |
| 5-Kurds | | | | 3.30 | 96.70 | Al-Khafaji,S.D., et al. 1976 |
| 6-Karaite Jews | 72 | 13 | 59 | 9.48 | 90.52 | Goldschmidt,E., et al. 1976 |

Table 5.1.5.b. IV Kell blood groups distribution in Iraq tested with anti-K and anti-k sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | KK | Kk | kk | K | k | |
| 1-Kurdish Jews | 27 | - | - | 27 | 0.00 | 100.00 | Godber,Marilyn.J., et al. 1973 |
| 2-Kurdish Jews.South east | 50 | - | 1 | 49 | 1.00 | 99.00 | Tills,D., et al. 1977 |
| 3-Kurdish Jews.North west | 61 | - | 4 | 57 | 3.28 | 96.72 | Tills,D., et al. 1977 |
| 4-Kurds | | | | | 3.30 | 96.70 | Al-Khafaji,S.D.,et al. 1976 |

Table 5.1.5.C. IV Kell blood groups distribution in Iraq tested with anti-K, anti-k, and anti-Js$^{a}$ sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | KKJs(a+) | KKJs(a-) | KkJs(a+) | KkJs(a-) | kkJs(a+) | kkJs(a-) |
| 1- Kurdish Jews | 27 | - | - | - | - | - | 27 |

Table 5.1.5.C. IV (Cont.) Kell blood groups distribution in Iraq tested with anti-K, anti-k, and anti-Js$^{a}$ sera

| Population | Number Tested | Gene frequencies | | | Authors |
|---|---|---|---|---|---|
| | | KJs$^{b}$ | KJs$^{a}$ | kJs$^{b}$ | |
| 1-Kurdish Jews | 27 | 0.00 | 0.00 | 100.00 | Godber,Marilyn J., 1973 et al. |

Table 5.1.5. d IV Kell blood groups distribution in Iraq tested with anti-Js$^{a}$(Sutter) serum

| Population | Number Tested | Js(a+) | Js(a-) | Authors |
|---|---|---|---|---|
| Kurdish Jews. North west | 20 | - | 20 | Tills, D., et al. 1977 |
| Kurdish Jews. South East | 9 | - | 9 | Tills, D., et al. 1977 |

Table 5.1.5.a. V  Kell blood groups distribution in Kuwait tested with anti-K serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | K(+) | k(-) | K | k | | |
| 1-Kuwaiti Arabs | 162 | 6 | 156 | 1.87 | 98.13 | Sawhney, K.S. | 1975 |
| 2-General population | 74 | 8 | 66 | 5.00 | 95.00 | Khaled,E.,et al. | 1981 |
| 3-Suluba tribe | 51 | 6 | 45 | 7.00 | 93.00 | Khaled,E.,et al. | 1981 |
| 4-Ajman tribe | 52 | 12 | 40 | 12.00 | 88.00 | Khaled,E.,et al. | 1981 |

Table 5.1.5.b. V Kell blood groups distribution in Kuwait tested with anti-K and anti-k sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | KK | Kk | kk | K | k | | |
| 1-General population | 74 | - | 8 | 66 | 5.00 | .95.00 | Khaled,E.,et al. | 1981 |
| 2-Suluba tribe | 51 | 1 | 5 | 45 | 7.00 | 93.00 | Khaled,E.,et al. | 1981 |
| 3-Ajman tribe | 52 | - | 12 | 40 | 12.00 | 88.00 | Khaled,E.,et al. | 1981 |

Table 5.1.5.a. VI Kell blood groups distribution in Saudi Arabia tested with anti-K serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | K(+) | K(-) | K | k | | |
| 1-Shias.Qatif & Hasa Oases | 465 | 29 | 436 | 3.17 | 96.83 | Maranjian,G.,et al. | 1966 |
| 2-Sunnis.Najd | 180 | 35 | 145 | 4.84 | 95.16 | Maranjian,G.,et al. | 1966 |
| 3-Sunnis.Qatif &Hasa Oases | 318 | 36 | 282 | 5.83 | 94.17 | Maranjian, G.,et al. | 1966 |
| 4-Western Saudi Arabia | 205 | 23 | 182 | 6.10 | 93.90 | Saha, N., et al. | 1980 |
| 5-Sunnis.Asir,Hejaz,Najran | 174 | 26 | 148 | 7.77 | 92.23 | Maranjian,G.,et al. | 1966 |
| 6-Bedouins | 175 | 35 | 140 | 10.56 | 89.44 | Maranjian,G.,et al. | 1966 |

Table 5.1.5.b. VI Kell blood groups distribution in Saudi Arabia tested with anti-K and anti-k sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | KK | Kk | kk | K | k | |
| 1- Western Saudi Arabia | 205 | 2 | 21 | 182 | 6.10 | 93.90 | Saha,N., et al. 1980 |

Table 5.1.5.b. VII Kell blood groups distribution in the United Arab Emirates tested with anti-K and anti-k sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | KK | Kk | kk | K | k | |
| 1-Abu-Dhabians | 100 | 2 | 6 | 92 | 5.00 | 95.00 | Kamel,K.,et al. 1980 |

Table 5.1.5.C. VII Kell blood groups distribution in the United Arab Emirates tested with anti-K, anti-k, and anti-Js$^{a}$ sera

| Population | Number Tested | Phenotypes | | | | | |
|---|---|---|---|---|---|---|---|
| | | KKJs(a+) | KKJs(a- ) | KkJs(a+) | KkJs(a- ) | kkJs(a+) | kkJs(a-) |
| 1-Abu-Dhabians | 100 | - | 2 | 1 | 5 | 2 | 90 |

Table 5.1.5.C.VII (Cont.) Kell blood groups distribution in the United Arab Emirates tested with anti-K, anti-k, and anti-Js$^{a}$ sera

| Population | Number Tested | Gene frequencies | | | Authors |
|---|---|---|---|---|---|
| | | KJs$^{b}$ | kJs$^{a}$ | kJs$^{b}$ | |
| 1-Abu-Dhabians | 100 | 5.00 | 1.50 | 93.50 | Kamel,K.,et al. 1980 |

Table 5.1.5.a. VIII Kell blood groups distribution in Pakistan tested with anti-K serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | K(+) | K(- ) | K | k | | |
| 1-Baltis.Baltistan | 80 | - | 80 | 0.00 | 100.00 | Clegg,E.J.,et al. | 1961 |
| 2-Pathans.Swat,Saidu Sharif | 133 | 10 | 123 | 3.76 | 96.24 | Alciati, G. | 1968 |
| 3-Gilgit,Hunza | 23 | 2 | 21 | 4.45 | 95.55 | Ikin,Elizabeth W.,et al. | 1959 |
| 4-Punjabis.Lahore | 87 | 14 | 73 | 8.40 | 91.60 | Boyd,W.C., & Boyd,L.G. | 1954 |

Table 5.1.5.b. VIII Kell blood groups distribution in Pakistan tested with anti-K and anti-k sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | KK | Kk | kk | K | k | | |
| 1-Pathans.Swat,Saidu Sharif | 133 | - | 10 | 123 | 3.76 | 96.24 | Alciati, G. | 1968 |

Table 5.1.5. d VIII. Kell blood groups distribution in Pakistan tested with anti-Js$^{a}$(Sutter)serum

| Population | Number Tested | Js(a+) | Js(a-) | Authors |
|---|---|---|---|---|
| Pathans | 139 | - | 139 | Vos,G.H.,and Kirk,R.L. 1961 |
| Punjabis | 168 | - | 168 | Vos,G.H.,and Kirk,R.L. 1961 |

Table 5.1.5.b. IX Kell blood groups distribution in Afghanistan tested with anti-K and anti-k sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | KK | Kk | kk | K | k | |
| 1-Pushtus | 104 | - | 3 | 101 | 1.40 | 98.60 | Papiha,S.S.,et al. 1977 |
| 2-Timuri and related tribes | 118 | 1 | 2 | 115 | 1.69 | 98.31 | Woodd-Walker,R.B.,et al.1967 |
| 3-Daris | 179 | 1 | 5 | 173 | 2.00 | 98.00 | Papiha,S.S., et al. 1977 |

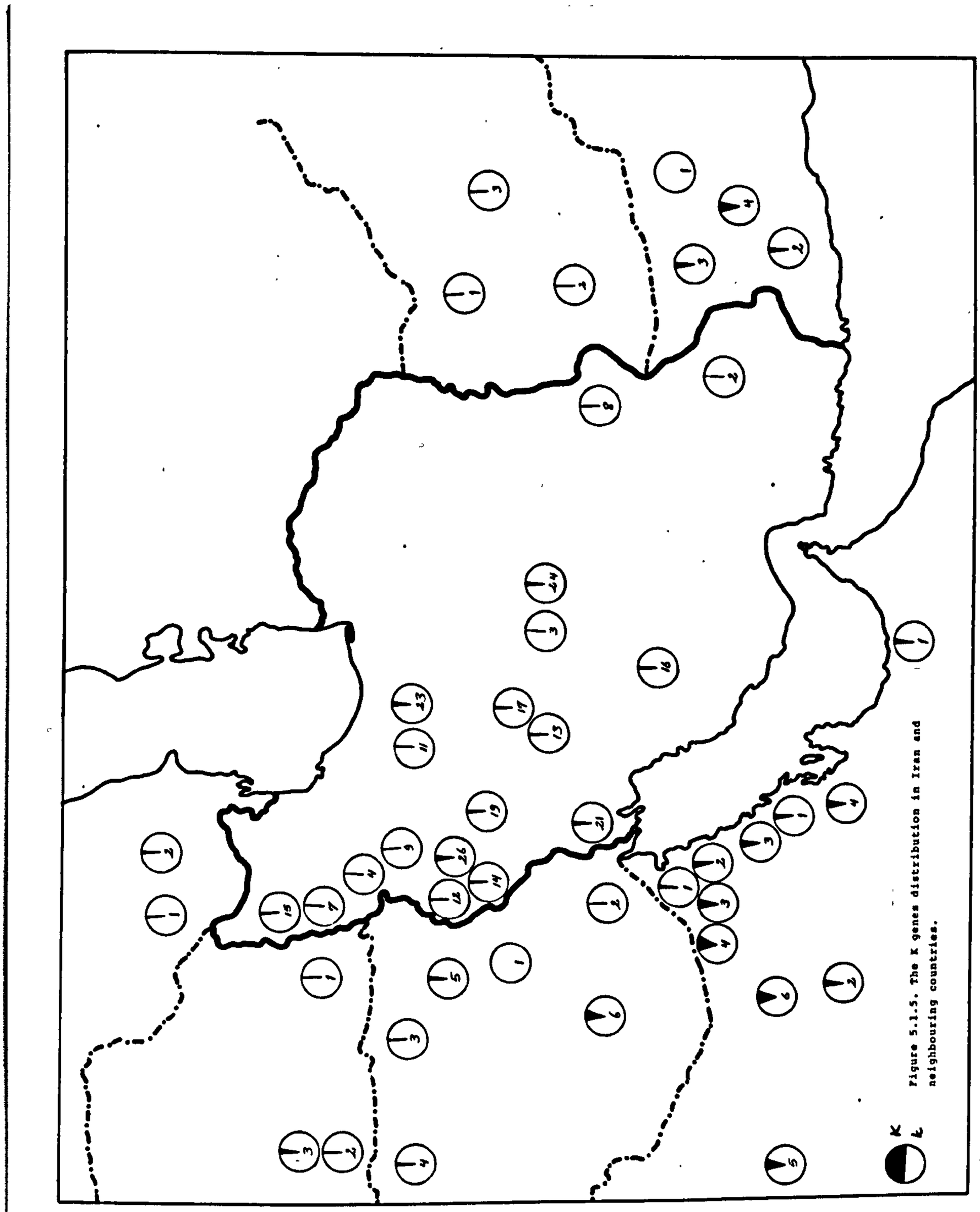


Figure 5.1.5. The K genes distribution in Iran and neighbouring countries.

## 5.1.6.The Duffy blood group system

The distribution of the Duffy blood groups and respective gene frequencies in Iranian and neighbouring populations, expressed in terms of 2 and 4 phenotypes after testing with 1 and 2 antisera, resepctively, is shown in Tables 5.1.6 a and b.

The frequency of the $Fy^a$ gene ranges from 24.97 to 57.83 percent in Iranians, being lowest in the Shi'a Moslems of Yazd ( Sunderland and Smith, 1966) and highest in the Tehrani series of Boue and Boue (1956). Values obtained in the present investigation are within this range of variation.

On the whole with an average $Fy^a$ frequency of 43.58 percent, the Iranian population appears to exhibit a higher $Fy^a$ frequency than that of around 40 percent found in Europeans(Mourant et al, 1976). Some differences in the distribution of the $Fy^a$ gene frequencies seem to exist in Iran, as the $Fy^a$ values ranging from 35.24 to 53.42 percent (average 46.47 percent) in western Iran appear to be higher than those varying between 36.42 and 43.02 percent (average 39.67 percent ) in eastern Iran.

The Kurdish Jews both in Iran and in Iraq appear to exhibit higher $Fy^a$frequencies, averaging 51.79 and 42.04 percent respectively, compared with those averaging 45.22 and 20 percent respectively found in the Kurds themselves.

The Arabs of south western Iran though seem to show one of the lowest $Fy^a$ frequencies within Iran but the frequency is still much higher than the very low ones found in the Arab populations of Arabia.

The $Fy^a$ frequency of 37.12 percent in the Zoroastrians (present investigation ) is also one of the lowest frequencies

within Iran and differs strongly from that of 24.97 percent in the Shi'a Moslems of Yazd ( Sunderland and Smith, 1966), the region that the Zoroastrians come from. It should be pointed out here that the extremely low $Fy^a$ frequency of 24.97 percent in the Yazdi Shi'a series of Sunderland and Smith (1966) may possibly be due to technical difficulties as was mentioned by the authors themselves.

Regarding neighbouring populations, with an $Fy^a$ frequency of 48.76 percent in the Svanis of Georgia, Abkhazskaya (Verbitsky et al, 1972), the population of the Caucasus appears to exhibit a higher $Fy^a$ frequency than that found in Iranians and higher than the European frequency.

The frequency of the $Fy^a$ gene in the populations of Turkey ranges from 42.76 percent in the Eti-Turks to 48.84 percent in the Turks (Aksoy et al, 1958). With an average $Fy^a$ frequency of 45.80 percent, the Turkish population seems to show a somewhat higher $Fy^a$ frequency than that found in Iranians and higher than the frequency in Europeans.

The $Fy^a$ gene frequency in the populations of Iraq varies between the limits of 20.00 percent in the Kurds (Al-Khafaji et al, 1976) and 75.38 percent in the Assyrians (Ikin et al, 1965).

With an average $Fy^a$ frequency of 45.48 percent, the Iraqi population appears to exhibit a slightly higher $Fy^a$ frequency than that found in Iranians and higher than the European frequency.

The very high $Fy^a$ gene frequency of 75.38 percent in the Assyrians of Iraq is more like that found in India and further east than that usual in the Near East.

The frequency of the $Fy^a$ gene in the Arab populations of Kuwait ranges from 23.00 percent in the Ajman tribe to 34.00 percent in the Suluba tribe (Khaled et al, 1981). With an average $Fy^a$ frequency of 28.45 percent, the Kuwaiti population seems to show a much lower $Fy^a$ frequency than that found in Iranians and much lower than the European frequency.

The $Fy^a$ gene frequency in the Arab populations of Saudi Arabia varies between 4.51 percent in the Shia sample of Qatif and Hasa Oases and 32.50 percent in the Sunni series of Najd (Maranjian et al, 1966). With an average $Fy^a$ frequency of 17.88 percent, the Arab population of Saudi Arabia, like that of Kuwait, appears to exhibit a much lower $Fy^a$ frequency than that found in Iranians and much lower than the frequency in Europeans.

A similarly low $Fy^a$ frequency of 18.70 percent is reported by Kamel et al (1980) in the Abu-Dhabians of the United Arab Emirates.

Low $Fy^a$ gene frequencies seems to be characteristic of Arab populations and is suggestive of African admixture.

The frequency of the $Fy^a$ gene in the populations of Pakistan ranges from 44.84 percent in the Hunza, Gilgit(Ikin et al, 1959) to 73.04 percent in the Pakistani series of Cutbush et al (1950). With an average $Fy^a$ frequency of 57.68 percent, the Pakistani population seems to show a much higher $Fy^a$ frequency than that found in Iranians and much higher than the European frequency. The frequency of the $Fy^a$ gene in the population of Pakistan appears to be more like that found in Indians.

The $Fy^a$ gene frequency in the populations of Afghanistan varies between the limits of 53.90 percent in the Pushtus and

59.80 percent in the Daris ( Papiha et al, 1977). With an average $Fy^a$ frequency of 57.17 percent, the Afghan population,like the population of Pakistan,appears to exhibit a much higher $Fy^a$ frequency than that found in Iranians and much higher than the frequency in Europeans.

With regard to the null allele ($Fy^-$) of the Duffy system ( Tables 5.1.6.b), since the majority of the samples were tested with anti-$Fy^a$serum only, it was impossible to detect the presence of the silent Fy gene.

The frequency of the Fy null allele in the Kurdish populations of Iran, both Jewish and non-Jewish, ranges from 13.47 percent in the Kurds of Sanandaj to 16.75 percent in the Kurds of Baneh, Marivan ( Lehmann et al, 1973). With an average Fy null frequency of 15.01 percent, the Kurdish population of Iran appears to exhibit a much higher Fy null frequency than that of below 3 percent found in Europeans (Mourant et al, 1976).

The Fy null allele frequency in the Iraqi Kurdish Jews varies between 21.36 percent (Tills et al, 1977) and 22.11 percent (Godber et al, 1973). With an average Fy null frequency of 21.73 percent, the Iraqi Kurdish Jews seem to exhibit even a higher Fy null frequency than that found in Iranians. These findings suggest that the Fy null allele, usually regarded as of African origin, extends as an indigenous feature into south west Asia.

Although in the Kurdish Jews the African marker genes are mostly absent or present in only a low frequency, the Fy null allele persists at higher levels than the other markers.

Evidence for the presence of African genes in the Arabian pool is suggested from the widespread distribution of the

Duffy null allele. Among the three Kuwaiti groups investigated by Khaled et al (1981), the Suluba sample has the lowest Fy null frequency (24.00 percent) and the Ajman, the highest (38.00 percent).

African admixture can also be expressed by the presence of the high Fy null frequency of 68.00 percent in the Abu-Dhabians of the United Arab Emirates (Kamel et al, 1980).

The Fy null allele was not present in the Pathans of west Pakistan investigated by Alciati (1968).

The frequency of the Fy null allele in the Afghan populations ranges from 2.00 percent in the Daris to 9.70 percent in the Pushtus (Papiha et al, 1977). With an average Fy null frequency of 5.85 percent the population of Afghanistan appears to exhibit a lower Fy null frequency than that found in Iranians but higher than the European frequency.

The presence of the Fy null allele in Kurdish Jews and Kurds has already been mentioned as an African marker.It is however present in many populations of the Near East at frequencies implying a higher proportion of African admixture than that indicated by other markers. This may in part be due to the fact that its detection depends upon negative results in tests with both anit-$Fy^a$ and anti-$Fy^b$, both of which tend to be weak reagents which may sometimes give false negative results. Now that a positive reagent, anti-$Fy^4$, has been discovered, it is important that it should be used to establish the distribution of the gene in a positive manner. However, following the findings of Miller et al (1975) that homozygotes for this gene are probably protected against malaria due to Plasmodium Vivax, the distribution of the gene needs further study in an epidemiological context, and in

comparison with that of other genes having a protective effect against malaria.

Conclusion

The frequency of the $Fy^a$ gene in most populations of European origin is around 40 percent. In India and eastern Asia it is considerably higher.

The frequency of $Fy^a$ is only about 5 percent in American Negroes, and very low indeed in African Negroes. Fy null may have a frequency as high as 3 percent among Europeans. The few African populations which have been tested show very low frequencies of both $Fy^a$ and $Fy^b$, with an Fy null frequency of around 90 percent (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arabs of Arabia with their much lower frequencies, the frequency of the $Fy^a$ gene appears to be higher than that in Europeans but lower than that in Indians.

The low $Fy^a$ gene frequencies together with the high values of Fy null seems to be characteristic of the Arab populations and suggestive of African admixture.

The frequency of the $Fy^a$ gene in the populations of Pakistan and Afghanistan appears to be much higher than that in Iranians and more like that found in Indians.

Until recently, owing to the great rarity of the anti-$Fy^b$ reagent, very few populations had been tested with both anti-$Fy^a$ and anti-$Fy^b$ sera. The number of the tests completed is still relatively small but the value of the system in tracing and measuring an African component in populations is becoming evident. This is particularly the case in the Near East where a series of isogenic lines for the amorph Fy gene can now be drawn.

Table 5.1.6.a. I Duffy blood groups distribution in Iran tested with anti-$Fy^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | FY(a+) | Fy(a-) | $Fy^a$ | $Fy^b$+Fy | |
| 1-Shi'a. Yazd | 151 | 66 | 85 | 24.97 | 75.03 | Sunderland,E., & Smith,H.M. 1966 |
| 2-Lurs.Luristan | 143 | 79 | 64 | 33.10 | 66.90 | Present study. |
| 3-Arabs.Abadan | 155 | 90 | 65 | 35.24 | 64.76 | Nijenhuis,L.E. 1964 |
| 4-Baluchis.Sistan & Baluchistan | 47 | 28 | 19 | 36.42 | 63.58 | Present study. |
| 5-Zoroastrians | 86 | 52 | 34 | 37.12 | 62.88 | Present study. |
| 6-Zabolis.Sistan & Baluchistan | 115 | 70 | 45 | 37.45 | 62.55 | Present study. |
| 7-Armenians | 78 | 48 | 30 | 37.98 | 62.02 | Nijenhuis,L.E . 1964 |
| 8-Central and South | 95 | 59 | 36 | 38.45 | 61.55 | Bajatzadeh,M., & Walter, H. 1969 |
| 9-Kurds. Rezaieh | 138 | 89 | 49 | 40.41 | 59.59 | Present study. |
| 10-Tehran | 92 | 60 | 32 | 41.02 | 58.98 | Bajatzadeh,M., & Walter,H. 1969 |
| 11-East | 62 | 41 | 21 | 41.80 | 58.20 | Bajatzadeh,M., & Walter, H. 1969 |

Table 5.1.6.a.I.(Cont.) Duffy blood groups distribution in Iran tested with anti-$Fy^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a- ) | $Fy^a$ | $Fy^b$+Fy | | |
| 12-South east | 348 | 235 | 113 | 43.02 | 56.98 | Nijenhuis,L.E. | 1964 |
| 13-Kurds.Sanandaj | 107 | 76 | 31 | 43.68 | 56.32 | Lehmann,H.,et al. | 1973 |
| 14-Bakhtiaris | 137 | 94 | 43 | 43.97 | 56.03 | Nijenhuis,L.E. | 1964 |
| 15-Assyrians | 32 | 22 | 10 | 44.10 | 55.90 | Nijenhuis,L.E. | 1964 |
| 16-Turks.Rezaieh | 140 | 98 | 42 | 45.23 | 54.77 | Present study. | |
| 17-Kurds.Kermanshah | 127 | 89 | 38 | 45.30 | 54.70 | Nijenhuis,L.E. | 1964 |
| 18-West | 95 | 68 | 27 | 46.69 | 53.31 | Bajatzadeh,M., & Walter, H. | 1969 |
| 19-Kurdish Jews | 94 | 73 | 21 | 50.16 | 49.84 | Godber,Marilyn.J.,et al. | 1973 |
| 20-Kurds.Baneh,Marivan | 77 | 61 | 16 | 51.50 | 48.50 | Lehmann,H., et al. | 1973 |
| 21-North west | 59 | 46 | 13 | 53.06 | 46.94 | Bajatzadeh,M., & Walter, H. | 1969 |
| 22-Kurdish Jews | 106 | 83 | 23 | 53.42 | 46.58 | Tills , D., et al. | 1977 |
| 23-North | 56 | 44 | 12 | 53.71 | 46.29 | Bajatzadeh,M., & Walter, H. | 1966 |

Table 5.1.6.a. I (Cont.) Duffy blood groups distribution in Iran tested with anti-$Fy^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a- ) | $Fy^a$ | $Fy^b+Fy$ | | |
| 24-Ghashghais | 66 | 52 | 14 | 53.95 | 46.05 | Nijenhuis, L.E. | 1964 |
| 25-Tehran | 135 | 111 | 24 | 57.83 | 42.17 | Boue' & Boue'.. | 1956 |

Table 5.1.6.a.II Duffy blood groups distribution in the Caucasus tested with anti-$Fy^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a-) | $Fy^a$ | $Fy^b+Fy$ | |
| 1-Svanis. Georgia, Abkhazeskaya | 339 | 250 | 89 | 48.76 | 51.24 | Verbitsky,M.Sh., 1972 et al. |

Table 5.1.6.a. III Duffy blood groups distribution in Turkey tested with anti-$Fy^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a-) | $Fy^a$ | $Fy^b+Fy$ | |
| 1-Eti-Turks.Asia Minor, near Mersin. | 116 | 78 | 38 | 42.76 | 57.24 | Aksoy,M.,et al. 1958 |
| 2-Turks.Asia Minor,Mersin | 107 | 79 | 28 | 48.84 | 51.16 | Aksoy,M.,et al. 1958 |

Table 5.1.6.a. IV Duffy blood groups distribution in Iraq tested with anti-$Fy^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a-) | $Fy^a$ | $Fy^b+Fy$ | |
| 1-Kurds | | | | 20.00 | 80.00 | Al-Khafaji,S.D., et al. 1976 |
| 2-Kurdish Jews.South east | 50 | 32 | 18 | 40.00 | 60.00 | Tills,D.,et al. 1977 |
| 3-Kurdish Jews | 27 | 18 | 9 | 40.44 | 59.56 | Godber,Marilyn.J., et al. 1973 |
| 4-Kurdish Jews.North West | 61 | 43 | 18 | 45.68 | 54.32 | Tills,D., et al. 1977 |
| 5-Karaite Jews | 72 | 55 | 17 | 51.41 | 48.59 | Goldschmidt,E.,et al. 1976 |
| 6-Assyrians | 99 | 93 | 6 | 75.38 | 24.62 | Ikin,Elizabeth W., et al. 1965 |

Table 5.1.6.a. V Duffy blood groups distribution in Kuwait tested with anti-$Fy^a$ serum only

| Population | Number Tested | Phenotype | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a-) | $Fy^a$ | $Fy^b+Fy$ | |
| 1-Ajman tribe | 52 | 21 | 31 | 23.00 | 77.00 | Khaled,E.,et al. 1981 |
| 2-General population | 74 | 36 | 38 | 28.00 | 72.00 | Khaled,E.,et al. 1981 |
| 3-Kuwaiti Arabs | 140 | 69 | 71 | 28.79 | 71.21 | Sawhney, K.S. 1975 |
| 4-Suluba tribe | 51 | 29 | 22 | 34.00 | 66.00 | Khaled,E.,et al. 1981 |

Table 5.1.6.a. VI

Duffy blood groups dsitribution in Saudi Arabia tested with anti-$Fy^a$ serum only.

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a-) | $Fy^a$ | $Fy^b+Fy$ | |
| 1-Shias.Qatif & Hasa Oases | 465 | 41 | 424 | 4.51 | 95.49 | Maranjian,G.,et al. 1966 |
| 2-Western Saudi Arabia | 208 | 53 | 155 | 13.68 | 86.32 | Saha, N., et al. 1980 |
| 3-Sunnis.Qatif & Hasa Oases | 319 | 88 | 231 | 14.91 | 85.09 | Maranjian,G.,et al. 1966 |
| 4-Sunnis Asir,Hejaz,Najran | 173 | 49 | 124 | 15.34 | 84.66 | Maranjian,G.,et al. 1966 |
| 5-Bedouins | 175 | 80 | 95 | 26.32 | 73.68 | Maranjian,G.,et al. 1966 |
| 6-Sunnis. Najd | 180 | 98 | 82 | 32.50 | 67.50 | Maranjian,G.,et al. 1966 |

Table 5.1.6.a.VII Duffy blood groups distribution in the United Arab Emirates tested with anti-$Fy^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a-) | $Fy^a$ | $Fy^b+Fy$ | | |
| 1-Abu-Dhabians | 100 | 34 | 66 | 18.70 | 81.30 | Kamel,K.,et al. | 1980 |

Table 5.1.6.a. VIII Duffy blood groups distribution in Pakistan tested with anit-$Fy^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a-) | $Fy^a$ | $Fy^b+Fy$ | |
| 1-Hunza,Gilgit | 23 | 16 | 7 | 44.84 | 55.16 | Ikin.,Elizabeth W., et al. 1959 |
| 2-Punjabis.Lahore | 68 | 52 | 16 | 51.49 | 48.51 | Boyd,W.C., & Boyd,L.G. 1954 |
| 3-Pathans.Swat,Saidu Sharif | 72 | 55 | 17 | 52.78 | 47.22 | Alciati,G. 1968 |
| 4-Baltis.Baltistan | 79 | 70 | 9 | 66.25 | 33.75 | Clegg,E.J.,et al. 1961 |
| 5-Pakistanis | 55 | 51 | 4 | 73.04 | 26.96 | Cutbush,Marie.,et al.1950 |

Table 5.1.6.a IX Duffy blood groups distribution in Afghanistan tested with anti-$Fy^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Fy(a+) | Fy(a-) | $Fy^a$ | $Fy^b$+Fy | |
| 1-Pushtus | 104 | 82 | 22 | 53.90 | 46.10 | Papiha,S.S.,et al. 1977 |
| 2-Timuri & related tribes | 118 | 97 | 21 | 57.81 | 42.19 | Woodd-Walker,R.B., et al. 1967 |
| 3-Daris | 179 | 152 | 27 | 59.80 | 40.20 | Papiha,S.S.,et al. 1977 |

Table 5.1.6.b.I Duffy blood groups distribution in Iran tested with anti-$Fy^a$ and anti-$Fy^b$ sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | Fy(a+b-) | Fy(a+b+) | Fy(a-b+) | Fy(a-b-) | $Fy^a$ | $Fy^b$ | Fy | |
| 1-Kurds. Sanandaj | 107 | 28 | 48 | 27 | 4 | 43.68 | 42.85 | 13.47 | Lehmann,H.,et al. 1973 |
| 2-Kurdish Jews | 87 | 31 | 38 | 14 | 4 | 51.10 | 34.28 | 14.62 | Tills,D.,et al. 1977 |
| 3-Kurdish Jews | 94 | 34 | 39 | 17 | 4 | 50.16 | 34.64 | 15.20 | Godber,Marilyn J., 1973 et al. |
| 4-Kurds.Baneh,Marivan | 77 | 30 | 31 | 12 | 4 | 51.50 | 31.75 | 16.75 | Lehmann,H.,et al. 1973 |

Table 5.1.6.b. IV Duffy blood groups distribution in Iraq tested with anti-$Fy^a$ and anti-$Fy^b$ sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | Fy(a+b-) | Fy(a+b+) | Fy(a-b+) | Fy(a-b-) | $Fy^a$ | $Fy^b$ | Fy | |
| 1-Kurdish Jews. North west | 20 | 9 | 6 | 4 | 1 | 49.59 | 29.05 | 21.36 | Tills,D.,et al. 1977 |
| 2-Kurdish Jews | 27 | 8 | 10 | 7 | 2 | 40.44 | 37.45 | 22.11 | Godber,Marilyn. 1973 J., et al. |

Table 5.1.6.b. V

Duffy blood groups distribution in Kuwait tested with anti-$Fy^a$ and anti-$Fy^b$ sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | Fy(a+b-) | Fy(a+b+) | Fy(a-b+) | Fy(a-b-) | $Fy^a$ | $Fy^b$ | Fy | |
| 1-Suluba tribe | 51 | 14 | 15 | 19 | 3 | 34.00 | 42.00 | 24.00 | Khaled,E.,et al.1981 |
| 2-General population | 74 | 21 | 15 | 27 | 11 | 28.00 | 34.00 | 38.00 | Khaled,E.,et al.1981 |
| 3-Ajman tribe | 52 | 11 | 10 | 23 | 8 | 23.00 | 39.00 | 38.00 | Khaled,E.,et al.1981 |

Table 5.1.6.b. VII Duffy blood groups distribution in the United Arab Emirates tested with anti-$Fy^a$ and anti-$Fy^b$ sera

| Popualtion | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Fy(a+b-) | Fy(a+b+) | Fy(a-b+) | Fy(a-b-) | $Fy^a$ | $Fy^b$ | Fy | | |
| 1-Abu-Dhabians | 100 | 28 | 6 | 19 | 47 | 18.70 | 13.30 | 68.00 | Kamel,K.,et al. | 1980 |

Table 5.1.6.b. VIII Duffy blood groups distribution in Pakistan tested with anti-$Fy^a$ and anti-$Fy^b$ sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | Fy(a+b-) | Fy(a+b+) | Fy(a-b+) | Fy(a-b-) | $Fy^a$ | $Fy^b$ | Fy | |
| 1-Pathans.Swat,Saidu Sharif | 72 | 21 | 34 | 17 | - | 52.78 | 47.22 | 0.00 | Alciati,G. 1968 |

Table 5.1.6.b. IX Duffy blood groups distribution in Afghanistan tested with anti-$Fy^a$ and anti-$Fy^b$ sera

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | Fy(a+b-) | Fy(a+b+) | Fy(a-b+) | Fy(a-b-) | $Fy^a$ | $Fy^b$ | Fy | |
| 1-Daris | 179 | 66 | 86 | 27 | - | 59.80 | 38.20 | 2.00 | Papiha,S.S.,et al. 1977 |
| 2-Pushtus | 104 | 41 | 41 | 21 | 1 | 53.90 | 36.40 | 9.70 | Papiha,S.S.,et al. 1977 |

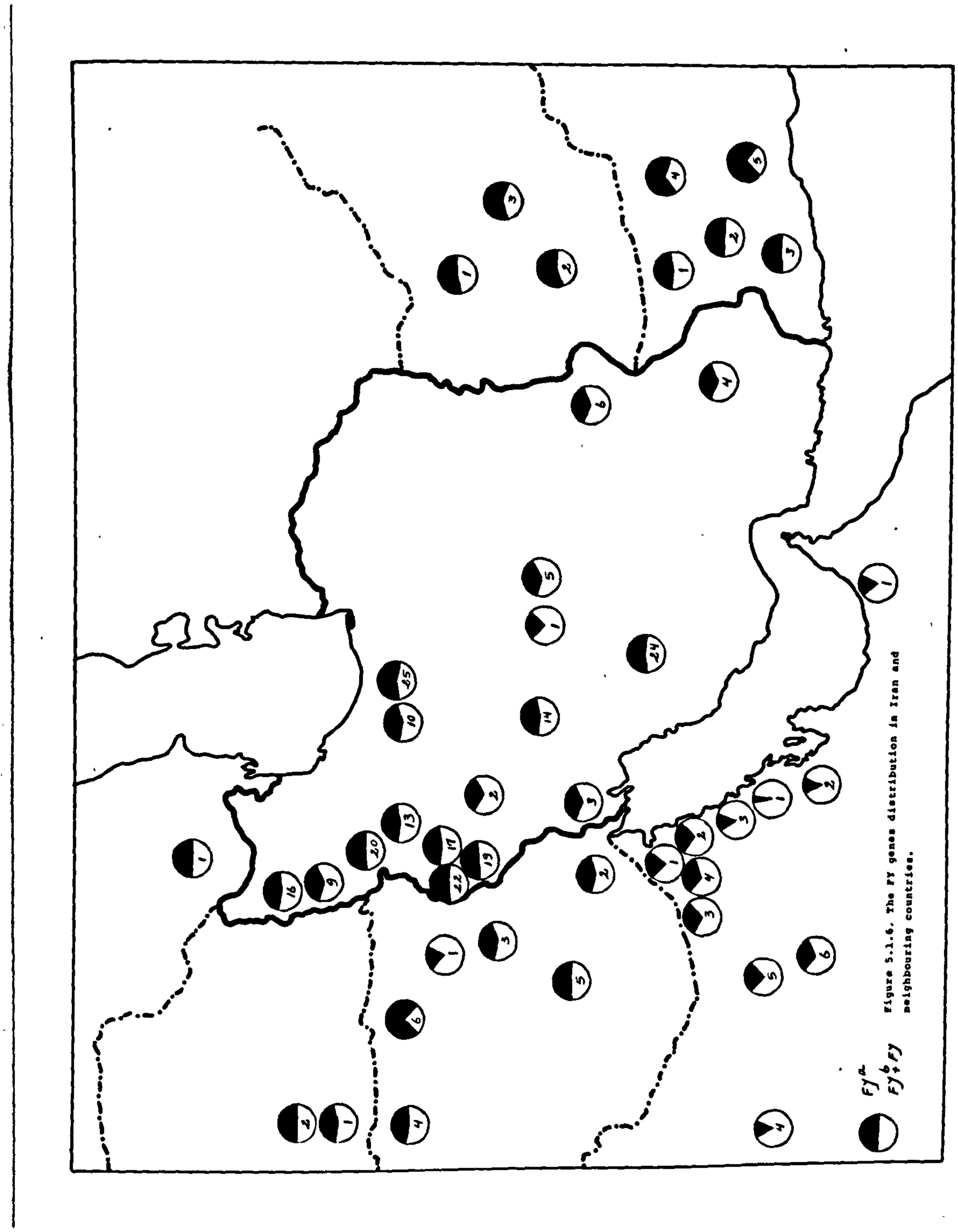



Figure 5.1.6. The FY genes distribution in Iran and neighbouring countries.

## 5.1.7 The Kidd blood group system

The distribution of the Kidd blood groups and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.1.7.

There are only a few reports of the Kidd blood groups in Iranian and neighbouring populations.

The frequency of the $JK^a$ gene ranges from 25.52 to 48.49 percent in Iranians, being lowest in the Lurs of Luristan (present investigation) and highest in the Tehrani series of Sawhney (1975).

On the whole, with an average $JK^a$ frequency of 37.34 percent, the Iranian population appears to exhibit a much lower $JK^a$ frequency than that of around 50 percent found in Europeans (Mourant et al, 1976).

Since the data available on the distribution of the Kidd system in various populations of Iran are still not large enough, full discussion is not yet possible.

Regarding neighbouring groups, a $JK^a$ frequency of 43.36 percent is reported by Verbitsky et al (1972) in the Svanis of Georgia, Abkhazskaya of the Caucasus, a figure higher than that found in Iranians but lower than the European frequency.

Information on the distribution of the Kidd genes in the inhabitants of Turkey is lacking.

The frequency of the $JK^a$ gene in the Iraqi populations varies between 32.92 percent in the Kurdish Jews of the north west and 52.86 percent in the Kurdish Jews of the south east (Tills et al, 1977). With an average $JK^a$ frequency of 41.03 percent, the Kurdish population of Iraq seems to show a higher $JK^a$ frequency than that found in Iranians but lower than the frequency in Europeans. The Iraqi Kurds show, however, a high-

er $JK^a$ frequency than the average for Iran but the frequency is lower than that of average 43.90 percent found in the Kurdish Jews of western Iran ( Godber et al, 1973; and Tills et al, 1977).

The $JK^a$ gene frequency in the populations of Kuwait ranges from 56.00 percent in the Suluba tribe to 76.00 percent in the Ajman tribe (Khaled et al, 1981). With an average $JK^a$ frequency of 63.33 percent, the Kuwaiti population appears to exhibit a much higher $JK^a$ frequency than that found in Iranians and even much higher than the frequency in Europeans. As with cDe(Ro) and Duffy null ($Fy^-$), the distribution of the $JK^a$ gene reflects the effect of African gene flow into the Arabian pool.

Unfortunately, there are no data available on the distribution of the Kidd genes in the Arab populations of Saudi Arabia and the United Arab Emirates.

The frequency of the $JK^a$ gene in the populations of Pakistan varies between 43.88 percent in the Pathans (Alciati, 1968) and 48.16 percent in the Punjabis (Boyd and Boyd, 1954). With an average $JK^a$ frequency of 46.02 percent, the Pakistani population seems to show a higher $JK^a$ frequency than that found in Iranians but still lower than the European fequency.

The $JK^a$ frequency in the populations of Afghanistan ranges from 50.00 percent in the Daris to 65.90 percent in the Pushtus (Papiha et al, 1977). With an average $JK^a$ frequency of 57.95 percent, the Afghan population appears to exhibit a much higher $JK^a$ frequency than that found in Iranians and somewhat higher than the frequency in Europeans.

## Conclusion

The frequency of the $JK^a$ gene in all European populations tested is near 50 percent ( average 51.40 percent). It is high, around 75 percent, in all African populations investigated (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arab population of Kuwait with their very high $JK^a$ frequency, the frequency of the $JK^a$ gene is below the typical European level. The average frequency of the $JK^a$ gene appears to be somewhat higher than the European level in the Afghan populations.

Again, as with cDe (Ro) and Duffy null ($Fy^-$), the high frequency of the $JK^a$ gene in the Arab population of Kuwait reflects the effect of African gene flow into the Arabian pool.

Table 5.1.7. I Kidd blood groups distribution in Iran tested with anti-$JK^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | JK(a+) | JK(a-) | $JK^a$ | $JK^b+JK$ | |
| 1-Lurs.Luristan | 146 | 65 | 81 | 25.52 | 74.48 | Present study. |
| 2-Kurds.Rezaieh | 136 | 69 | 67 | 29.81 | 70.19 | Present study. |
| 3-Baluchis.Sistan & Baluchistan | 111 | 58 | 53 | 30.90 | 69.10 | Present study. |
| 4-Turks.Rezaieh | 141 | 74 | 67 | 31.07 | 68.93 | Present study. |
| 5-Zabolis.Sistan & Baluchistan | 115 | 70 | 45 | 37.45 | 62.55 | Present study. |
| 6-Kurdish Jews | 94 | 64 | 30 | 43.51 | 56.49 | Godber,Marilyn J.,et al. 1973 |
| 7-Kurdish Jews | 87 | 60 | 27 | 44.30 | 55.70 | Tills,D.,et al. 1977 |
| 8-Zoroastrians | 86 | 60 | 26 | 45.02 | 54.98 | Present study. |
| 9-Tehran | 49 | 36 | 13 | 48.49 | 51.51 | Sawhney,K.S. 1975 |

Table 5.1.7. II

Kidd blood groups distribution in the Caucasus tested with anti-$JK^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | JK(a+) | JK(a-) | $JK^a$ | $JK^b$+JK | |
| 1-Svanis.Georgia, Abkhazskaya | 159 | 108 | 51 | 43.36 | 56.64 | Verbitsky,M.Sh.,et al. 1972 |

Table 5.1.7. IV Kidd blood groups distribution in Iraq tested with anti-$JK^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | JK(a+) | JK(a-) | $JK^a$ | $JK^b$+JK | |
| 1-Kurdish Jews.North West | 20 | 11 | 9 | 32.92 | 67.08 | Tills,D.,et al. 1977 |
| 2-Kurdish Jews | 27 | 15 | 12 | 33.34 | 66.66 | Godber,Marilyn J., et al. 1973 |
| 3-Kurds | | | | 45.00 | 55.00 | Al-Khafaji,S.D.,et al. 1976 |
| 4-Kurdish Jews.South east | 9 | 7 | 2 | 52.86 | 47.14 | Tills,D.,et al. 1977 |

Table 5.1.7. V Kidd blood groups distribution in Kuwait tested with anti-$JK^a$ serum only

| Population | Number tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | JK(a+) | JK(a-) | $JK^a$ | $JK^b$+JK | | |
| 1-Suluba tribe | 51 | 41 | 10 | 56.00 | 44.00 | Khaled,E.,et al. | 1981 |
| 2-General population | 74 | 61 | 13 | 58.00 | 42.00 | Khaled,E.,et al. | 1981 |
| 3-Ajman tribe | 52 | 49 | 3 | 76.00 | 24.00 | Khaled,E.,et al. | 1981 |

Table 5.1.7. VIII

Kidd blood groups distribution in Pakistan tested with anti-$JK^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | JK(a+) | JK(a- ) | $JK^a$ | $JK^b$+JK | |
| 1-Pathans.Swat,Saidu Sharif | 127 | 87 | 40 | 43.88 | 56.12 | Alciati, G. 1968 |
| 2-Punjabis.Lahore | 67 | 49 | 18 | 48.16 | 51.84 | Boyd,W.C., & Boyd,L.G.1954 |

Table 5.1.7. IX Kidd blood groups distribution in Afghanistan tested with anti-$JK^a$ and anti-$JK^b$ sera

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | JK(a+b+) | JK(a+b-) | JK(a-b+) | $JK^a$ | $JK^b$ | |
| 1-Daris | 38 | 22 | 8 | 8 | 50.00 | 50.00 | Papiha,S.S.,et al. 1977 |
| 2-Pushtus | 22 | 9 | 10 | 3 | 65.90 | 34.10 | Papiha,S.S.,et al. 1977 |

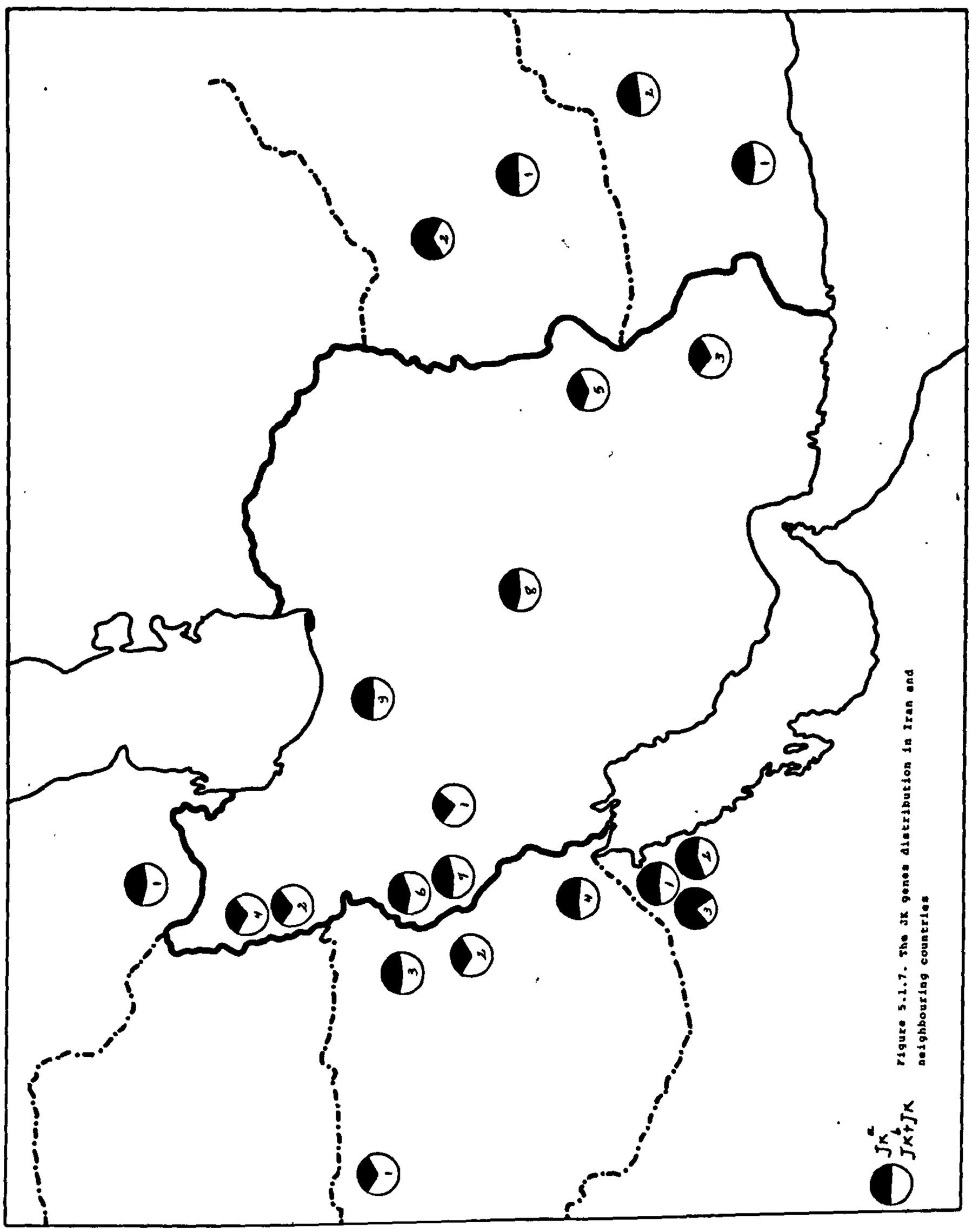



Figure 5.1.7. The JK genes distribution in Iran and neighbouring countries

## 5.1.8. The Lutheran blood group system

The distribution of the Lutheran blood groups and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.1.8.

The frequency of the $Lu^a$ gene ranges from zero to 6.90 percent in Iranians, being lowest in the Kurdish Jews (Godber et al, 1973) and highest in the Tehrani series of Sawhney and Farhud (1979). There are only a few reports of Lutheran blood groups studies in Iranians. Godber et al (1973) and Tills et al (1977) reported $Lu^a$ frequencies of zero and 0.47 percent in two groups of Kurdish Jews studied by them. Similar low $Lu^a$ frequencies of 0.65 and 2.36 percent are given by Lehmann et al (1973) in two groups of Kurds of western Iran.

The relatively low $Lu^a$ values in both the Kurds and the Kurdish Jews are about what would be expected in the region. More recently sawhney (1975) and Sawhney and Farhud (1979) reported high $Lu^a$ frequencies of 6.81 and 6.90 percent in two Esfahani and Tehrani samples, respectively, which are much higher than Middle eastern and even higher than European values, showing the presence of the gene $Lu^a$ at an appreciable level in Iran. However, as mentioned by the authors themselves, it must be substantiated by further research on ethnic, religious and geographically.isolated groups whether this frequency is a characteristic feature of Iranians or whether some other factors are involved.

On the whole, from the data available, the Iranian population with an average $Lu^a$ frequency of 2.86 percent appears to exhibit a lower $Lu^a$ frequency than that of about 4 percent in the populations of northern European origin. The average fre-

quency of the $Lu^a$ gene in Iranians seems to be more similar to that of about 2 percent in the Mediterranean area (Mourant et al, 1976).

Regarding neighbouring populations, the frequency of the $Lu^a$ gene in the populations of the Caucasus ranges from zero in the Georgians (Kherumian et al, 1954 ) to 8.44 percent in the Svanis of Georgia, Abkhazskaya ( Verbitsky et al, 1971 ). Apart from the $Lu^a$ frequency of zero which possibly is due to the very small sample size, the frequency of 8.44 percent is exceptionally high, even for Europe.

The relatively low $Lu^a$ frequencies of 0.43 and 1.40 percent in the Eti-Turks and the Turks of Turkey (Aksoy et al, 1958) are about what would be expected for the Middle East.

Similar low $Lu^a$ frequencies, varying between 0.82 and 1.87 percent are reported by Tills et al (1977) and Godber et al (1973) in Iraqi Kurdish Jews.

The frequency of the $Lu^a$ gene in the populations of Saudi Arabia ranges from zero in the Sunni sample of Asir, Hejaz, Najran (Maranjian et al, 1966) to 12.21 percent in western Saudi Arabians (Saha et al, 1980). With the exception of the extremely high $Lu^a$ value of 12.21 percent, which if true,possibly suggests African admixture, all other frequencies varying between zero and 1.09 percent are lower than the European frequency and about what would be expected for the region.

Clegg et al (1961) and Ikin and Warburton (1959) reported no Lu(a+) phenotype in the two Pakistani samples studied by them.

The $Lu^a$ gene frequency in the populations of Afghanistan varies between zero in the Pushtus and 3.90 percent in the Daris (papiha et al, 1977), figures similar to those found

in Iranians and about what would be expected for the region.

Conclusion

It is suggested that the gene $Lu^a$ has a frequency of about 4 percent in populations of northern European origin. In the Mediterranean area the frequency falls to about 2 percent, but in the Middle East the range lies between 1 and 2 percent. In Africa, though frequencies are variable, the average is nearly as high as in northern Europe. The gene is very rare or absent in all other indigenous populations examined (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the $Lu^a$ frequency of 8.44 percent in the Svanis of Georgia, Abkhazeskaya of the Caucasus ( Verbitsky, et al,1971) which is exceptionally high, even higher than the European frequency, all other reported frequencies of the $Lu^a$ gene ( average between about 1 and 2 percent ) are lower than the European frequency and about what would be expected for the region.

The extremely high $Lu^a$ frequency of 12.21 percent in western Saudi Arabians (Saha et al, 1980) contrasts strongly with the low frequencies varying between zero and 1.09 percent in other Saudi Arabian samples (Maranjian et al, 1966) and, if true, seems to be suggestive of African admixture.

Table 5.1.8.I Lutheran blood groups distribution in Iran tested with anti-$Lu^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | Lu(a+) | Lu(a-) | $Lu^a$ | $Lu^b$ | | |
| 1-Kurdish Jews | 94 | - | 94 | 0.00 | 100.00 | Godber,Marilyn J., et al. | 1973 |
| 2-Kurdish Jews | 106 | 1 | 105 | 0.47 | 99.53 | Tills,D.,et al. | 1977 |
| 3-Kurds.Baneh,Marivan | 77 | 1 | 76 | 0.65 | 99.35 | Lehmann,H.,et al. | 1973 |
| 4-Kurds.Sanandaj | 107 | 5 | 102 | 2.36 | 97.64 | Lehmann,H.,et al. | 1973 |
| 5-Esfahan | 76 | 10 | 66 | 6.81 | 93.19 | Sawhney,K.S. | 1975 |
| 6-Moslems.Tehran | 112 | 15 | 97 | 6.90 | 93.10 | Sawhney,K.S.,& Farhud,D.D. | 1979 |

Table 5.1.8. II Lutheran blood groups distribution in the Caucasus tested with anti-$Lu^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Lu(a+) | Lu(a-) | $Lu^a$ | $Lu^b$ | |
| 1-Georgians | 19 | - | 19 | 0.00 | 100.00 | Kherumian,R.,et al. 1954 |
| 2-Svanis.Georgia | 77 | 10 | 67 | 8.44 | 91.56 | Verbitsky,M.Sh., 1971 et al. |
| Abkhazskaya | | | | | | |

Table 5.1.8. III Lutheran blood groups distribution in Turkey tested with anti-$Lu^a$ serum only.

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | Lu(a+) | Lu(a-) | $Lu^a$ | $Lu^b$ | | |
| 1-Eti-Turks.Asia Minor, near Mersin | 116 | 1 | 115 | 0.43 | 99.57 | Aksoy,M.,et al. | 1958 |
| 2-Turks.Asia Minor, Mersin | 108 | 3 | 105 | 1.40 | 98.60 | Aksoy,M.,et al. | 1958 |

Table 5.1.8.IV Lutheran blood groups distribution in Iraq tested with anti-$Lu^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | Lu(a+) | Lu(a-) | $Lu^a$ | $Lu^b$ | | |
| 1-Kurdish Jews. North West | 61 | 1 | 60 | 0.82 | 99.18 | Tills,D., et al. | 1977 |
| 2-Kurdish Jews. South east | 50 | 1 | 49 | 1.01 | 98.99 | Tills,D., et al. | 1977 |
| 3-Kurdish Jews | 27 | 1 | 26 | 1.87 | 98.13 | Godber,Marilyn J., et al. | 1973 |

Table 5.1.8.VI Lutheran blood groups distribution in Saudi Arabia tested with anti-$Lu^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Lu(a+) | Lu(a-) | $Lu^a$ | $Lu^b$ | |
| 1-Sunnis.Asir,Hejaz | 176 | - | 176 | 0.00 | 100.00 | Maranjian,G.,et al. 1966 |
| 2-Sunnis.Najd | 180 | 3 | 177 | 0.84 | 99.16 | Maranjian,G.,et al. 1966 |
| 3-Bedouins | 178 | 3 | 175 | 0.85 | 99.15 | Maranjian,G.,et al. 1966 |
| 4-Shias.Qatif & Hasa Oases | 465 | 9 | 456 | 0.97 | 99.03 | Maranjian,G.,et al. 1966 |
| 5-Sunnis,Qatif & Hasa Oases and east | 323 | 7 | 316 | 1.09 | 98.91 | Maranjian,G.,et al. 1966 |
| 6-Western Saudi Arabia | 185 | 26 | 159 | 12.21 | 87.79 | Saha, N., et al. 1980 |

Table 5.1.8. VIII Lutheran blood groups distribution in Pakistan tested with anti-$Lu^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Lu(a+) | Lu(a-) | $Lu^a$ | $Lu^b$ | |
| 1-Baltis.Baltistan | 80 | - | 80 | 0.00 | 100.00 | Clegg,E.J.,et al. 1961 |
| 2-Hunza,Gilgit | 23 | - | 23 | 0.00 | 100.00 | Ikin,Elizabeth W., & Warburton,K. 1959 |

Table 5.1.8. IX Lutheran blood groups distribution in Afghanistan tested with anti-$Lu^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Lu(a+) | Lu(a-) | $Lu^a$ | $Lu^b$ | |
| 1-Pushtus | 22 | - | 22 | 0.00 | 100.00 | Papiha,S.S.,et al. 1977 |
| 2-Timuri & related tribes | 90 | 3 | 87 | 1.68 | 98.32 | Woodd-Walker,R.B., et al. 1967 |
| 3-Daris | 38 | 3 | 35 | 3.90 | 96.10 | Papiha,S.S.,et al. 1977 |

## 5.1.9. The Diego blood group system

The distribution of the Diego blood groups and respective gene frequencies in Iranian and neighbouring populations is shown in Tables 5.1.9.

Only a single example of Di(a+) has been found in the Kurds of Iran (Lehmann et al, 1973), the Baltis of Pakistan (Clegg et al, 1961) and the Timuri and related tribes of Afghanistan (Woodd-Walker et al, 1967).

Conclusion

The $Di^a$ gene is essentially characteristic of Mongoloids and has never been found in any survey of unselected persons of presumed unmixed European descent. It appears to be totally absent among Africans. The highest frequencies of the $Di^a$ gene( up to 40 percent) are found among south and central American Indians.

In eastern Asia it is constantly present, though frequencies are not quite as high as in south and central America. The highest gene frequencies in Asia, around 5 percent, are found in Koreans and Tibetans. It is also present in Chinese, Japanese, the people of Thailand, northewest India and Nepal ( Mourant et al, 1976).

The presence of even one Diego($Di^a$) positive among the Iranian, the Pakistani, and the Afghan populations suggests a Mongoloid contribution to the gene pools.

It is most desirable that data should be obtained on its distribution in the Iranian Turkmans with Mongoloid origin and in the indigenous peoples of the Asiatic part of the Soviet Union.

Table 5.1.9.I Diego blood groups distribution in Iran tested with anti-$Di^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Di(a+) | Di(a-) | $Di^a$ | $Di^b$ | |
| 1-Arabs.Abadan | 98 | - | 98 | 0.00 | 100.00 | Nijenhuis,L.E. 1964 |
| 2-Iranians | 16 | - | 16 | 0.00 | 100.00 | Nijenhuis,L.E. 1964 |
| 3-Kurdish Jews | 116 | - | 116 | 0.00 | 100.00 | Gödber,Marilyn.J., et al. 1973 |
| 4-Arabs | 100 | - | 100 | 0.00 | 100.00 | Nijenhuis,L.E. 1958 |
| 5-Kurds.Sanandaj | 107 | - | 107 | 0.00 | 100.00 | Lehmann,H.,et al. 1973 |
| 6-Kurds.Baneh,Mar-vivan | 77 | 1 | 76 | 0.65 | 99.35 | Lehmann,H.,et al. 1973 |

Table 5.1.9. VIII Diego blood groups distribution in Pakistan tested with anti-$Di^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Di(a+) | Di(a-) | $Di^a$ | $Di^b$ | |
| 1-Hunza,Gilgit | 23 | - | 23 | 0.00 | 100.00 | Ikin,Elizabeth W.,1959 & Warburton,K. |
| 2-Pathans | 139 | - | 139 | 0.00 | 100.00 | Vos,G.H.,& Kirk, R.L. 1961 |
| 3-Punjabis | 168 | - | 168 | 0.00 | 100.00 | Vos,G.H.,& Kirk, R.L. 1961 |
| 4-Baltis.Baltistan | 79 | 1 | 78 | 0.64 | 99.36 | Clegg,E.J.,et al. 1961 |

Table 5.1.9.IX Diego blood groups distribution in Afghanistan tested with anti-$Di^a$ serum only

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Di(a+) | Di(a-) | $Di^a$ | $Di^b$ | |
| 1-Timuri and related tribes | 100 | 1 | 99 | 0.50 | 99.50 | Woodd-Walker,R.B.,1967 et al. |

## 5.1.10. The ABH secretion system

The distribution of secretor and non-secretor phenotypes and respective gene frequencies in Iranian and neighbouring populations is shown in Tables 5.1.10.
There are few reports on secretor status in Iranian and neighbouring populations.

The frequency of the Se allele ranges from 29.30 to 44.00 percent in Iranians, being lowest in southern Gorgan, Behshahr, Sari and highest in Tavalesh, Astara (Kirk et al, 1977). With an average Se frequency of 33.98 percent, the Iranian population appears to exhibit a lower Se frequency than that of around 50 percent in Europeans ( Mourant et al, 1976).

Regarding neighbouring populations, the Se allele frequency of 49.29 percent in the Georgians of the Caucasus(Kherumian et al, 1954) is higher than the average for Iran and more similar to the European frequency.

Information on the distribution of secretor genes in other neighbouring populations is lacking.

Conclusion

The frequency of the Se gene in England is about 51 percent. Frequencies in continental Europe tend to be slightly higher than this, without any marked regional trend.
In the Indian region there is a wide Scatter of frequencies, on the whole high in the north and low in the south (Mourant et al, 1976).

In Iranians frequencies of the Se gene seem to be lower than those in Europeans.

Table 5.1.10.I ABH Secretor and Non-secretor phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | Sec | Non-Sec | Se | se | | |
| 1-Southern Gorgan, Behshahr,Sari | 44 | 22 | 22 | 29.30 | 70.70 | Kirk,R.L.,et al. | 1977 |
| 2-Shahsavar,Rudsar, Rudbar,Rasht,Langa-rud,Lahijan,Bandar-Pahlavi | 88 | 45 | 43 | 30.10 | 69.90 | Kirk,R.L.,et al. | 1977 |
| 3-Northern Gorgan | 48 | 25 | 23 | 30.80 | 69.20 | Kirk,R.L.,et al. | 1977 |
| 4-Arabs.Khuzistan | 91 | 48 | 43 | 31.20 | 68.80 | Marzban,M. | 1978 |
| 5-Gonbad | 153 | 83 | 70 | 32.40 | 67.60 | Kirk,R.L.,et al. | 1977 |
| 6-Babol,Shahi,Amol | 78 | 50 | 28 | 40.10 | 59.90 | Kirk,R.L.,et al. | 1977 |
| 7-Tavalesh,Astara | 51 | 35 | 16 | 44.00 | 56.00 | Kirk,R.L.,et al. | 1977 |

Table 5.1.10. II ABH Secretor and Non-Secretor phenotypes and gene frequencies distribution in the Caucasus

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Sec | Non-Sec | Se | se | |
| 1- Georgians | 35 | 26 | 9 | 49.29 | 50.71 | Kherumian,R., et al. 1954 |

## 5.1.11. Other blood group systems

In this part, data on some other blood group systems are presented. Since data available on these systems are not representative for the region, no discussion will be given.

Table 5.1.11. a. I. In a blood groups in Iran : tested with anti- $In^a$ serum

| Population | Number Tested | In(a+) | In(a+)% | Authors |
|---|---|---|---|---|
| Iranians | 557 | 59 | 10.59 | Badaker,S.S., et al. 1980 |

Table 5.1.11. b. I. Wright blood groups in Iran : tested with anit-$Wr^a$ serum

| Population | Number Tested | Wr(a+) | Wr(a+)% | Authors |
|---|---|---|---|---|
| 1-Kurdish Jews | 116 | - | 0.00 | Godber,Marylin J., et al. 1973 |
| 2-Kurds.Baneh,Marivan | 77 | - | 0.00 | Lehmann,H.,et al. 1973 |
| 3-Kurds.Sanandaj | 107 | 2 | 1.87 | Lehmann,H.,et al. 1973 |

Table 5.1.11. C. I. Radin blood groups in Iran : tested with anti-Rd serum

| Population | Number Tested | Rd+ | Rd+% | Authors |
|---|---|---|---|---|
| 1-Kurds.Baneh,Marivan | 77 | - | 0.00 | Lehmann,H., et al. 1973 |
| 2-Kurds. Sanandaj | 107 | - | 0.00 | Lehmann,H., et al. 1973 |

Table 5.1.11.d.VII. Vel blood groups in the United Arab Emirates: tested with anti-$Ve^a$ serum

| Population | Number Tested | Ve(a+) | Ve(a-) | $Ve^a$ | $Ve^b$ | Authors |
|---|---|---|---|---|---|---|
| Abu Dhabians | 100 | 98 | 2 | 85.90 | 14.10 | Kamel,K.,et al. 1980 |

## 5.2. SERUM PROTEINS

### 5.2.1. The haptoglobin(HP) system

The distribution of haptoglobin types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.2.1.

The frequency of the $HP^1$ gene ranges from 14.50 to 35.57 percent in Iranians, being lowest in northern Gorgan (Kirk et al, 1977) and highest in the Shirazi series of Walter and Djahanshahi (1963). Values obtained in the present investigation are within this range of variation.

On the whole, with an average $HP^1$ frequency of 26.85 percent, the Iranian population appears to exhibit a much lower $HP^1$ frequency than that of around 40 percent found in Europeans (Mourant et al, 1976).

It is difficult to see any definite regional trends but the $HP^1$ gene frequency tends to be lower in the east than the west.

The relatively lower $HP^1$ frequencies in the Caspian sea area of Iran (Kirk et al, 1977; Farhud et al, 1978) could be related to tropical disease syndromes, such as malaria, sickle cell disease, G6pD deficiency, etc, which are more common in this area.

Regarding neighbouring areas, the frequency of the $HP^1$ gene in the populations of the Caucasus ranges from 28.49 percent in the Azerbaijanians of Nukha to 35.04 percent in the Gurdjaanis of Georgia (Voronov, 1968). With an average $HP^1$ frequency of 31.76 percent, the population of the Caucasus appears to exhibit a higher $HP^1$ frequency than that found in Iranians but lower than the European frequency.

The frequency of the $HP^1$ gene in the populations of

Turkey varies between 25.09 percent (Hummel et al, 1970)and 31.72 percent (Erdem et al, 1966) both in the Turks of Turkey. with an average $HP^1$ frequency of 27.66 percent, the population of Turkey seems to show a slightly higher $HP^1$ frequency than that found in Iranians but lower than the frequency in Europeans.

The $HP^1$ gene frequency in the Jewish populations of Iraq ranges from 27.00 percent in the Kurdish Jews of the south east to 34.43 percent in the Kurdish Jews of the north west ( Tills et al, 1977). With an average $HP^1$ frequency of 29.88 percent, the Jewish population of Iraq, like that of Iran, appears to exhibit a relatively higher $HP^1$ frequency than the average for the region.

The frequency of the $HP^1$ gene in the populations of Kuwait varies between 26.00 percent in the general population of Kuwait and 51.00 percent in the Ajman tribe (Khaled et al, 1981). With an average $HP^1$ frequency of 37.65 percent, the Arab population of Kuwait seems to show a much higher $HP^1$ frequency than that found in Iranians.

The $HP^1$ gene frequency in the populations of Saudi Arabia ranges from 36.64 percent in western Saudi Arabians (Saha et al, 1980) to 43.20 percent in the Saudi Arabian sample of Goedde et al (1979). With an average $HP^1$ frequency of 39.92 percent, the Arab population of Saudi Arabia, like that of Kuwait, appears to exhibit a much higher $HP^1$ frequency than that found in Iranians.

On the whole, the overall frequency of $HP^1$ in Arab populations seems to be higher than in other Asiatic populations but lower than an average of about 70 percent in Africans (Giblett, 1969; Mourant et al, 1976). Generally, haptoglobin

is a system indicating a substantial influx of African genes in the Arabian Peninsular populations.

The frequency of the $HP^1$ gene in the populations of Pakistan varies between 20.00 percent in the Punjabis (Kirk et al, 1961) and 30.49 percent in the Pathanis (Spedini,1969). With an average $HP^1$ frequency of 25.52 percent, the population of Pakistan seems to show a slightly lower $HP^1$ frequency than that found in Iranians and lower than the European frequency.

The $HP^1$ gene frequency in the populations of Afghanistan ranges from 23.70 percent in the Pushtus to 29.10 percent in the Daris ( Papiha et al, 1977). With an average $HP^1$ frequency of 26.47 percent, the Afghan population appears to exhibit a $HP^1$ frequency similar to that found in Iranians and lower than the frequency in Europeans.

Conclusion

The frequency of the $HP^1$ gene in Europe varies around 40 percent; it is difficult to see any definite regional trends, but it tends to be lower in the south than the north. The $HP^1$ gene frequency is mostly low, around 15 percent, in the Indian region.

In Africa it is usually high, averaging about 70 percent(Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arab populations of Kuwait and Saudi Arabia with their higher frequencies, the frequency of the $HP^1$ gene varying between 25 percent in the Pakistani population and 32 percent in the population of the Caucasus, seems to be lower than that found in Europeans but higher than that in Indians. The overall frequency of $HP^1$ in Arab populations seems to be

higher than in other Asiatic populations. Generally, haptoglobin is a system indicating a substantial influx of African genes in the Arabian Peninsular populations.

However, there appears to be a cline of decreasing $HP^1$ gene frequencies from Europe to India via the Middle East (Farhud, 1980). From the data reported in the present investigation on Iranian and neighbouring populations the same trend seems to exist, as the $HP^1$ frequency decreases obviously from the west towards the east, whilst the $HP^2$ frequency increases.

The precise significance of this cline in terms of environmental and genetic causation, can not at present be gauged but under the assumption that the HP frequencies obtained in the published samples from India are not due to chance, genetic drift or isolation effects, this observation might be of some anthropological value. In this sense one may consider the possibility of selective forces acting on the HP frequencies. Assuming that the high $HP^2$ frequencies in Iran, Pakistan and India may indicate some slective advantage of this allele, by which it might be able to extend considerably.. In this connection on may consider that the relatively large $HP^2$ moluecules can not easily pass the Kidney barrier, by which the organism will be protected from serious losses of Hb-connected Iron, which becomes disengaged with haemolytic ananemias, caused by G6pD deficiency, sickle cell disease, etc. Similar ideas have been expressed by Schultz and heremans (1966), who wrote: "since haptoglobin is in some way involved in the metabolism of haemoglobin being catabolized in excess during haemolytic episodes such as occur in malaria, it is not unthinkable that the latter disease may have

had a hand in the presently observed ascendancy of the $HP^2$ genes". Assuming these considerations the hypothesis may be formulated that the increase of $HP^2$ frequencies from the west towards the east is a result of genetic adaptation to specific environmental conditons, in which haemolytic anaemias such as occur in malaria seem to be of importance. Unfortunately we have no exact data concerning the frequency and distribution of haemolytic anaemias in south western and southern Asia, so that confidence in this hypothesis can not be checked. Nevertheless, attention should be paid to this problem. The solution will enable us to understand the geographical differences in the distribution of the HP alleles, and moreover it will improve our knowledge on the action of selective mechanisms in man.

Table 5.2.1.I Haptoglobin(HP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP 0 | $HP^1$ | $HP^2$ | | |
| 1-Northern Gorgan | 38 | - | 11 | 27 | - | 14.50 | 85.50 | Kirk,R.L.,et al. | 1977 |
| 2-Zoroastrians.Tehran & Yazd | 145 | 6 | 43 | 96 | - | 18.97 | 81.03 | Bowman,J.E. | 1964 |
| 3-Southern Gorgan,Behshahr,Sari | 52 | 2 | 17 | 33 | - | 20.20 | 79.80 | Kirk,R.L.,et al. | 1977 |
| 4-Babol,Shahi,Amol | 61 | 1 | 24 | 36 | - | 21.30 | 78.70 | Kirk,R.L.,et al. | 1977 |
| 5-Gonbad | 153 | 5 | 56 | 91 | 1 | 21.70 | 78.30 | Kirk,R.L.,et al. | 1977 |
| 6-Zabolis.Sistan & Baluchistan | 118 | 3 | 45 | 69 | 1 | 21.79 | 78.21 | Present study. | |
| 7-Kurdish Jews | 94 | 7 | 27 | 59 | 1 | 22.04 | 77.96 | Godber,Marilyn.J.,et al. | 1973 |
| 8-Shahsavar,Rudsar,Rudbar, Rasht,Langarud,Lahijan, Bandar-Pahlavi | 84 | 3 | 30 | 48 | 3 | 22.20 | 77.80 | Kirk,R.L.,et al. | 1977 |
| 9-Lurs.Luristan | 178 | 10 | 61 | 106 | 1 | 22.88 | 77.12 | Present study. | |
| 10-Kurds.Shirvan,Khorasan | 102 | 4 | 39 | 58 | 1 | 23.27 | 76.73 | Present study. | |

Table 5.2.1.I (Cont.) Haptoglobin (HP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP 0 | $HP^1$ | $HP^2$ | | |
| 11-Turks.Shirvan,Khorasan | 106 | 3 | 43 | 57 | 3 | 23.79 | 76.21 | Present study. | |
| 12-Baluchis.Sistan & Baluchistan. | 111 | 5 | 43 | 59 | 4 | 24.77 | 75.23 | Present study. | |
| 13-East | 179 | 13 | 63 | 103 | - | 24.90 | 75.10 | Bajatzadeh,M., & Walter,H. | 1969 |
| 14-Caspian Sea area | 240 | 11 | 96 | 130 | 3 | 24.90 | 75.10 | Farhud,D.D.,et al. | 1978 |
| 15-Iranians | 34 | 2 | 13 | 19 | - | 25.00 | 75.00 | Harris, H. | 1959 |
| 16-Tavalesh.Astara | 60 | 1 | 28 | 31 | - | 25.00 | 75.00 | Kirk,R.L.,et al. | 1977 |
| 17-Kurdish Jews | 106 | 9 | 35 | 61 | 1 | 25.24 | 74.76 | Tills,D.,et al. | 1977 |
| 18-Turks.Rezaieh | 145 | 11 | 51 | 79 | 4 | 25.89 | 74.11 | Present study. | |
| 19-Zoroastrians | 171 | 15 | 56 | 95 | 5 | 25.90 | 74.10 | Present sutdy. | |
| 20-Central and South | 245 | 20 | 89 | 136 | - | 26.30 | 73.70 | Bajatzadeh,M., & Walter,H. | 1969 |
| 21-Dezfool | 545 | 33 | 217 | 288 | 7 | 26.30 | 73.70 | Farhud,D.D., et al. | 1977 |
| 22-Tehran | 136 | 14 | 41 | 76 | 5 | 26.34 | 73.66 | Present study. | |

Table 5.2.1.I (Cont.) Haptoglobin (HP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP 0 | $HP^1$ | $HP^2$ | | |
| 23-Bandar.Abass | 1246 | 105 | 441 | 678 | 22 | 26.60 | 73.40 | Farhud,D.D., et al. | 1978 |
| 24-Kurds.Sanandaj | 108 | 7 | 43 | 56 | 2 | 26.89 | 73.11 | Lehmann,H.,et al. | 1973 |
| 25-Tehran | 366 | 28 | 145 | 193 | - | 27.00 | 73.00 | Farhud,D.D., & Walter, H. | 1972 |
| 26-Ghashghais | 510 | 42 | 185 | 262 | 21 | 27.50 | 72.50 | Farhud,D.D., & Amirshahi,P. un-published | |
| 27-North West | 250 | 29 | 80 | 141 | - | 27.60 | 72.40 | Bajatzadeh,M., & Walter, H. | 1969 |
| 28-Iranians | 1061 | | | | | 28.00 | 72.00 | Miyashita,T.K.,et al. | 1975 |
| 29-Iranians | 627 | 49 | 254 | 324 | - | 28.07 | 71.93 | Farhud,D.D. | 1980 |
| 30-West | 313 | 33 | 111 | 169 | - | 28.20 | 71.80 | Bajatzadeh,M., & Walter,H. | 1969 |
| 31-Moslems.Shiraz | 429 | 34 | 176 | 219 | - | 28.44 | 71.56 | Bowman, J.E. | 1964 |
| 32-Jews | 101 | 8 | 43 | 50 | - | 29.21 | 70.79 | Fried,K.,et al. | 1963 |
| 33-Tehran | 186 | 19 | 71 | 96 | - | 29.30 | 70.70 | Sawhney, K.S. | 1975 |
| 34-Jews | 91 | 9 | 37 | 45 | - | 30.22 | 69.78 | Ramot,Bracha.,et al. | 1962 |

Table 5.2.1.I (Cont.) Haptoglobin (HP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP 0 | $HP^1$ | $HP^2$ | | |
| 35-Esfahan | 91 | 6 | 42 | 41 | 2 | 30.34 | 69.66 | Sawhney,K.S. | 1975 |
| 36-Kerman | 294 | 31 | 108 | 141 | 14 | 30.36 | 69.64 | Present study. | |
| 37-Kurds.Rezaieh | 145 | 18 | 50 | 72 | 5 | 30.71 | 69.29 | Present study. | |
| 38-North | 179 | 27 | 57 | 95 | - | 31.00 | 69.00 | Bajatzadeh,M., & Walter, H. | 1969 |
| 39-Jews | 158 | | | | | 31.00 | 69.00 | Simhai,B. | 1976 |
| 40-Shahsavar | 168 | 16 | 75 | 77 | - | 31.80 | 68.20 | Farhud,D.D.,et al. | 1978 |
| 41-Jews | 159 | 18 | 66 | 74 | 1 | 32.00 | 68.00 | Tabatabai, H. | 1977 |
| 42-Tehran | 400 | 53 | 153 | 194 | - | 32.40 | 67.60 | Bajatzadeh,M., & Walter, H. | 1969 |
| 43-Ghashghais | 117 | 15 | 46 | 56 | - | 32.48 | 67.52 | Bowman,J.E. | 1964 |
| 44-Kurds.Baneh,Marivan | 77 | 8 | 31 | 33 | 5 | 32.64 | 67.36 | Lehmann,H.,et al. | 1973 |
| 45-Armenians | 228 | 15 | 127 | 85 | 1 | 34.40 | 65.60 | Tabatabai, H. | 1977 |
| 46-Shiraz | 97 | 12 | 45 | 40 | - | 35.57 | 64.43 | Walter,H., & Djahanshahi, D. | 1963 |

Table 5.2.1.b.I. Haptoglobin (HP) Subtypes in Iran

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | HP 1s-1s | HP 1s-1F | HP 1F-1F | HP 2-1s | HP 2-1F | HP 2-2 | 1s HP | 1F HP | $Hp^2$ | |
| Tehran | 366 | 8 | 13 | 7 | 88 | 57 | 193 | 16.20 | 10.80 | 73.00 | Farhud,D.D., and Walter, H. 1972 |

Table 5.2.1. II Haptoglobin (HP) phenotypes and gene frequencies distribution in the Caucasus

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP 0 | $HP^1$ | $HP^2$ | | |
| 1- Azerbaijan .Nukha | 87 | 6 | 37 | 43 | 1 | 28.49 | 71.51 | Voronov, A.A. | 1968 |
| 2-Gurdjaanis.Georgia | 141 | 16 | 64 | 57 | 4 | 35.04 | 64.96 | Voronov, A.A. | 1968 |

Table 5.2.1.III Haptoglobin(HP) phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP 2-1M | HP 0 | $HP^1$ | $HP^2$ | | |
| 1-Turks | 274 | 12 | 113 | 148 | - | 1 | 25.09 | 74.91 | Hummel,K.,et al. | 1970 |
| 2-Turks | 102 | 5 | 42 | 53 | - | 2 | 26.00 | 74.00 | Aksoy,M.,et al. | 1980 |
| 3-Turks | 200 | 15 | 76 | 109 | - | - | 26.50 | 73.50 | Erdem,S., & Aksoy,M. | 1975 |
| 4-Kurds | | | | | | | 29.00 | 71.00 | Richard, P. | 1976 |
| 5-Turks | 299 | 24 | 138 | 136 | 1 | - | 31.27 | 68.73 | Erdem,S., et al. | 1966 |

Table 5.2.1. IV Haptoglobin (HP) phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes | | | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP 2-1M | HP 0 | $HP^1$ | $HP^2$ | | |
| 1-Kurdish Jews.South east | 50 | 4 | 19 | 27 | - | - | 27.00 | 73.00 | Tills,D.,et al. | 1977 |
| 2-Jews.Baghdad | 197 | 14 | 79 | 103 | 1 | - | 27.41 | 72.59 | Fried,K.,et al. | 1963 |
| 3-Karaite Jews | 69 | 3 | 33 | 33 | - | - | 28.30 | 71.70 | Goldschmidt,E.,et al. | 1976 |
| 4-Jews | 118 | 9 | 50 | 59 | - | - | 28.81 | 71.19 | Ramot,Brancha.,et al. | 1961 |
| 5-Kurdish Jews | 27 | 3 | 12 | 12 | - | - | 33.33 | 66.67 | Godber,Marilyn.J., et al. | 1973 |
| 6-Kurdish Jews.North west | 61 | 7 | 28 | 26 | - | - | 34.43 | 65.57 | Tills,D.,et al. | 1977 |

Table 5.2.1.V Haptoglobin (HP) phenotypes and gene frequencies distribution in Kuwait

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP 0 | $HP^1$ | $HP^2$ | | |
| 1-General population | 88 | 6 | 33 | 49 | - | 26.00 | 74.00 | Khaled,E.,et al. | 1981 |
| 2-Kuwaiti Arabs | 161 | 24 | 61 | 73 | 3 | 34.59 | 65.41 | Sawhney,K.S. | 1975 |
| 3-Suluba tribe | 51 | 9 | 22 | 20 | - | 39.00 | 61.00 | Khaled,E.,et al. | 1981 |
| 4-Ajman tribe | 52 | 13 | 27 | 12 | - | 51.00 | 49.00 | Khaled,E.,et al. | 1981 |

Table 5.2.1. VI Haptoglobin(HP) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP Calb. | HP 0 | $HP^1$ | $HP^2$ | | |
| 1-Western Saudi Arabia | 292 | 35 | 133 | 109 | - | 15 | 36.64 | 63.36 | Saha,N., et al. | 1980 |
| 2-Saudi Arabians | 352 | 64 | 168 | 111 | 1 | 8 | 43.20 | 56.80 | Goedde,H.W.,et al. | 1979 |

Table 5.2.1. VIII Haptoglobin (HP) phenotypes and gene frequencies distribution in Pakistan

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP 0 | $HP^1$ | $HP^2$ | | |
| 1-Punjabis | 201 | 12 | 52 | 130 | 7 | 20.00 | 80.00 | Kirk,R.L.,et al. | 1961 |
| 2-Pathans | 185 | 10 | 67 | 103 | 5 | 24.00 | 76.00 | Kirk,R.L., et al. | 1961 |
| 3-Peshawar | 135 | 12 | 50 | 72 | 1 | 27.61 | 72.39 | Walter,H.,et al. | 1966 |
| 4-Pathanis.Swat | 131 | 11 | 53 | 59 | 8 | 30.49 | 69.51 | Spedini,Gaberiella. | 1969 |

Table 5.2.1.IX Haptoglobin (HP) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|---|
| | | HP 1-1 | HP 2-1 | HP 2-2 | HP 0 | $HP^1$ | $HP^2$ | |
| 1-Pushtus | 95 | 3 | 39 | 53 | - | 23.70 | 76.30 | Papiha,S.S., et al. 1977 |
| 2-Uzbeks | 124 | 6 | 49 | 69 | - | 24.59 | 75.41 | Rahimi,A.G.,et al. 1977 |
| 3-Pushtus | 210 | 13 | 83 | 114 | - | 25.95 | 74.05 | Rahimi,A.G.,et al. 1977 |
| 4-Tajiks | 310 | 23 | 118 | 169 | - | 26.45 | 73.55 | Rahimi,A.G.,et al. 1977 |
| 5-Afghans | 141 | 12 | 53 | 76 | - | 27.30 | 72.70 | Shim,Bong-Sop.,et al.1964 |
| 6-Hazaras | 172 | 12 | 73 | 87 | - | 28.19 | 71.81 | Rahimi,A.G.,et al. 1977 |
| 7-Daris | 158 | 13 | 66 | 79 | - | 29.10 | 70.90 | Papiha,S.S., et al. 1977 |

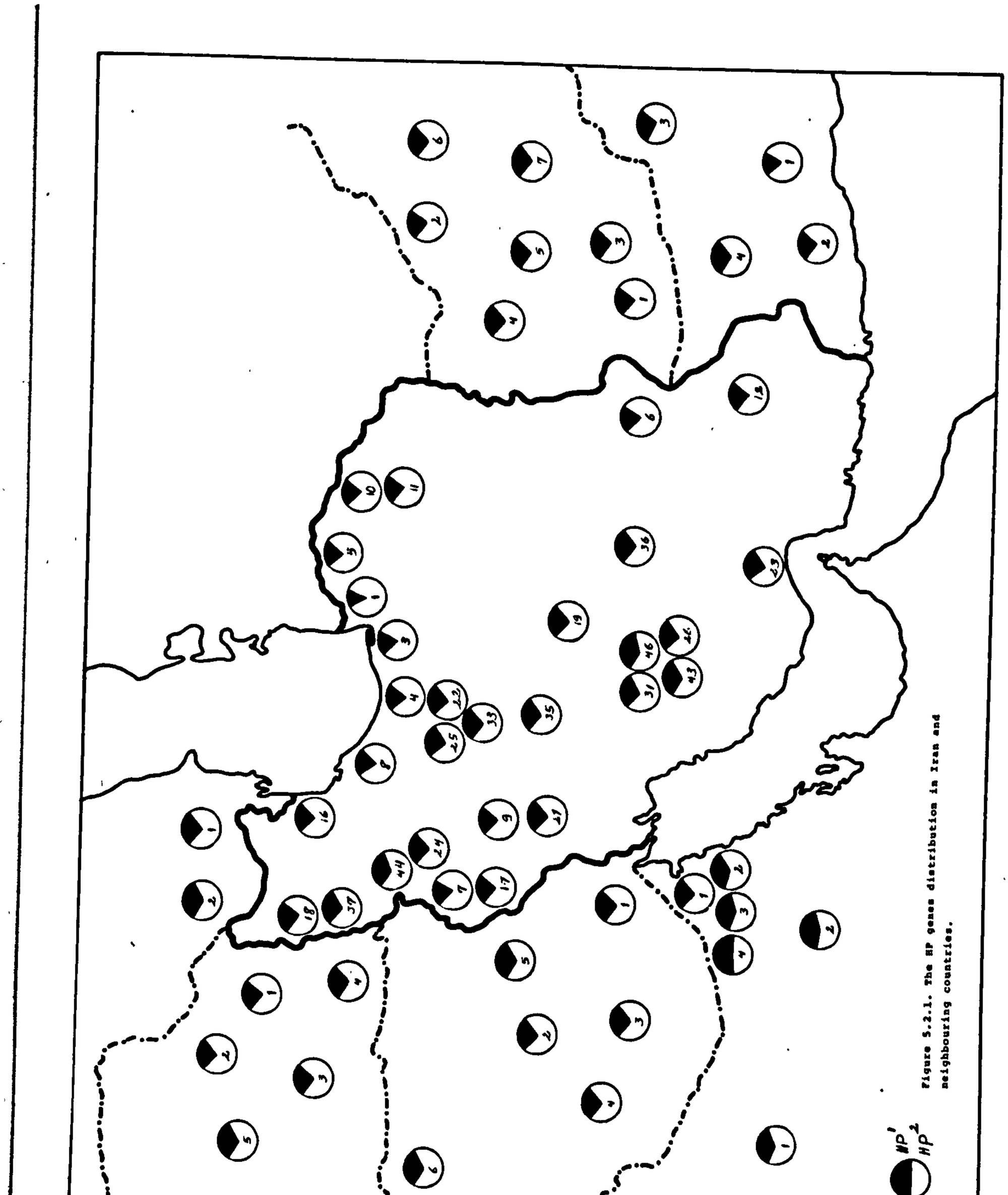



Figure 5.2.1. The HP genes distribution in Iran and neighbouring countries.

## 5.2.2. The transferrin (Tf) system

The distribution of transferrin groups and respective gene frequencies in Iranian and neighbouring populations is shown in Tables. 5.2.2.

Both TfB and TfD variants are found in Iranians but the TfD variant seems to be more frequent than TfB, the highest frequency of the $Tf^D$ allele being 2.56 percent in the Ghashghai tribe ( Bowman, 1964). It is difficult to see any definite regional trend of the distribution of the transferrin variants in Iran but it tends to be higher in the south than the north.

Regarding neighbouring groups, no transferrin variant other than type CC was observed in the Turks and the Kurds of Turkey ( Dincol, Guncag et al, 1976; Richard, 1976).

Tills et al (1977) found no TfD variants in the two groups of Iraqi Kurdish Jews studied by them but they observed the relatively high $Tf^B$ frequency of 1.64 percent in the Kurdish Jews of the north west and only TfCC type in the Kurdish Jews of the south east.

The only transferrin type observed in the three Kuwaiti samples investigated by Khaled et al (1981) was transferrin CC.

Saha et al (1980) and Goedde et al (1979) found no TfB but a high TfD variant frequency in the population of Saudi Arabia, the highest frequency of the $Tf^D$ allele being 3.40 percent in western Saudi Arabians. A high frequency of TfD variant seems to be an indication of African admixture in the Arabs of Saudi Arabia.

Kirk et al (1961) observed no TfD variant in the Pathans

and the Punjabis of Pakistan but they found $Tf^B$ variant with a low frequency of 0.50 percent in the Pathans.

Both TfD and TfB variants are found in the Afghan populations(Rahimi et al,1977;Papiha et al,1977).The high $Tf^{Dchi}$ frequency of 1.73 percent in the Hazaras of Afghanistan(Rahimi et al,1977) can be explained as a Mongoloid marker(Kirk et al, 1968) because of its high frequency in Asiatic populations.

Conclusion

Frequencies of the transferrin variants other than $Tf^C$ are very low in every population studied. The rarity of the variants suggests that the common TfC type has under most circumstances a selective advantage over all the others.
The transferrin B variants, though always rare, are the main ones found in European populations while the main distribution of the TfD variants are in the peoples of southern and south eastern Asia including India.
In Africa transferrin D variants are found to be more frequent( Mourant et al, 1976).

In Iranian and neighbouring populations,like other Asiatic populations,transferrin D variants seem to be more frequent than the B variants.

Generally, it is suggested that the transferrin D variants are much more frequent in tropical biotopes compared with non-tropical ones. Thus the finding of higher transferrin D variant frequencies than those of B variants in Iranian and neighbouring populations is in agreement with the hypothesis given by Walter and Bajatzadeh(1971) that: The nowadays recognizable geographical distribution of transferrin variants in man reflects the result of selective adaptations to particular environmental conditions . It is hypo-

thesized furthermore, that slowly moving transferrins in man are somehow associated with a better thermoregulation under the conditions of hot biotopes. On the other hand, faster moving transferrins might be of a greater adaptive value in non-tropical areas, indicating by higher $Tf^B$ frequencies in human populations living in biotopes of moderate or arctic climate.

Table 5.2.2. I Transferrin (Tf) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | TfCC | TfCB | TfCD | $Tf^C$ | $Tf^B$ | $Tf^D$ | | |
| 1-Ghashghais | 117 | 111 | - | 6 | 97.44 | 0.00 | 2.56 | Bowman,J.E. | 1964 |
| 2-Moslems.Shiraz | 429 | 409 | 3 | 17 | 97.67 | 0.35 | 1.98 | Bowman,J.E. | 1964 |
| 3-Ghashghais | 548 | 532 | 14 | 2 | 98.55 | 1.27 | 0.18 | Farhud,D.D., & Daneshmand,P. | unpublished |
| 4-Shahsavar | 175 | 171 | - | 4 | 98.86 | 0.00 | 1.14 | Farhud,D.D.,et al. | 1978 |
| 5-Tehran | 228 | 223 | 5 | - | 98.90 | 1.10 | 0.00 | Farhud,D.D. | 1979 |
| 6-Dezfool | 587 | 576 | 5 | 6 | 99.07 | 0.42 | 0.51 | Farhud,D.D.,& Daneshmand,P. | unpublished |
| 7-Turks.Shirvan, Khorasan | 122 | 120 | 1 | 1 | 99.18 | 0.41 | 0.41 | Present study. | |
| 8-Armenians | 258 | 254 | 2 | 2 | 99.24 | 0.38 | 0.38 | Farhud,D.D., & Daneshmand, P. | unpublished |
| 9-Zoroastrians | 145 | 143 | - | 2 | 99.31 | 0.00 | 0.69 | Bowman,J.E. | 1964 |
| 10-Jews | 287 | 283 | 4 | - | 99.31 | 0.69 | 0.00 | Farhud,D.D., & Daneshmand, P. | unpublished |

Table 5.2.2. I(Cont.) Transferrin (Tf) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | TfCC | TfCB | TfCD | $Tf^C$ | $Tf^B$ | $Tf^D$ | | |
| 11-Caspian Sea area | 344 | 340 | 1 | 3 | 99.42 | 0.15 | 0.43 | Farhud,D.D.,et al. | 1978 |
| 12-Esfahan | 89 | 88 | - | 1 | 99.44 | 0.00 | 0.56 | Sawhney,K.S. | 1975 |
| 13-Bandar-Abass | 901 | 893 | 3 | 5 | 99.56 | 0.16 | 0.28 | Farhud,D.D., & Daneshmand, P. | unpublished |
| 14-Zoroastrians | 175 | 174 | - | 1 | 99.71 | 0.00 | 0.29 | Present study. | |
| 15-Kerman | 313 | 312 | - | 1 | 99.84 | 0.00 | 0.16 | Present study. | |
| 16-Tehran | 186 | 186 | - | - | 100.00 | 0.00 | 0.00 | Sawhney,K.S. | 1975 |
| 17-Tehran | 136 | 136 | - | - | 100.00 | 0.00 | 0.00 | Present study. | |
| 18-Zabolis.Sistan & Baluchistan | 118 | 118 | - | - | 100.00 | 0.00 | 0.00 | Present study. | |
| 19-Baluchis.Sistan & Baluchistan | 111 | 111 | - | - | 100.00 | 0.00 | 0.00 | Present study. | |
| 20-Kurds.Shirvan, Khorasan | 112 | 112 | - | - | 100.00 | 0.00 | 0.00 | Present study. | |
| 21-Kurds.Rezaieh | 147 | 147 | - | - | 100.00 | 0.00 | 0.00 | Present study. | |
| 22-Kurds.Sanandaj | 105 | 105 | - | - | 100.00 | 0.00 | 0.00 | Lehmann,H.,et al. | 1973 |

Table 5.2.2.I(Cont.) Transferrin(Tf) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | TfCC | TfCB | TfCD | $Tf^C$ | $Tf^B$ | $Tf^D$ | | |
| 23-Kurdish Jews | 106 | 106 | - | - | 100.00 | 0.00 | 0.00 | Tills,D.,et al. | 1977 |
| 24-Turks.Rezaieh | 149 | 149 | - | - | 100.00 | 0.00 | 0.00 | Present study. | |
| 25-Caspian Sea area | 447 | 447 | - | - | 100.00 | 0.00 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 26-Lurs.Luristan | 178 | 178 | - | - | 100.00 | 0.00 | 0.00 | Present study. | |

Table 5.2.2. III Transferrin (Tf) phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|
| | | TfCC | TfCB | TfCD | $Tf^C$ | $Tf^B$ | $Tf^D$ | |
| 1-Turks | 300 | 300 | - | - | 100.00 | 0.00 | 0.00 | Dinçol, Gunçag., et al. 1976 |
| 2-Kurds | | | | | 100.00 | 0.00 | 0.00 | Richard,P. 1976 |

Table 5.2.2. IV Transferrin (Tf) phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | TfCC | TfCB | TfCD | $Tf^C$ | $Tf^B$ | $Tf^D$ | | |
| 1-Kurdish Jews. North west | 61 | 59 | 2 | - | 98.36 | 1.64 | 0.00 | Tills,D., et al. | 1977 |
| 2-Kurdish Jews. South east | 50 | 50 | - | - | 100.00 | 0.00 | 0.00 | Tills,D., et al. | 1977 |

Table 5.2.2.V Transferrin (Tf) phenotypes and gene frequencies distribution in Kuwait

| Population | Number Tested | Phenotypes | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|
| | | TfCC | TfCB | TfCD | $Tf^C$ | $Tf^B$ | $Tf^D$ | |
| 1-General population | 89 | 89 | - | - | 100.00 | 0.00 | 0.00 | Khaled,E., et al. 1981 |
| 2-Ajman tribe | 52 | 52 | - | - | 100.00 | 0.00 | 0.00 | Khaled,E., et al. 1981 |
| 3-Suluba tribe | 52 | 52 | - | - | 100.00 | 0.00 | 0.00 | Khaled,E., et al. 1981 |

Table 5.2.2. VI Transferrin (Tf) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|
| | | TfCC | TfCB | TfCD | $Tf^C$ | $Tf^B$ | $Tf^D$ | |
| 1-Western Saudi Arabia | 191 | 178 | - | 13 | 96.60 | 0.00 | 3.40 | Saha,N., et al. 1980 |
| 2-Saudi Arabians | 359 | 355 | - | 4(chi) | 99.40 | 0.00 | 0.60 Dchi | Goedde,H.W.,et al. 1979 |

Table 5.2.2. VIII Transferrin (Tf) phenotypes and gene frequencies distribution in Pakistan

| Population | Number Tested | Phenotypes | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|
| | | TfCC | TfCB | TfCD | $Tf^C$ | $Tf^B$ | $Tf^D$ | |
| 1- Pathans | 185 | 183 | 2 | - | 99.50 | 0.50 | 0.00 | Kirk,R.L.,et al. 1961 |
| 2-Punjabis | 207 | 207 | - | - | 100.00 | 0.00 | 0.00 | Kirk,R.L.,et al. 1961 |

Table 5.2.2. IX Transferrin (Tf) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | TfCC | Tf CB | | TfCD | | | | |
| | | | $B_2C$ | $B_1C$ | | $Tf^C$ | $Tf^B$ | $Tf^D$ chi | |
| 1-Hazaras | 172 | 164 | 1 | 1 | 6 chi | 97.68 | 0.59 | 1.73 | Rahimi,A.G.,et al. 1977 |
| 2-Pushtus | 95 | 92 | 3 | | - | 98.40 | 1.60 | 0.00 | Papiha,S.S.,et al. 1977 |
| 3-Tajiks | 310 | 305 | 3 | 1 | 1 chi | 99.19 | 0.64 | 0.17 | Rahimi,A.G.,et al. 1977 |
| 4-Pushtus | 210 | 207 | 2 | - | 1 chi | 99.28 | 0.48 | 0.24 | Rahimi,A.G.,et al. 1977 |
| 5-Daris | 160 | 160 | - | | - | 100.00 | 0.00 | 0.00 | Papiha,S.S.,et al. 1977 |
| 6-Uzbeks | 124 | 124 | - | - | - | 100.00 | 0.00 | 0.00 | Rahimi,A.G.,et al. 1977 |

5.2.3. The third component of human complement (C3)system

The distribution of C3 types and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.2.3.

There are only a few reports of C3 studies in Iranian and neighbouring populations.

The frequency of the $C3^F$ gene ranges from 5.78 to 23.30 percent in Iranians, being lowest in the Kermani series (present investigation ) and highest in the Caspian sea area (Farhud et al, 1978).

On the whole, with an average $C3^F$ frequency of 13.00 percent, the Iranian population appears to exhibit a lower $C3^F$ frequency than that of 21 percent found in Europeans(Goedde et al, 1972).

Since the data available on the distribution of C3 types in various populations of Iran are still not large enough, full discussion is not yet possible.

Regarding neighbouring populations, the frequency of the $C3^F$ gene in the populations of Iraq varies between 19.00 percent in the Arabs of Baghdad and 27.20 percent in the Kurds of Mosul (Roberts and Al-Agidi, 1979). With an average $c3^F$ frequency of 23.30 percent, the population of Iraq seems to show a higher $C3^F$ frequency than that found in Iranians and more similar to the European frequency.

The $C3^F$ frequency of 9.20 percent in Saudi Arabians (Goedde et al, 1979) is lower than the average for Iran and much lower than the frequency in Europe.

The frequency of the $C3^F$ gene in the populations of Afghanistan ranges from 8.72 percent in the Hazaras to 14.03 percent in the Tajiks (Rahimi et al, 1977). With an average

$C3^F$ frequency of 12.11 percent, the Afghan population appears to exhibit a lower $C3^F$ frequency than that found in Iranians and lower than the frequency in Europeans.

Conclusion

The frequency of the $C3^F$ gene ranges from 19 to 23 percent ( average 21 percent) in European populations(Goedde et al, 1972).

$C3^F$ frequency, ranging from 2.5 to 10 percent (average 6.4 percent), is consistently lower in the Indian region(Papiha et al, 1979).

Low frequencies of $C3^F$ have also been observed in Mongoloids and Negroids (Bender, 1975).

In Iranian and neighbouring populations, with the exception of the population of Iraq with their higher $C3^F$ values which are more similar to the European frequencies, the frequency of the $C3^F$ gene seems to be lower than that found in Europeans but higher than that in Indians.

Table 5.2.3.I The third component of human complement (C3) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | C3 SS | C3 SF | C3 FF | F C3 | S C3 | |
| 1-Kerman | 199 | 177 | 21 | 1 | 5.78 | 94.22 | Present study. |
| 2-Kurds.Shirvan, Khorasan | 60 | 53 | 6 | 1 | 6.67 | 93.33 | Present study. |
| 3-Tehran | 51 | 44 | 7 | - | 6.87 | 93.13 | Present study. |
| 4-Turks.Rezaieh | 90 | 77 | 12 | 1 | 7.78 | 92.22 | Present study. |
| 5-Kurds.Rezaieh | 89 | 76 | 12 | 1 | 7.87 | 92.13 | Present study. |
| 6-Turks.Shirvan , Khorasan | 82 | 70 | 11 | 1 | 7.92 | 92.08 | Present study. |
| 7-Zabolis.Sistan & Baluchistan | 109 | 80 | 27 | 2 | 14.22 | 85.78 | Present study. |
| 8-Zoroastrians | 131 | 93 | 34 | 4 | 16.03 | 83.97 | Present study. |
| 9-Lurs.Luristan | 143 | 101 | 37 | 5 | 16.43 | 83.57 | Present study. |
| 10-Iranians | 101 | 66 | 28 | 7 | 20.79 | 79.21 | Farhud,D.D., et al.1973 |
| 11-Baluchis.Sistan & Baluchistan | 107 | 67 | 32 | 8 | 22.43 | 77.57 | Present study. |

Table 5.2.3.I (Cont.) The third component of human complement (C3) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | C3 SS | C3 SF | C3 FF | F C3 | S C3 | |
| 12-Caspian Sea area | 43 | 25 | 16 | 2 | 23.30 | 76.70 | Farhud,D.D.,et al. 1978 |

Table 5.2.3. IV The third component of human complement (C3) phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes C3 SS | C3 SF | C3 FF | Gene frequencies F C3 | S C3 | Authors | |
|---|---|---|---|---|---|---|---|---|
| 1-Arabs.Baghdad | 174 | 119 | 44 | 11 | 19.00 | 81.00 | Roberts,D.E., & Al-Agidi,S.K. | 1979 |
| 2-Arabs.Mosul | 112 | 70 | 35 | 7 | 21.90 | 78.10 | Roberts,D.E., & Al-Agidi,S.K. | 1979 |
| 3-Arabs.Ramadi | 130 | 82 | 39 | 9 | 21.90 | 78.10 | Roberts,D.E., & Al-Agidi,S.K. | 1979 |
| 4-Arabs.Basrah | 158 | 97 | 51 | 10 | 22.50 | 77.50 | Roberts,D.E., & Al-Agidi,S.K. | 1979 |
| 5-Turks.Kirkuk | 120 | 74 | 36 | 10 | 23.30 | 76.70 | Roberts,D.E., & Al-Agidi,S.K. | 1979 |
| 6-Kurds.Baghdad | 145 | 86 | 46 | 13 | 24.80 | 75.20 | Roberts,D.E., & Al-Agidi,S.K. | 1979 |
| 7-Turks.Baghdad | 89 | 51 | 30 | 8 | 25.80 | 74.20 | Roberts,D.E., & Al-Agidi,S.K. | 1979 |
| 8-Kurds.Mosul | 90 | 50 | 31 | 9 | 27.20 | 72.80 | Roberts,D.E., & Al-Agidi,S.K. | 1979 |

Table 5.2.3. VI The third component of human complement (C3) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|
| | | C3 SS | C3 SF | C3 FF | C3 SS0.4 | C3 FS0.4 | F C3 | S C3 | S0.4 C3 | |
| 1- Saudi Arabians | 359 | 292 | 57 | 4 | 5 | 1 | 9.20 | 90.00 | 0.80 | Goedde,H.W.,et al. 1979 |

Table 5.2.3. VIII The third component of human complement (C3) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | C3 SS | C3 SF | C3 FF | C3 SS0.4 SS0.25 FS0.25 | $C3^F$ | $C3^S$ | rare variants C3 | |
| 1-Hazaras | 172 | 142 | 26 | 2 | 2 | 8.72 | 9.69 | 0.59 | Rahimi,A.G.,et al. 1977 |
| 2-Pushtus | 210 | 164 | 42 | 4 | - | 11.91 | 88.09 | 0.00 | Rahimi,A.G.,et al. 1977 |
| 3-Uzbeks | 124 | 89 | 30 | 2 | 3 | 13.80 | 85.00 | 1.20 | Rahimi,A.G.,et al. 1977 |
| 4-Tajiks | 310 | 227 | 75 | 6 | 2 | 14.03 | 85.63 | 0.34 | Rahimi,A.G.,et al. 1977 |

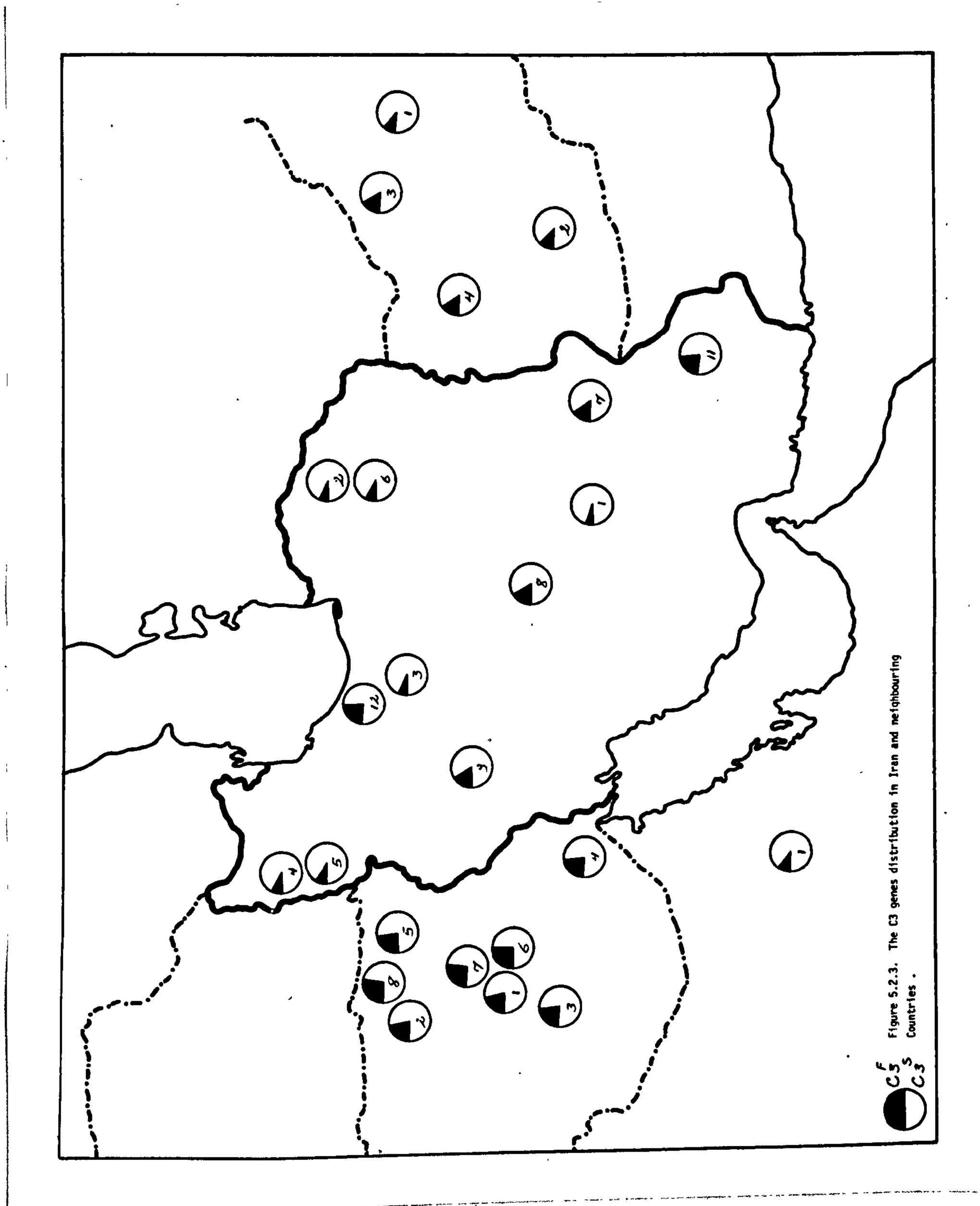



Figure 5.2.3. The C3 genes distribution in Iran and neighbouring Countries .

5.2.4. The group specific component(Gc) system

The distribution of Gc types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.2.4.

The frequency of the allele $Gc^2$ ranges from 19.05 to 37.90 percent in Iranians, being lowest in the Kurdish Jews(Cleve et al, 1962) and highest in the Tehrani series of Bajatzadeh and Walter(1969).

On the whole, with an average $Gc^2$ frequency of 30.40 percent, the Iranian population appears to exhibit a slightly higher $Gc^2$ frequency than that of around 27 percent found in Europeans (Mourant et al, 1976). It is difficult to see any definite regional trend of the distribution of $Gc^2$ in Iran but it tends to be lower in the south than the north.

Regarding neighbouring areas, the $Gc^2$ frequency of 25.73 percent in the Turks of Turkey (Hummel et al, 1970) seems to be lower than the average for Iran and also lower than the European frequency.

The frequency of the $Gc^2$ gene in the populations of Iraq varies between 17.20 percent in the Kurds (Constans et al, 1980) and 24.12 percent in the Jews (Cleve et al, 1962). With an average $Gc^2$ frequency of 20.66 percent, the Iraqi population appears to exhibit a lower $Gc^2$ frequency than that found in Iranians and also lower than the frequency in Europeans. The $Gc^2$ frequency of 24.12 percent in the Iraqi Jews, though lower than the average for Iran, is similar to the frequencies of 24.49 and 24.50 percent in the Iranian Jews (Kitchin and bearn,1964; Cleve et al, 1962). On the whole, it seems that Jewish populations both in Iran and Iraq resemble Europeans in Gc frequencies.

The $Gc^2$ frequency of 14.60 percent in Saudi Arabians(Goedde et al, 1979) appears to be much lower than the Iranian frequency and also much lower than the frequency in Europeans but more similar to the low frequencies in Africans (Mourant et al, 1976).

The frequency of the $Gc^2$ gene in the populations of Pakistan ranges from 19.63 percent in Peshawar (Walter, 1966) to 31.96 percent in the Pathanis (Spedini, 1969). With an average $Gc^2$ frequency of 26.09 percent, the Pakistani populations appears to exhibit a lower $Gc^2$ frequency than that found in Iranians and also lower than that in Europeans.

The $Gc^2$ gene frequency in the Afghan populations varies between 22.68 percent in the Hazaras (Rahimi et al, 1977) and 29.79 percent in the mixed Pakistani sample of Kitchin and Bearn (1964). With an average $Gc^2$ frequency of 24.70 percent, the population of Afghanistan, like that of Pakistan, seems to show a lower $Gc^2$ frequency than that found in Iranians and lower than the European frequency.

Conclusion

In Europe the frequency of the gene $Gc^2$ is nearly always between 20 and 30 percent.

In southern and south-western Asia frequencies are on the whole similar to those in Europe. There is considerable variation in the frequency of $Gc^2$ in India, varying between 10 and 37 percent. In south-east Asia $Gc^2$ frequencies are mostly a little over 20 percent.

The African populations differ considerably from those of Europe and Asia, mostly having less than 10 percent of $Gc^2$ (Mourant et al, 1976).

In Iranian and neighbouring populations, with the excep-

tion of Iranians with their slightly higher frequencies, the frequency of the $Gc^2$ allele appears to be lower than that found in Europeans but higher than that in Africans. The frequency of the gene $Gc^2$ in the Arab population of Saudi Arabia, being much lower than in the other population groups in the area, seems to be more similar to the African frequency.

It has recently been realized as a result of the discovery by Schanfield et al (1975) that the Gc proteins are the vitamin D carriers of the Plasma. Kirk et al (1963), Walter and Steegmuller (1969), Mourant et al (1976) and later Daiger (1979) and Constans(1980) have demonstrated that the frequency of the gene $Gc^2$ follows a cline, diminishing as the mean intensity of solar radiation increases. There is also a north-south gradient in the Gc gene frequencies. This seems to parallel the gradient seen in skin pigmentation. Thus $Gc^1$ has in general, though with several exceptions, a high frequency in sunny climates and $Gc^2$ a relatively high one in dull climates, suggesting that the Gc types are affected by natural selection related to the availability of vitamin D. However, the distribution of the Gc alleles seems to correlate with geographical clines, notably the incidence of solar radiation, as might be expected for the protein which transports vitamin D.

The very low $Gc^2$ frequency in the Arabs of Saudi Arabia, like that in African populations, can perhaps be best explained by the action of geographical factors in relation to the metabolic role of this protein in the organism (Daiger and Cavalli-Sforza, 1977; Constans et al, 1979). These findings confirm the assumption of the presence of a gradient in the frequency of the gene $Gc^2$ which decreases from Europe to equa-

torial Africa. This gradient may be superposed on that of skin pigmentation ( Loomis, 1967) which has always been considered to be linked to genetic and adaptative conditions( Hiernaux, 1977).

Table 5.2.4. I Group specific component (Gc) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | Gc 1-1 | Gc 2-1 | Gc 2-2 | $Gc^1$ | $Gc^2$ | | |
| 1-Kurdish Jews | 42 | 27 | 14 | 1 | 80.95 | 19.05 | Cleve,H.,et al. | 1962 |
| 2- Shiraz | 94 | 55 | 34 | 5 | 76.60 | 23.40 | Walter,H.,& Djahan-shahi,D. | 1963 |
| 3- Jews | 49 | 30 | 14 | 5 | 75.51 | 24.49 | Cleve,H.,et al. | 1962 |
| 4- Jews | 149 | 91 | 43 | 15 | 75.50 | 24.50 | Kitchin,F.D., & Bearn,A.G. | 1964 |
| 5-West | 308 | 135 | 148 | 25 | 67.80 | 32.20 | Bajatzadeh,M., & Walter, H. | 1969 |
| 6-East | 178 | 68 | 84 | 26 | 66.80 | 33.20 | Bajatzadeh,M.,& Walter, H. | 1969 |
| 7-Central and South | 238 | 99 | 115 | 24 | 65.70 | 34.30 | Bajatzadeh,M.,& Walter, H. | 1969 |
| 8-North | 177 | 68 | 95 | 14 | 65.20 | 34.80 | Bajatzadeh,M., & Walter,H. | 1969 |
| 9-Shahsavar | 108 | 44 | 52 | 12 | 64.80 | 35.20 | Farhud,D.D., et al. | 1978 |

Table 5.2.4. I(Cont.) Group specific component (Gc) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | Gc 1-1 | Gc 2-1 | Gc 2-2 | $Gc^1$ | $Gc^2$ | | |
| 10-North west | 245 | 106 | 106 | 33 | 64.60 | 35.40 | Bajatzadeh,M., & Walter, H. | 1969 |
| 11-Tehran | 385 | 144 | 190 | 51 | 62.10 | 37.90 | Bajatzadeh,M., & Walter, H. | 1969 |

Table 5.2.4. III Group specific component (Gc) phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | Gc 1-1 | Gc 2-1 | Gc 2-2 | $Gc^1$ | $Gc^2$ | | |
| 1-Turks | 274 | 148 | 111 | 15 | 74.27 | 25.73 | Hummel,K.,et al. | 1970 |

Table 5.2.4. IV Group specific component (Gc) phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | Gc 1-1 | Gc 2-1 | Gc 2-2 | $Gc^1$ | $Gc^2$ | |
| 1-Kurds | 58 | 40 | 16 | 2 | 82.80 | 17.20 | Constans,G.,et al. 1980 |
| 2-Jews | 85 | 48 | 33 | 4 | 75.88 | 24.12 | Cleve,H.,et al. 1962 |

Table 5.2.4.b. IV Group specific Component(Gc) subtypes in Iraq

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Gc 1F-1F | Gc 1F-1s | Gc 1s-1s | Gc 1A1-1F | Gc 1A1-1s | Gc 2-1F | Gc 2-1s | Gc 1A1-2 | Gc 2-2 |
| Kurds | 58 | 2 | 14 | 24 | 0 | 0 | 8 | 7 | 1 | 2 |

Table 5.2.4.b. IV (Cont.) Group specific component (Gc) subtypes in Iraq

| Population | Number Tested | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | $Gc^{1F}$ | $Gc^{1s}$ | $Gc^{1A1}$ | $Gc^{2}$ | |
| Kurds | 58 | 22.40 | 59.50 | 0.80 | 17.20 | Constans,G., et al. 1980 |

Table 5.2.4. VI Group specific component (Gc) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | Gc 1-1 | Gc 2-1 | Gc 2-2 | $Gc^1$ | $Gc^2$ | |
| 1-Saudi Arabians | 352 | 256 | 89 | 7 | 85.40 | 14.60 | Goedde,H.W.,et al. 1979 |

Table 5.2.4. VIII Group specific component (Gc ) phenotypes and gene frequencies distribution in Pakistan

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | Gc 1-1 | Gc 2-1 | Gc 2-2 | $Gc^1$ | $Gc^2$ | | |
| 1-Peshawar | 135 | 86 | 45 | 4 | 80.37 | 19.63 | Walter, H. | 1966 |
| 2-Pathans.Peshawar | 60 | 36 | 20 | 4 | 76.67 | 23.33 | Kirk,R.L.,et al. | 1963 |
| 3-Punjabis.Lahore | 90 | 47 | 33 | 10 | 70.56 | 29.44 | Kirk,R.L.,et al. | 1963 |
| 4-Pathanis.Swat | 61 | 32 | 19 | 10 | 68.04 | 31.96 | Spedini,Gabriella. | 1969 |

Table 5.2.4. IX Group specific component (Gc) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | Gc 1-1 | Gc 2-1 | Gc 2-2 | $Gc^1$ | $Gc^2$ | | |
| 1-Hazaras | 172 | 107 | 52 | 13 | 77.32 | 22.68 | Rahimi,A.G.,et al. | 1977 |
| 2-Pushtus | 210 | 128 | 67 | 15 | 76.90 | 23.10 | Rahimi,A.G.,et al. | 1977 |
| 3-Uzbeks | 124 | 76 | 38 | 10 | 76.61 | 23.39 | Rahimi,A.G.,et al. | 1977 |
| 4-Tajiks | 310 | 183 | 102 | 25 | 75.48 | 24.52 | Rahimi,A.G.,et al. | 1977 |
| 5-Hazara,Tarjek,Timuri. Kabul | 141 | 72 | 54 | 15 | 70.21 | 29.79 | Kitchin,F.D., & Bearn,A.G. | 1964 |

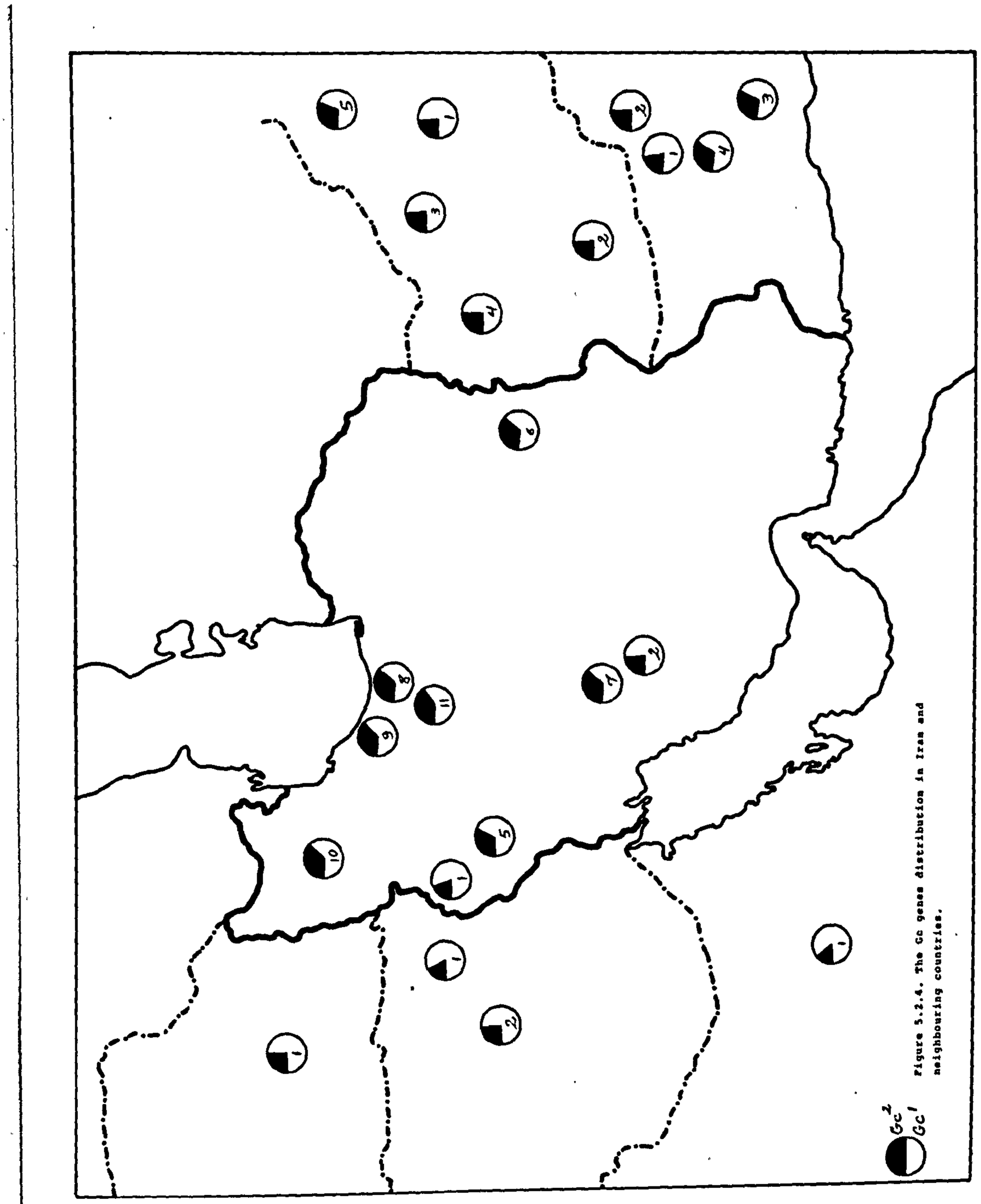



Figure 5.2.4. The Gc genes distribution in Iran and neighbouring countries.

## 5.2.5. The Caeruloplasmin (CP) system

The distribution of Caeruloplasmin groups and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.2.5.

There are few reports of Caeruloplasmin studies in Iranian and neighbouring populations but the results obtained confirm essentially the data given by Shreffler et al (1967/1968), who found very high frequencies of the common $CP^B$ (98-99 percent) and very low frequencies of the $CP^A$ and $CP^C$ alleles in Caucasoids.

### Conclusion

The allele $CP^B$ accounts for about 99 percent of the Caeruloplasmin genes present in European populations and about 94 percent of those in American Negroes. The latter have 5 percent of the $CP^A$ gene.

The other genes have very low frequencies in all the relatively few populations so far examined (Mourant et al,1976).

The Iranian and neighbouring populations, like other Caucasoids, seem also to be characterized by very high $CP^B$ and low $CP^A$ and $CP^C$ frequencies.

Table 5.2.5. I Caeruloplasmin (CP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CP A | CP AB | CP B | CP AC | CP BC | CP C | $CP^A$ | $CP^B$ | $CP^C$ | | |
| 1-Caspian Sea area | 99 | - | - | 99 | - | - | - | 0.00 | 100.00 | 0.00 | Farhud,D.D.,et al. | 1978 |
| 2-Caspian Sea area | 448 | - | 2 | 442 | - | 4 | - | 0.23 | 99.33 | 0.44 | Kirk,R.L., et al. | 1977 |
| 3-Iranians | 198 | - | 1 | 195 | - | 1 | 1 | 0.25 | 98.99 | 0.76 | Bajatzadeh,M.,& Walter, H. | 1969 |
| 4-Shahsavar | 134 | - | 5 | 127 | - | 2 | - | 1.80 | 97.40 | 0.80 | Farhud,D.D.,et al. | 1978 |

Table 5.2.5. VI Caeruloplasmin (CP) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CP A | CP AB | CP B | CP AC | CP BC | CP C | $CP^A$ | $CP^B$ | $CP^C$ | |
| I-Western Saudi Arabia | 185 | - | - | 185 | - | - | - | 0.00 | 100.00 | 0.00 | Saha,N.,et al. 1980 |

Table 5.2.5. VIII Caeruloplasmin(CP) phenotypes and gene frequencies distribution in Pakistan

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CP A | CP AB | CP B | CP AC | CP BC | CP C | $CP^A$ | $CP^B$ | $CP^C$ | | |
| 1-Pakistanis | 96 | - | 1 | 95 | - | - | - | 0.52 | 99.48 | 0.00 | Bajatzadeh,M.,& Walter, H. | 1969 |

Table 5.2.5. IX Caeruloplasmin (CP) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | CP A | CP AB | CP B | CP AC | CP BC | CP C | $CP^A$ | $CP^B$ | $CP^C$ | | |
| 1-Tajiks | 310 | - | - | 310 | - | - | - | 0.00 | 100.00 | 0.00 | Rahimi,A.G.,et al. | 1977 |
| 2-Uzbeks | 124 | - | - | 124 | - | - | - | 0.00 | 100.00 | 0.00 | Rahimi,A.G.,et al. | 1977 |
| 3-Pushtus | 210 | - | - | 210 | - | - | - | 0.00 | 100.00 | 0.00 | Rahimi,A.G.,et al. | 1977 |
| 4-Hazaras | 172 | - | - | 172 | - | - | - | 0.00 | 100.00 | 0.00 | Rahimi,A.G.,et al. | 1977 |

## 5.2.6. The ∝ 1-antitrypsin (Pi) system

The distribution of ∝ 1-antitrypsin types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.2.6.

There are few reports of ∝ 1-antitrypsin studies in Iranian and neighbouring populations.

The Iranian sample consists of three population groups. Comparing the three groups, the lowest frequency of the commonest $Pi^M$ gene is 88.38 percent in the Mixed Iranian sample of Kellermann and Walter(1970) and the highest, 99.64 percent in the Caspian sea area(Kirk et al, 1977).

The mixed Iranian sample of Kellermann and Walter(1970) and the Tehrani series of Daneshmand (1979)show the presence of the $Pi^F$ and $Pi^S$ genes with polymorphic frequencies but in the Caspian sea sample of Kirk et al (1977) the $Pi^F$ and $Pi^S$ genes show very low frequencies.

The $Pi^Z$ gene is only present with a frequency of 2.21 percent in the mixed Iranian sample of Kellermann and Walter (1970)and totally absent in other Iranian samples.

On the whole, as in Europeans, the $Pi^F$ gene seems to be more frequent than the $Pi^S$ in Iranians.

Regarding neighbouring populations, the only report from Saudi Arabia is that of Goedde et al (1979). The most common $Pi^M$ gene has a frequency of 98.30 percent in Saudi Arabians. Unlike that in Iranians, the $Pi^S$ gene seems to be more frequent than the $Pi^F$ in Saudi Arabians. The $Pi^Z$ gene is present with a frequency of 0.70 percent which is much lower than that of 2.21 percent in the mixed Iranian sample of Kellermann and Walter (1970). On the whole the Pi variants other than $Pi^M$ seem to be less frequent in Saudi Arabians than in Iranians.

The only report on the population of Pakistan(Kellermann and Walter, 1970) shows a $Pi^M$ gene frequency of 95.28 percent. This is an intermediate frequency compared with those found in Iranians. Like that in Iranians, the $Pi^F$ gene with a frequency of 3.78 percent appears to be the most common Pi variant other than $Pi^M$ in the Pakistani population. The $Pi^S$ is totally absent and the $Pi^Z$ is present with a low frequency of 0.94 percent.

The afghan sample consists of four population groups(Rahimi et al, 1977). The frequency of the $Pi^M$ gene ranges from 95.36 percent in the Hazaras to 99.59 percent in the Uzbeks. The $Pi^S$ gene seems to be more frequent than the $Pi^F$ gene in Afghanistan and appears to be the most common Pi variant other than $Pi^M$. The third common variant seems to be $Pi^Z$. This contrast with the findings in Iranian and other neighbouring populations.

Conclusion

The commonest Pi allele, $Pi^M$ has a frequency of at least 85 percent in all populations tested, and over 97 percent in Negroes and Mongoloids examined, as well as in some Indian populations.

The commonest variant, $Pi^F$ reaches a level of 11 percent in Ireland, only 1.3 percent in Norway, 7 percent in Germany and Hungry, but is rare or absent in most European populations other than those mentioned (Mourant et al, 1976).

In Iranian and neighbouring populations, the allele $Pi^M$ has a frequency of at least 88.38 percent. The commonest variant, $Pi^F$ reaches a level of 6.83 percent in Iranians and 3.78 percent in Pakistanis but is rare or absent in Afghan and Saudi Arabian populations as it is in most Europeans.

The other Pi variants are absent or very rare.

In view of the known pathological association of the deficient variant ($Pi^Z$) with pulmonary emphysema ( Laurell and Eriksson, 1963) and Cirrhosis( Sharp, 1970), and the marked variations in frequency of the electrophoretically detectable alleles, this system merits further application both anthropologically and in disease surveys.

Table 5.2.6. I ∝1-antitrypsin(Pi) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Pi MM | Pi MS | Pi MF | Pi MZ | Pi MI | Pi SS | Pi SF | Pi FF | Pi ZZ |
| 1-Iranians | 271 | 218 | 7 | 28 | 6 | 2 | 1 | 3 | 3 | 3 |
| 2-Tehran | 143 | 122 | 8 | 11 | - | - | 1 | 1 | - | - |
| 3-Caspian Sea area | 412 | 409 | 2 | 1 | - | - | - | - | - | - |

Table 5.2.6. I (Cont.) ∝ 1-antitrypsin (Pi) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Gene frequencies | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | $Pi^M$ | $Pi^S$ | $Pi^F$ | $Pi^Z$ | $Pi^i$ | | |
| 1-Iranians | 271 | 88.38 | 2.21 | 6.83 | 2.21 | 0.37 | Kellermann,G., & Walter, H. | 1970 |
| 2-Tehran | 143 | 91.95 | 3.85 | 4.20 | 0.00 | 0.00 | Daneshmand,P. | 1979 |
| 3-Caspian Sea area | 412 | 99.64 | 0.24 | 0.12 | 0.00 | 0.00 | Kirk,R.L.,et al. | 1977 |

Table 5.2.6. VI ∝ 1- antitrypsin (Pi) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Pi MM | Pi MS | Pi MF | Pi MZ | Pi MI | Pi SS | Pi SF | Pi FF | Pi ZZ |
| 1-Saudi Arabians | 357 | 345 | 7 | - | 5 | - | - | - | - | - |

Table 5.2.6. VI (Cont.) ∝1- antitrypsin (Pi) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Gene frequencies | | | | | Authors |
|---|---|---|---|---|---|---|---|
| | | $Pi^M$ | $Pi^S$ | $Pi^F$ | $Pi^Z$ | $Pi^i$ | |
| 1-Saudi Arabians | 357 | 98.30 | 1.00 | 0.00 | 0.70 | 0.00 | Goedde,H.W.,et al. 1979 |

Table 5.2.6.b. VI  Alpha$_1$-antitrypsin(Pi) subtypes in Saudi Arabia

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Pi $M_1M_1$ | Pi $M_1M_3$ | Pi $M_3M_3$ | Pi $M_1S$ | Pi $M_3S$ | Pi SS | Pi $M_1Z$ | Pi $M_3Z$ | Pi ZZ |
| Saudi Arabians | 178 | 112 | 52 | 10 | 1 | 1 | - | 1 | 1 | - |

Table 5.2.6.b. VI (Cont.) Alpha$_1$-antitrypsin (Pi) subtypes in Saudi Arabia

| Population | Number Tested | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | $M_1$ | $M_3$ | S | Z | |
| Saudi Arabians | 178 | 78.00 | 21.00 | 0.50 | 0.50 | Goedde,H.W.,et al. 1980 |

Table 5.2.6. VIII ∝ 1-antitrypsin(Pi) phenotypes and gene frequencies distribution in Pakistan

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Pi MM | Pi MS | Pi MF | Pi MZ | Pi MI | Pi SS | Pi SF | Pi FF | Pi ZZ |
| 1- Pakistanis | 53 | 49 | - | 2 | 1 | - | - | - | 1 | - |

Table 5.2.6. VIII (Cont.) ∝1- antitrypsin (Pi) phenotypes and gene frequencies distribution in Pakistan

| Population | Number Tested | Gene frequencies | | | | | Authors |
|---|---|---|---|---|---|---|---|
| | | $Pi^M$ | $Pi^S$ | $Pi^F$ | $Pi^Z$ | $Pi^i$ | |
| 1-Pakistanis | 53 | 95.28 | 0.00 | 3.78 | 0.94 | 0.00 | Kellermann,G., & Walter, H. 1970 |

Table 5.2.6. IX  $\alpha$ 1- antitrypsin(Pi) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Pi MM | Pi MS | Pi MF | Pi MZ | Pi MI | Pi SS | Pi SF | Pi FF | Pi ZZ |
| 1-Hazaras | 172 | 156 | 10 | 3 | 3 | - | - | - | - | - |
| 2-Pushtus | 210 | 196 | 4 | 1 | 8 | - | 1 | - | - | - |
| 3-Tajiks | 310 | 291 | 4 | 1 | 14 | - | - | - | - | - |
| 4-Uzbeks | 124 | 123 | 1 | - | - | - | - | - | - | - |

Table 5.2.6. IX (Cont.) ∝1-antitrypsin (Pi) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Gene frequencies | | | | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | $Pi^M$ | $Pi^S$ | $Pi^F$ | $Pi^Z$ | $Pi^i$ | | |
| 1-Hazaras | 172 | 95.36 | 2.90 | 0.87 | 0.87 | 0.00 | Rahimi,A.G.,et al. | 1977 |
| 2-Pushtus | 210 | 96.44 | 1.42 | 0.23 | 1.91 | 0.00 | Rahimi,A.G.,et al. | 1977 |
| 3-Tajiks | 310 | 96.94 | 0.64 | 0.16 | 2.26 | 0.00 | Rahimi,A.G.,et al. | 1977 |
| 4-Uzbeks | 124 | 99.59 | 0.41 | 0.00 | 0.00 | 0.00 | Rahimi,A.G.,et al. | 1977 |

Table 5.2.6.b. IX Alpha$_1$-antitrypsin (Pi) subtypes in Afghanistan

| Population | Number Tested | Phenotypes | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | Pi $M_1M_1$ | Pi $M_1M_3$ | Pi $M_3M_3$ | Pi $M_1S$ | Pi $M_3S$ | Pi SS | Pi $M_1Z$ | Pi $M_3Z$ | Pi ZZ |
| 1-Hazaras | 135 | 90 | 40 | 5 | - | - | - | - | - | - |
| 2-Pushtus | 167 | 118 | 39 | 8 | 1 | - | - | 1 | - | - |
| 3-Tajiks | 232 | 171 | 50 | 9 | 1 | - | - | 1 | - | - |
| 4-Uzbeks | 153 | 127 | 26 | - | - | - | - | - | - | - |

Table 5.2.6.b. IX (Cont.) Alpha$_1$-antitrypsin(Pi) subtypes in Afghanistan

| Population | Number Tested | Gene frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|
| | | $M_1$ | $M_3$ | S | Z | | |
| 1-Hazaras | 135 | 81.50 | 18.50 | 0.00 | 0.00 | Goedde,H.W.,et al. | 1980 |
| 2-Pushtus | 167 | 82.90 | 16.50 | 0.30 | 0.30 | Goedde,H.W.,et al. | 1980 |
| 3-Tajiks | 232 | 84.90 | 14.70 | 0.20 | 0.20 | Goedde,H.W.,et al. | 1980 |
| 4-Uzbeks | 153 | 91.50 | 8.50 | 0.00 | 0.00 | Goedde,H.W.,et al. | 1980 |

## 5.2.7. The Pseudocholinesterase($E_1$ locus ) system

The distribution of Pseudocholinesterase types and respective gene frequencies in Iranian and neighbouring populations is shown in Tables 5.2.7.

The frequency of the $E_1^a$ gene ranges from zero to 5.12 percent in Iranians, being lowest in the Kurds of Sanandaj (Lehmann et al, 1973) and highest in the Jews(Szeinberg et al, 1966). Apart from the Jews with their exceptionally high $E_1^a$ frequency of 5.12 percent, the Iranian population appears to exhibit $E_1^a$ frequencies similar to those varying between 1 and 2 percent in Europeans (Mourant et al, 1976).

Regarding neighbouring populations, the $E_1^a$ gene frequencies in the populations of Turkey varying between 1.64 percent in the Jews (Szeinberg et al, 1966) and 3.31 percent in the Turks of Ankara (Sayek et al, 1967) seem to be slightly higher than those found in Europeans.

Frequencies of the $E_1^a$ gene in the populations of Pakistan ranging from 1.24 percent in Peshawar ( Neumann and Walter, 1968) to 3.20 percent in the Punjabis (Singh et al, 1971) appear also to be slightly higher than in Europeans.

The $E_1^a$ gene frequencies in the populations of Afghanistan varying between zero in both the Uzbeks and Pushtus and 0.59 percent in the Hazaras (Rahimi et al, 1977) seem to be below the European level.

Conclusion

In nearly all European populations the frequency of the $E_1^a$ gene lies between 1 and 2 percent, but somewhat higher frequencies around 3 percent are found in Czechoslovakia and Greece

Jews in general show similar frequencies to Europeans, but those of the Yemen have 3.6 percent.

In the Indian region the frequency of $E_1^a$ is about 1.5 percent, but in Africa and the Far East is only a few per mille.

The frequency of $E_1^f$, where ascertained, is usually about 0.5 percent in Europe ( Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Irani Jews with their exceptionally high $E_1^a$ frequency (5.12), frequencies of this gene seem to be similar to those in Europeans. The Turks of Turkey with somewhat higher $E_1^a$ frequency ( around 3 percent) appear to be more similar to the population of Greece.

Table 5.2.7.I. Pseudocholinesterase $E_1$ phenotypes and gene frequencies distribution in Iran

| Populations | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | U | I | A | $E_1^u$ | $E_1^a$ | | |
| 1-Kurds.Sanandaj | 107 | 107 | 0 | 0 | 100.00 | 0.00 | Lehmann,H., et al. | 1973 |
| 2- Kurds.Baneh Marivan | 77 | 77 | 0 | 0 | 100.00 | 0.00 | Lehmann,H., et al. | 1973 |
| 3- Arabs | 36 | 35 | 1 | 0 | 98.61 | 1.39 | Whittaker, M. | 1968 |
| 4- Jews | 381 | 343 | 37 | 1 | 94.88 | 5.12 | Szeinberg,A.,et al. | 1966 |

Table 5.2.7. III Pseudocholinesterase $E_1$ phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | U | I | A | UF | IF | S | $E_1^u$ | $E_1^a$ | $E_1^f$ | $F_1^s$ | |
| 1- Jews | 214 | 207 | 7 | 0 | 0 | 0 | 0 | 98.36 | 1.64 | 0.00 | 0.00 | Szeinberg,A., et al. 1966 |
| 2- Turks.Ankara | 725 | 675 | 43 | 1 | 3 | 3 | 0 | 96.28 | 3.31 | 0.41 | 0.00 | Sayek,I., et al. 1967 |

Table 5.2.7. VIII Pseudocholinesterase $E_1$ phenotypes and gene frequencies distribution in Pakistan

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | U | I | A | $E_1^u$ | $E_1^a$ | |
| 1-Peshawar | 121 | 118 | 3 | 0 | 98.76 | 1.24 | Neumann,S., and Walter,H. 1968 |
| 2-Punjabis | 202 | | | | 96.80 | 3.20 | Singh,S.,et al. 1971 |

Table 5.2.7. IX Pseudocholinesterase $E_1$ phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | U | UA | A | $E_1^u$ | $E_1^a$ | | |
| 1-Pushtus | 210 | 210 | 0 | 0 | 100.00 | 0.00 | Rahimi,A.G., et al. | 1977 |
| 2-Uzbeks | 124 | 124 | 0 | 0 | 100.00 | 0.00 | Rahimi,A.G., et al. | 1977 |
| 3-Tajiks | 310 | 308 | 2 | 0 | 99.67 | 0.33 | Rahimi,A.G., et al. | 1977 |
| 4-Hazaras | 172 | 170 | 2 | 0 | 99.41 | 0.59 | Rahimi,A.G., et al. | 1977 |

## 5.2.8. Other serum protein systems

In this part, data on some other serum protein systems are presented. Since data available on these systems are not representative for the region, no discussion will be given.

Table 5.2.8. a.I. Albumin types in Iran

| Population | Number Tested | Common type | Variant | Authors | |
|---|---|---|---|---|---|
| Caspian Sea area | 448 | 448 | - | Kirk,R.L., et al. | 1977 |

Table 5.2.8.a. VI . Albumin types in Saudi Arabia

| Population | Number Tested | Common type | Variant | Authors | |
|---|---|---|---|---|---|
| Western Saudi Arabia | 191 | 191 | - | Saha,N., et al. | 1980 |

Table 5.2.8.b. VI Properdin factor B(Bf) types in Saudi Arabia

| Population | Number Tested | Phenotypes | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Bf SS | Bf FS | Bf FF | Bf $SS_1$ | Bf $FS_1$ | Bf $F_1S$ | Bf $FF_1$ | Bf $F_1S_1$ |
| Saudi Arabians | 246 | 68 | 78 | 62 | 13 | 15 | 6 | 3 | 1 |

| Population | Number Tested | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|
| | | $Bf^S$ | $Bf^F$ | $Bf^F1$ | $Bf^S1$ | |
| Saudi Arabians | 246 | 47.40 | 44.70 | 2.00 | 5.90 | Goedde, H.W., et al. 1979 |

Table 5.2.8. C.I. Betalipoprotein (Ag) types in Iran:tested with anti-$Ag^X$

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Ag(x+) | Ag(x-) | $Ag^x$ | $Ag^y$ | |
| Kurdish Jews | 19 | 12 | 7 | | | Tills,D.,et al. 1977 |

Table 5.2.8. C. IV Betalipoprotein (Ag) types in Iraq: tested with anti-$Ag^X$

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Ag(x+) | Ag(X-) | $Ag^x$ | $Ag^y$ | |
| Kurdish Jews North west | 41 | 21 | 20 | 30.16 | 69.48 | Tills, D., et al. 1977 |

Table 5.2.8.d.I. Betalipoprotein (Ag) types in Iran: tested with C. de B serum

| Population | Number Tested | Ag(+) | Ag(-) | Ag(+)% | Authors | |
|---|---|---|---|---|---|---|
| Jews | 37 | 26 | 11 | 70.27 | Blumberg, B.S. | 1963 |
| Kurdish Jews | 17 | 12 | 5 | 70.59 | Blumberg, B.S. | 1963 |

Table 5.2.8.e. I. Betalipoprotein (LP) types in Iran

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | LP(a+) | LP(a-) | $LP^a$ | LP | |
| Kurdish Jews | 19 | 12 | 7 | | | Tills,D., et al. 1977 |

Table 5.2.8.e.IV Betalipoprotein (LP) types in Iraq

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | LP(a+) | LP(a-) | $LP^a$ | LP | | |
| 1-Kurdish Jews. North west | 41 | 14 | 27 | 18.85 | 81.15 | Tills, D.,et al. | 1977 |
| 2-Kurdish Jews. South west | 41 | 20 | 21 | 28.43 | 71.57 | Tills, D.,et al. | 1977 |

Table 5.2.8.e. VIII. Betalipoprotein (LP) types in Pakistan

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | LP(a+) | LP(a-) | $LP^a$ | LP | | |
| Pakistanis | 125 | 40 | 85 | 17.54 | 82.46 | Wassenich,W.K., & Walter, H. | 1968 |

Table 5.2.8.f.IX Beta$_2$ - Glycoprotein (Bg) types in Afghanistan

| Population | Number Tested | Phenotypes | | | Gene frequencis | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | Bg NN | Bg DN | Bg DD | N Bg | D Bg | | |
| 1-Hazaras | 172 | 112 | 60 | - | 82.55 | 17.45 | Rahimi,A.G.,et al. | 1977 |
| 2-Pushtus | 210 | 160 | 48 | 2 | 87.61 | 12.39 | Rahimi,A.G.,et al. | 1977 |
| 3-Tajiks | 310 | 242 | 62 | 6 | 88.06 | 11.94 | Rahimi,A.G.,et al. | 1977 |
| 4-Uzbeks | 124 | 97 | 26 | 1 | 88.70 | 11.30 | Rahimi,A.G.,et al. | 1977 |

Table 5.2.8.g.IV. Pseudocholinesterase $E_2$ types in Iraq

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | $C_5+$ | $C_5-$ | $E_2^+$ | $E_2^-$ | | |
| Jews | 64 | - | 64 | 0.00 | 100.00 | Robson,E.B.,& Harris,H. | 1966 |

## 5.3. Red cell enzymes

### 5.3.1. The acid Phosphatase (AcP) system

The distribution of acid phosphatase types and respective allele frequencies in Iranian and neighbouring populations is shown in Tables 5.3.1.

The frequency of the $AcP^A$ gene ranges from 21.01 to45.50 percent in Iranians, being lowest in the Zoroastrians (present study) and highest in northern Gorgan (Kirk et al, 1977), that of the $AcP^B$ gene varies between 53.40 percent in northern Gorgan(Kirk et al, 1977) and 77.73 percent in the Zoroastrians (present investigation). The rarest $AcP^C$ allele is present in most Iranian populations studied with frequencies ranging from 0.45 percent in the Baluchis of Sistan and Baluchistan (present study) to 4.26 percent in the Kurdish Jews (Godber et al, 1973).

Both the Turkish and the Kurdish series of Rezaieh(present investigation) and also two population groups from the Caspian sea area ( Kirk et al, 1977) show an absence of this gene.With the exception of the Zoroastrians and the Kurds of Shirvan, Khorasan values obtained in the present investigation are within the range of variation.

On the whole, with an average $AcP^A$ frequency of 31.97 percent, an $AcP^B$ frequency of 66.18 percent and an $AcP^C$frequency of 1.85 percent, the Iranian population appears to exhibit a higher $AcP^B$ but a lower $AcP^C$ frequency than the average frequencies of about $AcP^A$ 32, $AcP^B$ 61 and $AcP^C$ 6 percent found in Europeans (Mourant et al,1976). From the table it seems that the frequencies of the alleles $AcP^A$and $AcP^C$ decrease but that of the $AcP^B$ allele increases from west to east in Iran.

The Kurdish Jews, like the Jews themselves, both in Iran and

in Iraq appear to exhibit higher $AcP^A$ and $AcP^C$ but lower $AcP^B$ frequencies than the regional averages.

The Iranian Zoroastrians seem to be characterized by lower $AcP^A$ but higher $AcP^B$ frequencies than the averages for the region.

Relatively higher $AcP^A$ but lower $AcP^B$ and $AcP^C$ frequencies than the regional averages appear to be characteristic of the populations of the Caspian sea area of Iran.

Regarding neighbouring groups, the frequency of the $AcP^A$ gene in the populations of Turkey ranges from 29.20 percent in the Turks (Hummel et al, 1970) to 35.00 percent in the Kurds (Richard, 1976), that of the $AcP^B$ allele varies between 62.00 percent in the Kurdish sample of Richard (1976) and 68.06 percent in the Turkish series of Hummel et al (1970). The rarest $AcP^C$ allele is present in both samples with frequencies ranging from 2.74 percent in the Turks to 3.00 percent in the Kurds. With an average $AcP^A$ frequency of 32.10 percent, an $AcP^B$ frequency of 65.03 percent and an $AcP^C$ frequency of 2.87 percent, the population of Turkey, like that of Iran, seems to show a higher $AcP^B$ but a lower $AcP^C$ frequency than those found in Europeans.

The frequency of the $AcP^A$ gene in the populations of Iraq ranges from zero in the Karaite Jews (Goldschmidt et al, 1976) to 40.98 percent in the Kurdish Jews of north western Iraq(Tills et al, 1977), that of the $AcP^B$ allele varies between 54.92 percent in the north western Kurdish Jews series of Tills et al (1977) and 96.50 percent in the Karaite Jews sample of Goldschmidt et al (1976). The frequency of the rarest $AcP^C$ gene ranges from zero in the Kurdish Jews of south eastern Iraq(Tills et al, 1977) to 7.41 percent in the Kurdish Jews series of

Godber et al (1973). With an average $AcP^A$ frequency of 28.66 percent, an $AcP^B$ frequency of 67.97 percent and an $AcP^C$ frequency of 3.37 percent, the Jewish population of Iraq, though with slightly lower $AcP^A$ but higher $AcP^B$ and $AcP^C$ frequencies than those found in Iranians, again appears to exhibit higher $AcP^B$ but lower $AcP^C$ frequencies than those found in Europeans. The complete absence of the allele $AcP^A$ and a correspondingly high frequency of $AcP^B$ (96.50 percent) in the Karaite Jews of Iraq clearly indicate the strong influence of isolation and genetic drift operating in this community.

The frequency of the $AcP^A$ gene in the populations of Kuwait ranges from 16.00 percent in the Suluba tribe (Khaled et al, 1981) to 20.32 percent in the Kuwaiti Arabs (Sawhney,1975), that of the $AcP^B$ allele varies between 77.74 percent in the Kuwaiti Arabs sample of Sawhney (1975) and 84.00 percent in the Suluba tribe series of Khaled et al (1981). The rarest $AcP^C$ gene is present only in the Kuwaiti Arabs series of Sawhney (1975) with a frequency of 1.94 percent, the other Kuwaiti samples investigated showing complete absence of this gene. With an average $AcP^A$ frequency of 18.58 percent, an $AcP^B$ frequency of 80.94 percent and an $AcP^C$ frequency of 0.48 percent, the population of Kuwait seems to show much lower $AcP^A$ and $AcP^C$ but higher $AcP^B$ frequencies than those found in Iranians.

The frequency of the $AcP^A$ gene in the populations of Saudi Arabia ranges from 9.79 percent in western Saudi Arabia(Saha et al, 1980) to 32.30 percent in Saudi Arabians (Goedde et al, 1979), that of the $AcP^B$ allele varies between 66.70 percent in the Saudi Arabians sample of Goedde et al (1979) and 84.62 percent in western Saudi Arabian series of Saha et al (1980).The rarest $AcP^C$ gene is present in both the

Saudi Arabian samples with frequencies varying between 1.00 percent in the Saudi Arabians sample of Goedde et al (1979) and 5.59 percent in the western Saudi Arabian series of Saha et al (1980). With an average $AcP^A$ frequency of 21.05 percent, an $AcP^B$ frequency of 75.66 percent and an $AcP^C$ frequency of 3.29 percent, the Arab population of Saudi Arabia, like that of Kuwait, appears to exhibit much lower $AcP^A$ but higher $AcP^B$ frequencies than those found in Iranians. The frequency of the $AcP^C$ allele seems to be higher in Saudi Arabians than in Kuwaiti Arabs and also in Iranians but, is still lower than the European frequency.

The frequency of the $AcP^A$ gene in the populations of Afghanistan ranges from 26.82 percent in the Pushtus to 37.09 percent in the Uzbeks (Goedde et al, 1977), that of the $AcP^B$ gene varies between 62.50 percent in the Uzbeks and 72.46 percent in the Pushtus (Goedde et al, 1977). The rarest $AcP^C$ allele is present in all the Afghan samples with frequencies ranging from 0.28 percent in Hazaras ( Goedde et al,1977)to 2.80 percent in the Daris (Papiha et al, 1977). With an average $AcP^A$ frequency of 30.18 percent, an $AcP^B$ frequency of 68.90 percent and an $AcP^C$ frequency of 0.92 percent, the population of Afghanistan with higher $AcP^B$ and lower $AcP^C$ frequencies than in Iranians, also seems to show higher $AcP^B$ but lower $AcP^C$ values than those found in Europeans.

## Conclusion

The average frequencies of the three AcP alleles in European populations are about 32, 61, and 6 percent respectively, with the frequency of $AcP^A$ falling and that of $AcP^B$ rising from north west to south east.

Negroes have about 20, 79 and 1 percent respectively of these

three alleles.

Asiatic populations vary considerably, they exhibit an $AcP^A$ average of about 30 percent and an $AcP^B$ of 70 percent,with very low frequencies of $AcP^C$( Mourant et al, 1976).

Iranian and neighbouring populations, like other Asiatic populations, seem to be characterized by lower $AcP^A$ and $AcP^C$ but higher $AcP^B$ frequencies than those found in Europeans. The frequencies of the three AcP alleles in the Arab populations of Kuwait and Saudi Arabia, with much lower $AcP^A$ but higher $AcP^B$ frequencies, appear to be more like those found in Africans.

The high incidence of the allele $AcP^B$ in Iranian and neighbouring populations agrees with the suggestion of a possible correlation of high $AcP^B$ frequency and increasing mean annual temperature ( Walter and Bajatzadeh, 1968; Wysloúchowa, 1970; Ananthakrishnan and Walter, 1972).

Table 5.3.1.I Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | AcP A | AcP BA | AcP B | AcP CA | AcP CB | AcP C | $AcP^A$ | $AcP^B$ | $AcP^C$ | | |
| 1-Zoroastrians | 119 | 4 | 42 | 70 | - | 3 | - | 21.01 | 77.73 | 1.26 | Present study. | |
| 2-Kurds.Shirvan,Khorasan | 101 | 8 | 28 | 63 | - | 2 | - | 21.78 | 77.23 | 0.99 | Present study. | |
| 3-Armenians | 180 | | | | | | | 24.70 | 72.10 | 3.20 | Simhai,B. | 1978 |
| 4-North west | 59 | 5 | 18 | 32 | 2 | 2 | - | 25.50 | 71.10 | 3.40 | Walter,H., & Bajatzadeh, M. | 1968 |
| 5-Lurs.Luristan | 178 | 16 | 61 | 94 | 2 | 5 | - | 26.69 | 71.35 | 1.96 | Present study. | |
| 6-Turks.Shirvan,Khorasan | 115 | 11 | 40 | 62 | - | 2 | - | 26.96 | 72.17 | 0.87 | Present study. | |
| 7-Tavalesh,Astara | 61 | 5 | 23 | 32 | - | 1 | - | 27.10 | 72.10 | 0.80 | Kirk,R.L.,et al. | 1977 |
| 8-Esfahan | 77 | 6 | 30 | 39 | - | 2 | - | 27.27 | 71.43 | 1.30 | Sawhney, K.S. | 1975 |
| 9-North | 54 | 6 | 17 | 27 | 2 | 2 | - | 28.80 | 67.40 | 3.80 | Walter,H., & Bajatzadeh,M. | 1968 |
| 10-Turks . Rezaieh | 125 | 12 | 48 | 65 | - | - | - | 28.80 | 71.20 | 0.00 | Present study. | |
| 11-Babol,Shahi,Amol | 64 | 3 | 31 | 30 | - | - | - | 28.90 | 71.10 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 12-West | 95 | 16 | 23 | 51 | 2 | 3 | - | 30.00 | 67.40 | 2.60 | Walter,H.,& Bajatzadeh, M. | 1968 |

Table 5.3.1.I (Cont) Red cell Acid phosphatase(AcP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Acp A | AcP BA | AcP B | AcP CA | AcP CB | AcP C | $AcP^A$ | $AcP^B$ | $AcP^C$ | | |
| 13-East | 58 | 7 | 20 | 28 | 1 | 2 | - | 30.30 | 67.20 | 2.50 | Walter,H., & Bajatzadeh, M. | 1968 |
| 14-Central and South | 92 | 11 | 32 | 43 | 3 | 3 | - | 30.90 | 65.70 | 3.40 | Walter,H., & Bajatzadeh,M. | 1968 |
| 15-Kurds.Sanandaj | 105 | 13 | 41 | 48 | - | 3 | - | 31.90 | 66.67 | 1.43 | Lehmann,H.,et al. | 1973 |
| 16-Kerman | 307 | 31 | 133 | 134 | 4 | 5 | - | 32.41 | 66.12 | 1.47 | Present study. | |
| 17-Tehran | 91 | 16 | 27 | 42 | 3 | 3 | - | 34.10 | 62.70 | 3.20 | Walter,H., & Bajatzadeh,M. | 1968 |
| 18-Tehran | 161 | 21 | 68 | 65 | - | 7 | - | 34.16 | 63.67 | 2.17 | Sawhney,K.S. | 1975 |
| 19-Jews | 164 | | | | | | | 34.20 | 63.00 | 2.80 | Simhai,B. | 1974 |
| 20-Kurds.Baneh,Marivan | 77 | 8 | 35 | 32 | 2 | - | - | 34.41 | 64.29 | 1.30 | Lehmann,H.,et al. | 1973 |
| 21-Kurdish Jews | 94 | 11 | 41 | 34 | 2 | 6 | - | 34.57 | 61.17 | 4.26 | Godber,Marilyn.,et al.. | 1973 |
| 22-Tehran | 352 | 49 | 145 | 146 | 1 | 11 | - | 34.66 | 63.64 | 1.70 | Present study. | |
| 23-Kurdish Jews | 131 | 19 | 53 | 55 | 1 | 3 | - | 35.11 | 63.36 | 1.53 | Goldschmidt,Elizabeth.,et al. | 1967 |
| 24-Zabolis.Sistan & Baluchistan | 117 | 19 | 44 | 51 | 1 | 2 | - | 35.47 | 63.25 | 1.28 | Present study. | |

Table 5.3.1.I (Cont.) Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | AcP A | AcP BA | AcP B | AcP CA | AcP CB | AcP C | $AcP^A$ | $AcP^B$ | $AcP^C$ | |
| 25-Shahsavar,Rudsar, Rudbar,Rasht,Langarud,Lahijan,Bandar-Pahlavi | 86 | 8 | 44 | 30 | 1 | 3 | - | 35.50 | 62.20 | 2.30 | Kirk,R.L.,et al. 1977 |
| 26-Baluchis.Sistan & Baluchistan | 111 | 17 | 47 | 46 | - | 1 | - | 36.49 | 63.06 | 0.45 | Present study. |
| 27-Iranians | 49 | 10 | 16 | 21 | - | 2 | - | 36.74 | 61.22 | 2.04 | Goldschmidt,Elizabeth.,et al. 1967 |
| 28-Kurds.Rezaieh | 146 | 17 | 74 | 55 | - | - | - | 36.99 | 63.01 | 0.00 | Present study. |
| 29-Gonbad | 155 | 24 | 65 | 59 | 2 | 5 | - | 37.10 | 60.60 | 2.30 | Kirk,R.L.,et al. 1977 |
| 30-Kurdish Jews | 106 | 18 | 41 | 39 | 2 | 6 | - | 37.27 | 58.96 | 3.77 | Tills,D.,et al. 1977 |
| 31-Southern Gorgan Behshahr, Sari | 53 | 7 | 26 | 20 | - | - | - | 37.70 | 62.30 | 0.00 | Kirk,R.L.,et al. 1977 |
| 32-Northern Gorgan | 44 | 8 | 24 | 11 | - | 1 | - | 45.50 | 53.40 | 1.10 | Kirk,R.L.,et al. 1977 |

Table 5.3.1. III Red cell Acid Phosphatase (AcP) phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | AcP A | AcP BA | AcP B | AcP CA | AcP CB | AcP C | $AcP^A$ | $AcP^B$ | $AcP^C$ | |
| 1-Turks | 274 | 23 | 109 | 127 | 5 | 10 | - | 29.20 | 68.06 | .2.74 | Hummel,K.,et al. 1970 |
| 2-Kurds | | | | | | | | 35.00 | 62.00 | '3.00 | Richard,P. 1976 |

Table 5.3.1. IV Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | AcP A | AcP BA | AcP B | AcP CA | AcP CB | AcP C | $AcP^A$ | $AcP^B$ | $AcP^C$ | |
| 1-Karaite Jews | 72 | - | - | 67 | - | 5 | - | 0.00 | 96.50 | 3.50 | Goldschmidt, Elizabeth.,et al. 1976 |
| 2-Kurdish Jews.South east | 50 | 6 | 21 | 23 | - | - | - | 33.00 | 67.00 | 0.00 | Tills,D.,et al. 1977 |
| 3-Jews | 82 | 11 | 33 | 35 | 1 | 2 | - | 34.15 | 64.02 | 1.83 | Goldschmidt, Elizabeth.,et al. 1967 |
| 4-Kurdish Jews | 27 | 3 | 13 | 7 | - | 4 | - | 35.18 | 57.41 | 7.41 | Godber,Marilyn.J., et al. 1973 |
| 5-Kurdish Jews. North west | 61 | 10 | 30 | 16 | - | 5 | - | 40.98 | 54.92 | 4.10 | Tills,D.,et al. 1977 |

Table 5.3.1. V Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Kuwait

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | AcP A | AcP BA | AcP B | AcP CA | AcP CB | AcP C | $AcP^A$ | $AcP^B$ | $AcP^C$ | |
| 1-Suluba tribe | 51 | 2 | 12 | 37 | - | - | - | 16.00 | 84.00 | 0.00 | Khaled,E.,et al. 1981 |
| 2-Ajman tribe | 50 | 5 | 9 | 36 | - | - | - | 19.00 | 81.00 | 0.00 | Khaled,E.,et al. 1981 |
| 3-General population | 86 | 3 | 26 | 57 | - | - | - | 19.00 | 81.00 | 0.00 | Khaled,E.,et al. 1981 |
| 4-Kuwaiti Arabs | 155 | 8 | 46 | 95 | 1 | 5 | - | 20.32 | 77.74 | 1.94 | Sawhney,K.S. 1975 |

Table 5.3.1. VI Red cell Acid phosphatase(AcP) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | AcP A | AcP BA | AcP B | AcP CA | AcP CB | AcP C | $AcP^A$ | $AcP^B$ | $AcP^C$ | |
| 1-Western Saudi Arabia | 143 | 6 | 16 | 107 | - | 12 | 2 | 9.79 | 84.62 | 5.59 | Saha,N.,et al. 1980 |
| 2-Saudi Arabians | 350 | 36 | 153 | 154 | 1 | 6 | - | 32.30 | 66.70 | 1.00 | Goedde,H.W., et al. 1979 |

Table 5.1.3.IX Red cell Acid phosphatase (AcP) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | AcP A | AcP BA | AcP B | AcP CA | AcP CB | AcP C | $AcP^A$ | $AcP^B$ | $AcP^C$ | |
| 1-Pushtus | 210 | 17 | 77 | 113 | 1 | 2 | - | 26.82 | 72.46 | 0.72 | Goedde,H.W.,et al. 1977 |
| 2-Pushtus | 103 | 13 | 33 | 56 | - | 1 | - | 28.60 | 70.90 | 0.50 | Papiha,S.S.,et al. 1977 |
| 3-Hazaras | 174 | 11 | 78 | 84 | - | 1 | - | 28.74 | 70.98 | 0.28 | Goedde,H.W.,et al. 1977 |
| 4-Tajiks | 310 | 28 | 126 | 151 | 1 | 4 | - | 29.52 | 69.68 | 0.80 | Goedde,H.W.,et al. 1977 |
| 5-Daris | 178 | 20 | 66 | 82 | 2 | 8 | - | 30.30 | 66.90 | 2.80 | Papiha,S.S.,et al. 1977 |
| 6-Uzbeks | 124 | 17 | 58 | 48 | - | 1 | - | 37.09 | 62.50 | 0.41 | Goedde,H.W.,et al. 1977 |

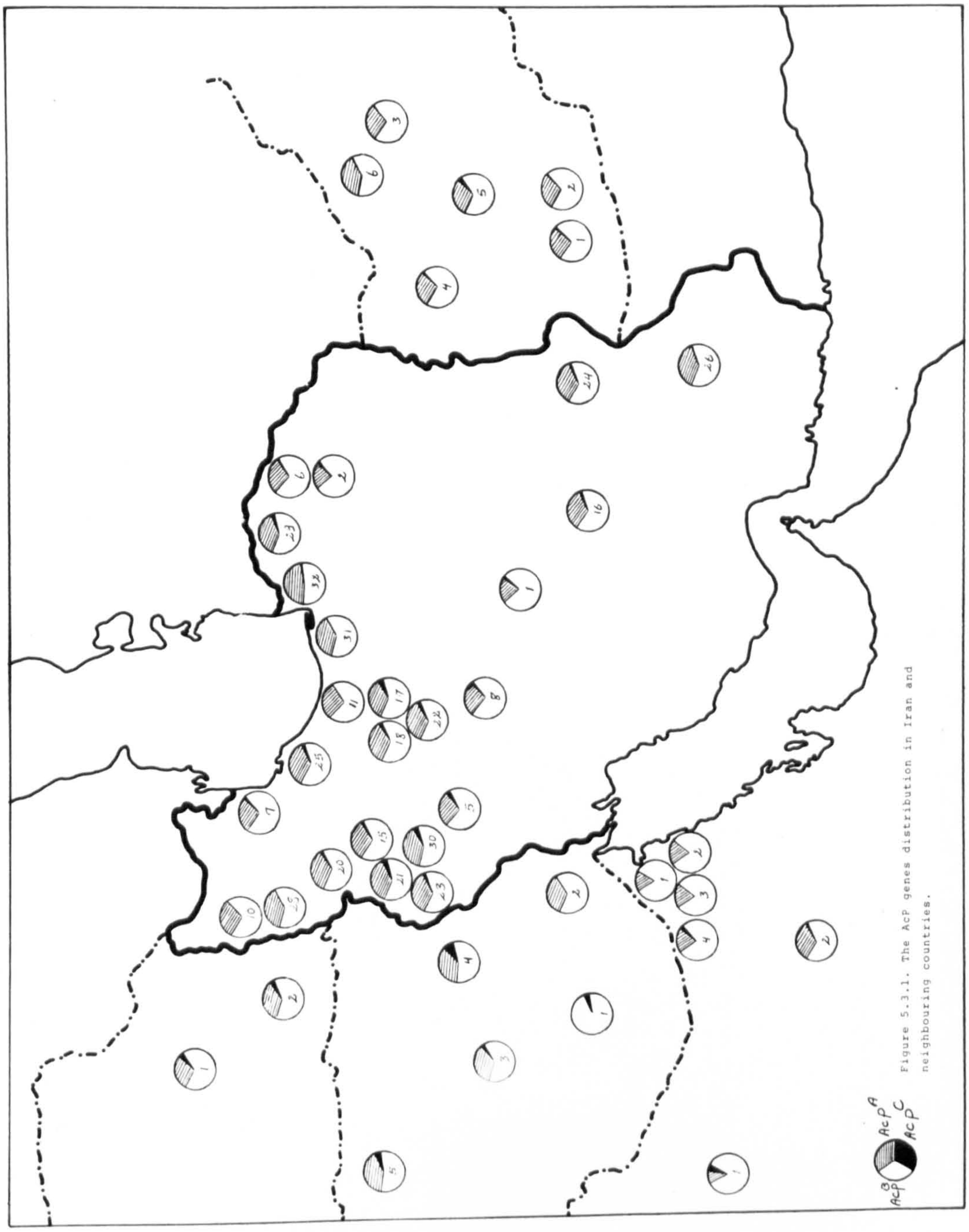



Figure 5.3.1. The AcP genes distribution in Iran and neighbouring countries.

## 5.3.2. The adenylate Kinase (AK) system

The distribution of adenylate kinase types and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.3.2.

The frequency of the $AK^2$ gene ranges from 1.38 to 10.81 percent in Iranians, being lowest in the Kurds of Rezaieh and highest in the Baluchis of Sistan and Baluchistan (Present investigation).

On the whole, with an average $AK^2$ frequency of 6.03 percent, the Iranian population appears to exhibit a higher $AK^2$ frequency than those varying between 2.5 and 6 percent in Europeans (Mourant et al, 1976).

It is difficult to see any definit regional trend of the distribution of the $K^2$ gene in Iran but it tends to be higher in the south east than the north west.

The extremely high $AK^2$ frequencies of 10.81 and 10.60 percent obtained in the present investigation in the Baluchis and the Zabolis of Sistan and Baluchistan (south eastern Iran) seem to be more similar to the high values found in Pakistan and India.

Regarding neighbouring areas, the frequency of the $AK^2$ gene in the populations of Turkey ranges from 4.00 percent in the Kurds (Richard, 1976) to 4.20 percent in the Turks (Hummel et al, 1970). With an average $AK^2$ frequency of 4.10 percent, the population of Turkey appears to exhibit a lower $AK^2$ frequency than that found in Iranians and more similar to the European frequency.

The $AK^2$ gene frequency in the populations of Iraq varies between zero in the Karaite Jews (Goldschmidt et al, 1976)

and 4.68 percent in the Jews (Adam, 1967). With an average $AK^2$ frequency of 2.04 percent, the Iraqi population seems to show a much lower $AK^2$ frequency than that found in Iranians and is even lower than the frequency in Europeans. The loss of the allele $AK^2$ in the Karaite Jews of Iraq clearly indicates the strong influence of isolation and genetic drift operating in this community.

The frequency of the $AK^2$ gene in the populations of Kuwait ranges from zero in the Ajman tribe to 5.00 percent in the general population of Kuwait (Khaled et al, 1981). With an average $AK^2$ frequency of 2.46 percent, the Kuwaiti population appears to exhibit a much lower $AK^2$ frequency than that found in Iranians and lower than the European frequency.

The $AK^2$ gene frequency of zero in the Ajman tribe of Kuwait may possibly be due to the relatively small sample size (N=50) and cannot be assumed as monomorphism of $AK^1$ though this situation occurs in several African populations (Tills et al, 1971).

The $Ak^2$ frequency of 3.50 percent in Saudi Arabians(Goedde et al, 1979) seems to be lower than the Iranian frequency and is more similar to that in Europeans.

The population of Pakistan with an $AK^2$ frequency of 12.96 percent (Cleghorn, 1967) appears to exhibit a much higher $AK^2$ frequency than that found in Iranians and of course much higher than the frequency in Europeans.

The frequency of the $AK^2$ gene in the populations of Afghanistan varies between 6.04 percent in the Hazaras and 10.72 percent in the Pushtus (Goedde et al, 1977). With an average $AK^2$ frequency of 8.53 percent, the Afghan population, like

that of Pakistan, shows a much higher $AK^2$ frequency than that found in Iranians and much higher than the European frequency. The frequency of the $AK^2$ gene is consistently higher in the Indian region than in Europe, and the $AK^2$ frequencies in Pakistan and Afghanistan are closer to the former.

## Conclusion

In European populations the $AK^2$ gene nearly always has a frequency between 2.5 and 6 percent. In the populations of the Near East, including the Jews, frequencies are similar to those found in Europe.

In Africa $AK^2$ levels are about 1 percent and several Negroid populations tested have shown only the $AK^1$ allele.

The highest known frequencies of $AK^2$ are found in India(Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arab populations of Iraq, Kuwait and Saudi Arabia with their relatively lower frequencies, the frequencies of the $AK^2$ gene appear to be higher than those in Europeans.

In Iranians though the $AK^2$ gene frequency seems to be higher than that in Europeans it is lower than in Indians but, the populations of Pakistan and Afghanistan with their much higher $AK^2$ frequencies appear to show more similarity with Indian populations.

On the whole, the frequency of the $AK^2$ gene seems to increases from Europe to India and the Iranian frequency is intermediate.

Table 5.3.2. I Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | AK 1-1 | AK 2-1 | AK 2-2 | $AK^1$ | $AK^2$ | | |
| 1-Kurds.Rezaieh | 145 | 141 | 4 | - | 98.62 | 1.38 | Present study. | |
| 2-Southern Gorgan, Behshahr,Sari | 53 | 51 | 2 | - | 98.10 | 1.90 | Kirk,R.L.,et al. | 1977 |
| 3-Turks.Rezaieh | 127 | 120 | 7 | - | 97.24 | 2.76 | Present study. | |
| 4-Jews | 164 | | | | 96.80 | 3.20 | Simhai,B. | 1974 |
| 5-Esfahan | 83 | 76 | 7 | - | 95.78 | 4.22 | Sawhney,K.S. | 1975 |
| 6-Gonbad | 155 | 140 | 15 | - | 95.20 | 4.80 | Kirk,R.L.,et al. | 1977 |
| 7-Tavalesh,Astara | 61 | 55 | 6 | - | 95.10 | 4.90 | Kirk,R.L.,et al. | 1977 |
| 8-Moslems.Shiraz | 322 | 290 | 32 | - | 95.03 | 4.97 | Bowman,J.E., and Ronaghy,H. | 1967 |
| 9-Tehran | 168 | 150 | 18 | - | 94.64 | 5.36 | Sawhney,K.S. | 1975 |
| 10.Babol,Shahi,Amol | 64 | 58 | 5 | 1 | 94.50 | 5.50 | Kirk,R.L.,et al. | 1977 |
| 11-Turks.Shirvan,Khorasan | 116 | 103 | 13 | - | 94.40 | 5.60 | Present study. | |
| 12-Kurds.Shirvan,Khorasan | 103 | 91 | 12 | - | 94.17 | 5.83 | Present study. | |

Table 5.3.2.I.(Cont.) Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | AK 1-1 | AK 2-1 | AK 2-2 | $AK^1$ | $AK^2$ | |
| 13-Zoroastrians | 120 | 105 | 15 | - | 93.75 | 6.25 | Present study. |
| 14-Kurdish Jews | 94 | 82 | 12 | - | 93.62 | 6.38 | Godber,Marilyn.J.,et al. 1973 |
| 15-Northern Gorgan | 44 | 38 | 6 | - | 93.20 | 6.80 | Kirk,R.L.,et al. 1977 |
| 16-Kurds | 182 | 159 | 21 | 2 | 93.13 | 6.87 | Tills,D.,et al. 1971 |
| 17-Kurdish Jews | 106 | 91 | 15 | - | 92.92 | 7.08 | Tills,D.,et al. 1977 |
| 18-Kerman | 309 | 268 | 38 | 3 | 92.88 | 7.12 | Present study. |
| 19-Tehran | 357 | 306 | 51 | - | 92.86 | 7.14 | Present study. |
| 20-Kurds.Baneh,Marivan | 77 | 68 | 7 | 2 | 92.86 | 7.14 | Lehmann,H.,et al. 1973 |
| 21-Lurs.Luristan | 178 | 154 | 22 | 2 | 92.70 | 7.30 | Present study. |
| 22-Kurds.Sanandaj | 106 | 91 | 14 | 1 | 92.45 | 7.55 | Lehmann,H.,et al. 1973 |
| 23-Shahsavar,Rudsar,Rudbar,Rasht,Langarud, Lahijan,Bandar Pahlavi | 86 | 70 | 16 | - | 90.70 | 9.30 | Kirk,R.L.,et al. 1977 |
| 24-Zabolis.Sistan & Baluchistan | 118 | 94 | 23 | 1 | 89.40 | 10.60 | Present study. |
| 25-Baluchis.Sistan & Baluchistan | 111 | 88 | 22 | 1 | 89.19 | 10.81 | Present study. |

Table 5.3.2. III Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | AK 1-1 | AK 2-1 | AK 2-2 | $AK^1$ | $AK^2$ | | |
| 1- Kurds | | | | | 96.00 | 4.00 | Richard,P. | 1976 |
| 2-Turks | 274 | 251 | 23 | - | 95.80 | 4.20 | Kummel,K.,et al. | 1970 |

Table 5.3.2. IV Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Iraq.

| Population | Number Tested | Phenotypes AK 1-1 | AK 2-1 | AK 2-2 | Gene frequencies $AK^1$ | $AK^2$ | Authors |
|---|---|---|---|---|---|---|---|
| 1-Karaite Jews | 71 | 71 | - | - | 100.00 | 0.00 | Goldschmidt,Elizabeth., et al. 1976 |
| 2-Kurdish Jews.North west | 61 | 60 | 1 | - | 99.18 | 0.82 | Tills,D.,et al. 1977 |
| 3-Kurdish Jews | 27 | 26 | 1 | - | 98.15 | 1.85 | Godber,Marilyn.J.,et al. 1973 |
| 4-Kurdish Jews.South east | 50 | 48 | 2 | - | 98.00 | 2.00 | Tills,D.,et al. 1977 |
| 5-Jews | 190 | - | - | - | 97.10 | 2.90 | Szeinberg,A.,et al. 1971 |
| 6-Jews | 139 | 126 | 13 | - | 95.32 | 4.68 | Adam.edited by : Rapley,S., et al. 1967 |

Table 5.3.2. V Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Kuwait

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | AK 1-1 | AK 2-1 | AK 2-2 | $AK^1$ | $AK^2$ | | |
| 1-Ajman tribe | 50 | 50 | - | - | 100.00 | 0.00 | Khaled,E.,et al. | 1981 |
| 2-Suluba tribe | 51 | 49 | 2 | - | 98.00 | 2.00 | Khaled,E.,et al. | 1981 |
| 3-Kuwaiti Arabs | 159 | 150 | 9 | - | 97.17 | 2.83 | Sawhney,K.S. | 1975 |
| 4-General population | 86 | 77 | 9 | - | 95.00 | 5.00 | Khaled,E.,et al. | 1981 |

Table 5.3.2. VI Red cell Adenylate Kinase(AK) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | AK 1-1 | AK 2-1 | AK 2-2 | $AK^1$ | $AK^2$ | |
| 1-Saudi Arabians | 359 | 335 | 23 | 1 | 96.50 | 3.50 | Goedde,H.W.,et al. 1979 |

Table 5.3.2. VIII Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Pakistan

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | AK 1-1 | AK 2-1 | AK 2-2 | $AK^1$ | $AK^2$ | |
| 1-Pakistanis | 54 | 40 | 14 | - | 87.04 | 12.96 | Cleghorn et al.edited 1967 by: Rapley, S.,et al. |

Table 5.3.2. IX Red cell Adenylate Kinase (AK) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | AK 1-1 | AK 2-1 | AK 2-2 | $AK^1$ | $AK^2$ | | |
| 1-Hazaras | 174 | 155 | 17 | 2 | 93.96 | 6.04 | Goedde,H.W.,et al. | 1977 |
| 2-Daris | 178 | 152 | 24 | 2 | 92.10 | 7.90 | Papiha,S.S.,et al. | 1977 |
| 3-Pushtus | 102 | 85 | 17 | - | 91.70 | 8.30 | Papiha,S.S.,et al. | 1977 |
| 4-Tajiks | 310 | 259 | 49 | 2 | 91.45 | 8.55 | Goedde,H.W.,et al. | 1977 |
| 5-Uzbeks | 124 | 103 | 18 | 3 | 90.32 | 9.68 | Goedde,H.W.,et al. | 1977 |
| 6-Pushtus | 210 | 167 | 41 | 2 | 89.28 | 10.72 | Goedde,H.W.,et al. | 1977 |

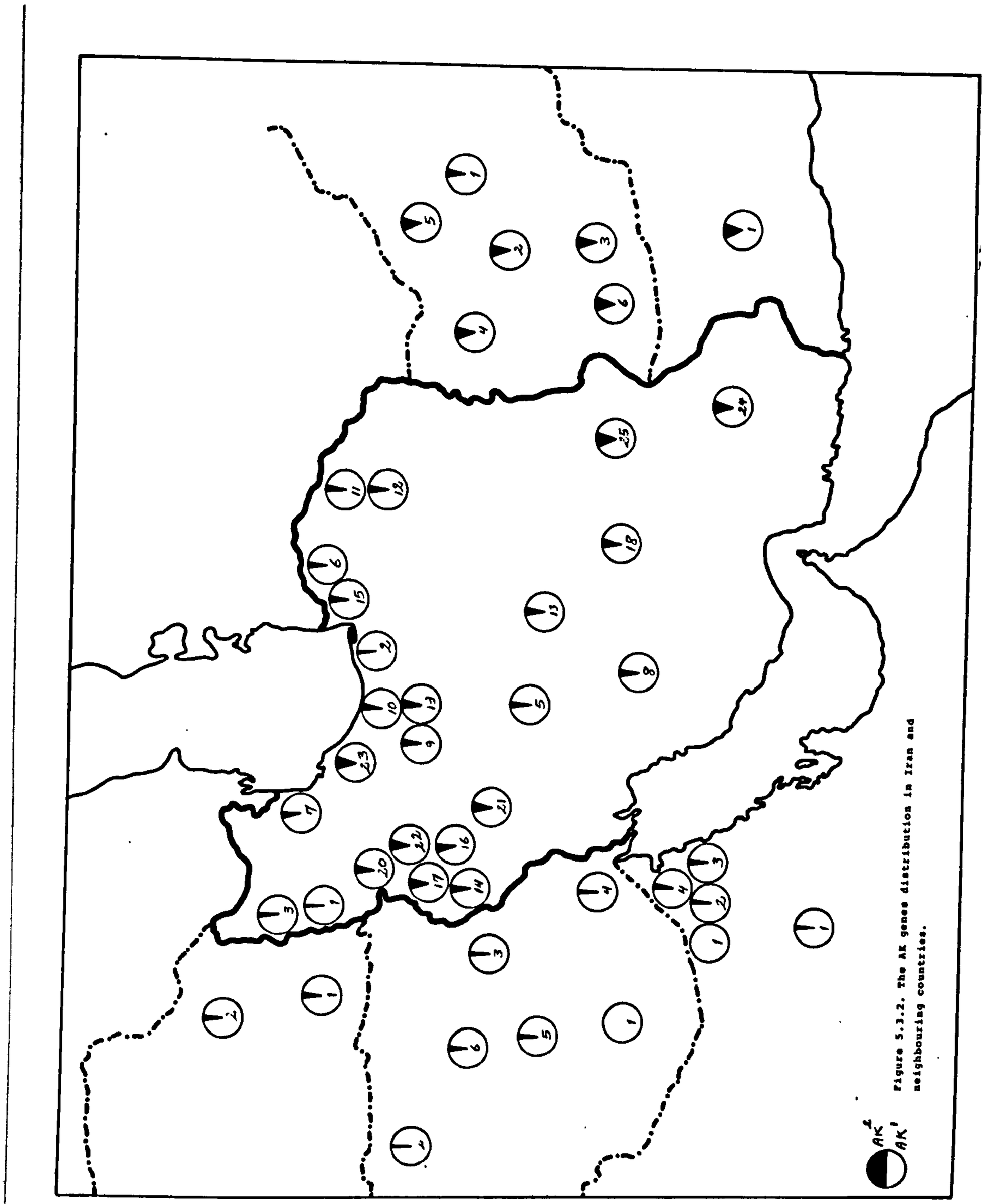


Figure 5.3.2. The AK genes distribution in Iran and neighbouring countries.

## 5.3.3. The Phosphoglucomutase Locus 1 ($PGM_1$) system

The distribution of $PGM_1$ types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.3.3.

The frequency of the $PGM_1^2$ gene varies between 22.08 and 34.56 percent in Iranians, being lowest in the Kurds of Baneh, Marivan (Lehmann et al, 1973) and highest in the Turks of Rezaieh (Present investigation). With the exception of the $PGM_1^2$ frequency of 43.56 percent in the Turks of Rezaieh, values obtained in the present study are within the range of variation. On the whole, with an average $PGM_1^2$ frequency of 31.43 percent, the Iranian population appears to exhibit a higher $PGM_1^2$ frequency than that of around 20 percent found in northern Europeans. The Iranian frequency seems to be more similar to that of around 30 percent in southern European and Mediterranean populations. Some differences in the distribution of the $PGM_1^2$ gene frequencies seem to exist in Iran as $PGM_1^2$ frequencies, averaging 30.82 percent, in the populations of western Iran are lower than those averaging 34.10 percent in the populations of eastern Iran.

The Kurdish Jews, both in Iran and in Iraq, seem to be characterized by lowerfrequencies of the $PGM_1^2$ gene but the Jews exhibit relatively higher values in both countries.

Regarding neighbouring populations, the frequency of the $PGM_1^2$ gene in the populations of Turkey ranges from 32.30 percent in the Turks (Hummel et al, 1970) to 34.00 percent in the Kurds ( Richard, 1976). With an average $PGM_1^2$ frequency of 33.15 percent, the population of Turkey appears to exhibit a $PGM_1^2$ frequency similar to that found in Iranians and comparable with other southern European and Mediterranean populations.

The frequency of the $PGM_1^2$ gene in the populations of Iraq varies between 16.39 percent in the Kurdish Jews of the north west (Tills et al, 1977) and 32.60 percent in the Jews (Hopkinson et al, 1966). With an average $PGM_1^2$ frequency of 25.36 percent, the Jewish population of Iraq seems to show a lower $PGM_1^2$ frequency than the average for Iran, but similar to the relatively lower values found in Iranian Jews both Kurdish and non-Kurdish.

The $PGM_1^2$ gene frequency in the populations of Kuwait ranges from 25.00 percent in the Ajman tribe to 31.00 percent in the Suluba tribe(Khaled et al, 1981). With an average $PGM_1^2$ frequency of 29.15 percent, the Kuwaiti population appears to exhibit a $PGM_1^2$ frequency similar to that found in Iranians.

A similar $PGM_1^2$ frequency of 29.80 percent is reported by Goedde et al (1979) for Saudi Arabians.

The frequency of the $PGM_1^2$ gene in the populations of Afghanistan varies between 14.66 percent in the Hazaras and 27.42 percent in the Tajiks (Goedde et al,1977). With an average $PGM_1^2$ frequency of 23.65 percent, the Afghan population seems to show lower $PGM_1^2$ frequency than that found in Iranians.

Conclusion

European populations have frequencies of $PGM_1^2$ increasing from 14 percent in Ireland and 18 percent in Iceland,eastward and southward to 31 percent in Greece.

Frequencies of $PGM_1^2$ in India exceed 30 percent but the Chinese and Japanese have between 20 and 25 percent.

African Negroid populations vary between 15 and 24 percent (Mourant et al, 1976).

In Iranian and neighbouring populations,with the exception of the Afghans with their relatively lower frequency,the

frequency of $PGM_1^2$ appears to be higher than that found in northern Europeans and more similar to that in southern European and Mediterranean populations.

Table 5.3.3.I Red cell Phosphoglucomutase Locus 1 ($PGM_1$) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PGM 1-1 | PGM 2-1 | PGM 2-2 | PGM 6-1 | $PGM_1^1$ | $PGM_1^2$ | $PGM_1^6$ | | |
| 1-Kurds.Baneh,Marivan | 77 | 45 | 30 | 2 | - | 77.92 | 22.08 | 0.00 | Lehmann, H.,et al. | 1973 |
| 2-Kurdish Jews | 106 | 65 | 34 | 7 | - | 77.36 | 22.64 | 0.00 | Tills,D., et al. | 1977 |
| 3-Zoroastrians | 104 | 60 | 40 | 4 | - | 76.92 | 23.08 | 0.00 | Present study. | |
| 4-Tehran | 165 | 101 | 50 | 14 | - | 76.36 | 23.64 | 0.00 | Sawhney,K.S. | 1975 |
| 5-Esfahan | 86 | 50 | 28 | 7 | 1 | 75.00 | 24.42 | 0.58 | Sawhney,K.S. | 1975 |
| 6-Kurdish Jews | 94 | 55 | 32 | 7 | - | 75.53 | 24.47 | 0.00 | Godber,Marilyn.J.,et al. | 1973 |
| 7-Armenians | 180 | - | - | - | - | 74.00 | 26.00 | 0.00 | Simhai,B. | 1978 |
| 8-Babol,Shahi,Amol | 64 | 36 | 22 | 6 | - | 73.40 | 26.60 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 9-Jews | 164 | - | - | - | - | 71.70 | 28.30 | 0.00 | Simhai,B. | 1974 |
| 10-Lurs.Luristan | 175 | 90 | 68 | 17 | - | 70.86 | 29.14 | 0.00 | Present study. | |
| 11-Northern Gorgan | 44 | 20 | 22 | 2 | - | 70.50 | 29.50 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 12-Tehran | 127 | 61 | 52 | 14 | - | 68.50 | 31.50 | 0.00 | Farhud,D.D.,et al. | 1973 |
| 13-Kurds.Sanandaj | 106 | 48 | 49 | 9 | - | 68.40 | 31.60 | 0.00 | Lehmann,H.,et al. | 1973 |

Table 5.3.3.I Red cell Phosphoglucomutase Locus 1 ($PGM_1$) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PGM 1-1 | PGM 2-1 | PGM 2-2 | PGM 6-1 | $PGM_1^1$ | $PGM_1^2$ | $PGM_1^6$ | | |
| 1-Kurds.Baneh,Marivan | 77 | 45 | 30 | 2 | - | 77.92 | 22.08 | 0.00 | Lehmann, H.,et al. | 1973 |
| 2-Kurdish Jews | 106 | 65 | 34 | 7 | - | 77.36 | 22.64 | 0.00 | Tills,D., et al. | 1977 |
| 3-Zoroastrians | 104 | 60 | 40 | 4 | - | 76.92 | 23.08 | 0.00 | Present study. | |
| 4-Tehran | 165 | 101 | 50 | 14 | - | 76.36 | 23.64 | 0.00 | Sawhney,K.S. | 1975 |
| 5-Esfahan | 86 | 50 | 28 | 7 | 1 | 75.00 | 24.42 | 0.58 | Sawhney,K.S. | 1975 |
| 6-Kurdish Jews | 94 | 55 | 32 | 7 | - | 75.53 | 24.47 | 0.00 | Godber,Marilyn.J.,et al. | 1973 |
| 7-Armenians | 180 | - | - | - | - | 74.00 | 26.00 | 0.00 | Simhai,B. | 1978 |
| 8-Babol,Shahi,Amol | 64 | 36 | 22 | 6 | - | 73.40 | 26.60 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 9-Jews | 164 | - | - | - | - | 71.70 | 28.30 | 0.00 | Simhai,B. | 1974 |
| 10-Lurs.Luristan | 175 | 90 | 68 | 17 | - | 70.86 | 29.14 | 0.00 | Present study. | |
| 11-Northern Gorgan | 44 | 20 | 22 | 2 | - | 70.50 | 29.50 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 12-Tehran | 127 | 61 | 52 | 14 | - | 68.50 | 31.50 | 0.00 | Farhud,D.D.,et al. | 1973 |
| 13-Kurds.Sanandaj | 106 | 48 | 49 | 9 | - | 68.40 | 31.60 | 0.00 | Lehmann,H.,et al. | 1973 |

Table 5.3.3.I (Cont.) Red cell Phosphoglucomutase Locus 1 ($PGM_1$) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | PGM 1-1 | PGM 2-1 | PGM 2-2 | PGM 6-1 | $PGM_1^1$ | $PGM_1^2$ | $PGM_1^6$ | |
| 14-Turks.Shirvan,Khorasan | 110 | 50 | 50 | 10 | - | 68.18 | 31.82 | 0.00 | Present study. |
| 15-Zabolis.Sistan & Baluchistan | 117 | 53 | 51 | 13 | - | 67.09 | 32.91 | 0.00 | Present study. |
| 16-Tehran | 346 | 152 | 153 | 41 | - | 66.04 | 33.96 | 0.00 | Present study. |
| 17-Baluchis.Sistan & Baluchistan | 110 | 45 | 54 | 11 | - | 65.45 | 34.55 | 0.00 | Present study. |
| 18-Kurds.Shirvan,Khorasan | 92 | 40 | 40 | 12 | - | 65.22 | 34.78 | 0.00 | Present study. |
| 19-Shahsavar,Rudsar, Rudbar,Rasht,Langarud,Lahijan,Bandar-Pahlavi | 86 | 34 | 40 | 11 | 1 | 63.40 | 36.00 | 0.60 | Kirk,R.L.,et al. 1977 |
| 20-Tavalesh,Astara | 61 | 27 | 24 | 10 | - | 63.90 | 36.10 | 0.00 | Kirk,R.L.,et al. 1977 |
| 21-Kerman | 310 | 123 | 144 | 43 | - | 62.91 | 37.09 | 0.00 | Present study. |
| 22-Gonbad | 155 | 49 | 88 | 18 | - | 60.00 | 40.00 | 0.00 | Kirk,R.L.,et al. 1977 |

Table 5.3.3.I. (Cont). Red cell Phosphoglucomutase Locus 1($PGM_1$) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PGM 1-1 | PGM 2-1 | PGM 2-2 | PGM 6-1 | $PGM_1^1$ | $PGM_1^2$ | $PGM_1^6$ | | |
| 23-Southern Gorgan Behshahr,Sari | 53 | 18 | 25 | 9 | 1 | 58.50 | 40.60 | 0.90 | Kirk,R.L.,et al. | 1977 |
| 24-Kurds.Rezaieh | 138 | 47 | 68 | 23 | - | 58.70 | 41.30 | 0.00 | Present study. | |
| 25-Turks.Rezaieh | 101 | 36 | 42 | 23 | - | 56.44 | 43.56 | 0.00 | Present study. | |

Table 5.3.3. III Red cell Phosphoglucomutase Locus 1($PGM_1$) phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | PGM 1-1 | PGM 2-1 | PGM 2-2 | $PGM_1^1$ | $PGM_1^2$ | | |
| 1-Turks | 274 | 119 | 133 | 22 | 67.70 | 32.30 | Hummel,K.,et al. | 1970 |
| 2-Kurds | | | | | 66.00 | 34.00 | Richard,P. | 1976 |

Table 5.3.3. IV. Red cell Phosphoglucomutase Locus 1 ($PGM_1$) phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | PGM 1-1 | PGM 2-1 | PGM 2-2 | $PGM_1^1$ | $PGM_1^2$ | | |
| 1-Kurdish Jews. North west | 61 | 42 | 18 | 1 | 83.61 | 16.39 | Tills,D.,et al. | 1977 |
| 2-Kurdish Jews | 27 | 17 | 10 | - | 81.48 | 18.52 | Godber,Marilyn.J., et al. | 1973 |
| 3-Karaite Jews | 70 | 36 | 27 | 7 | 70.70 | 29.30 | Goldschmidt,Elizabeth., et al. | 1976 |
| 4-Kurdish Jews. South east | 50 | 24 | 22 | 4 | 70.00 | 30.00 | Tills,D.,et al. | 1977 |
| 5-Jews | 69 | | | | 67.40 | 32.60 | Hopkinson,D.A.,et al. | 1966 |

Table 5.3.3. V Red cell Phosphoglucomutase Locus 1($PGM_1$) phenotypes and gene frequencies distribution in Kuwait

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | PGM 1-1 | PGM 2-1 | PGM 2-2 | $PGM_1^1$ | $PGM_1^2$ | | |
| 1-Ajman tribe | 50 | 29 | 17 | 4 | 75.00 | 25.00 | Khaled,E., et al. | 1981 |
| 2-Kuwaiti Arabs | 147 | 77 | 53 | 17 | 70.41 | 29.59 | Sawhney,K.S. | 1975 |
| 3-General population | 88 | 45 | 32 | 11 | 69.00 | 31.00 | Khaled,E., et al. | 1981 |
| 4-Suluba tribe | 51 | 22 | 26 | 3 | 69.00 | 31.00 | Khaled,E., et al. | 1981 |

Table 5.3.3. VI Red cell Phosphoglucomutase Locus 1 ($PGM_1$) phenotypes and gene frequencies distribution in Saudi Arabia.

| Population | Number Tested | Phenotypes | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | PGM 1-1 | PGM 2-1 | PGM 2-2 | PGM 3-1 | PGM 6-1 | $PGM_1^1$ | $PGM_1^2$ | $PGM_1^3$ | $PGM_1^6$ | |
| 1-Saudi Arabians | 359 | 180 | 136 | 39 | 2 | 2 | 69.60 | 29.80 | 0.30 | 0.30 | Goedde,H.W.,et al. 1979 |

Table 5.3.3. IX Red cell Phosphoglucomutase Locus 1( $PGM_1$) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | PGM 1-1 | PGM 2-1 | PGM 2-2 | PGM 4-1 | PGM 6-1 | PGM 6-2 | $PGM_1^1$ | $PGM_1^2$ | $PGM_1^4$ | $PGM_1^6$ | |
| 1-Hazaras | 174 | 124 | 46 | 2 | 1 | - | 1 | 84.78 | 14.66 | 0.28 | 0.28 | Goedde,H.W.,et al. 1977 |
| 2-Uzbeks | 124 | 80 | 36 | 8 | - | - | - | 79.03 | 20.97 | 0.00 | 0.00 | Goedde,H.W.,et al. 1977 |
| 3-Pushtus | 210 | 119 | 74 | 16 | - | 1 | - | 74.53 | 25.24 | 0.00 | 0.23 | Goedde,H.W.,et al. 1977 |
| 4-Daris | 173 | 100 | 54 | 19 | - | - | - | 73.40 | 26.60 | 0.00 | 0.00 | Papiha,S.S., et al. 1977 |
| 5-Pushtus | 102 | 60 | 29 | 13 | - | - | - | 73.00 | 27.00 | 0.00 | 0.00 | Papiha,S.S., et al. 1977 |
| 6-Tajiks | 310 | 163 | 124 | 23 | - | - | - | 72.58 | 27.42 | 0.00 | 0.00 | Goedde,H.W.,et al. 1977 |

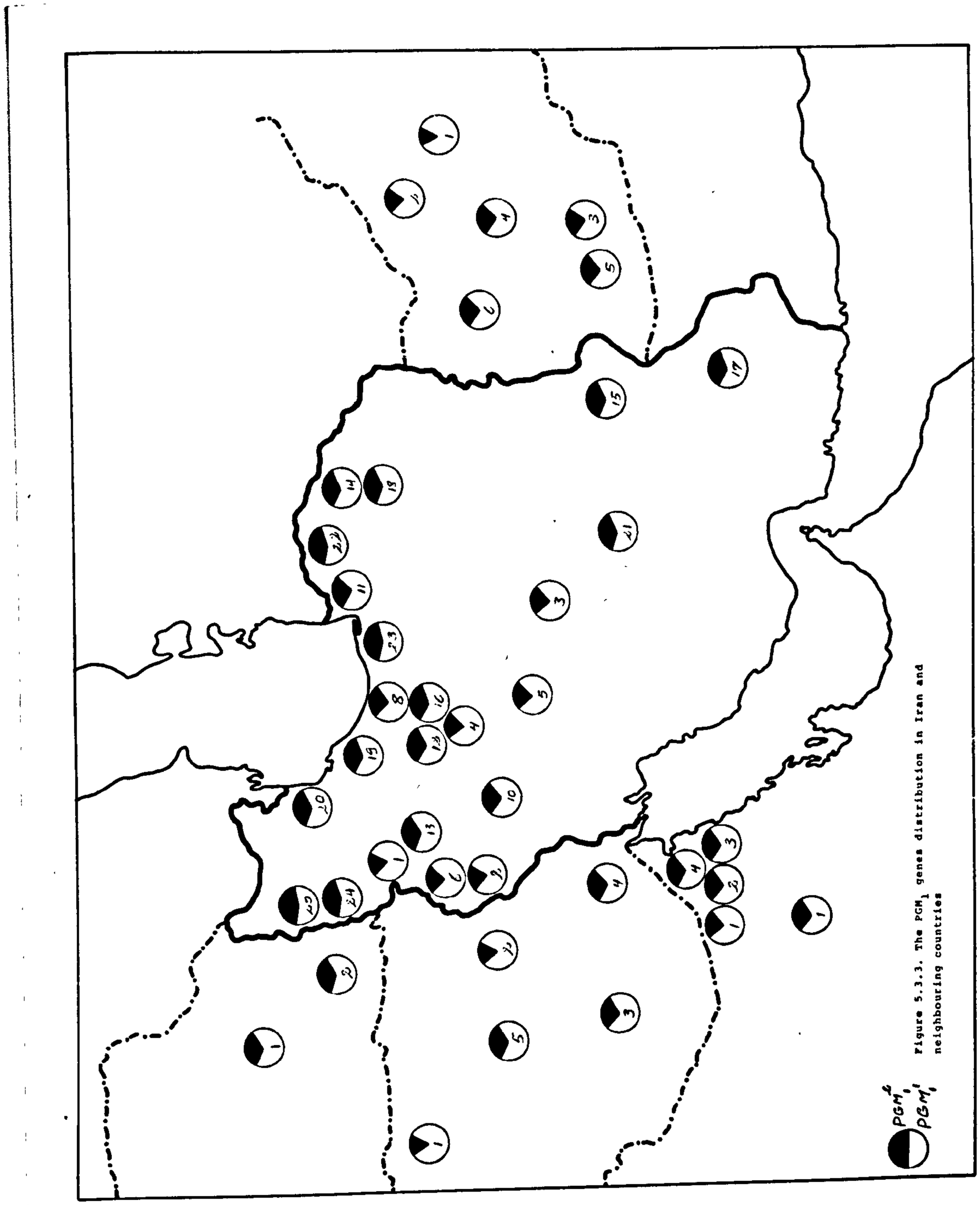



Figure 5.3.3. The $PGM_1$ genes distribution in Iran and neighbouring countries

## 5.3.4. The adenosine deaminase (ADA) system

The distribution of adenosine deaminase types and respective gene frequencies in Iranian and neighbouring populations is shown in Tables 5.3.4.

The frequency of the $ADA^2$ gene varies between 6.80 and 19.56 percent in Iranians, being lowest in northern Gorgan (Kirk et al, 1977) and highest in the Zoroastrians(Present investigation). With the exception of $ADA^2$ frequency of 19.56 percent in the Zoroastrians, values obtained in the present study are within the range of variation.

On the whole, with an average $ADA^2$ frequency of 13.29 percent, the Iranian population appears to exhibit a higher $ADA^2$ frequency than that of around 7 percent found in Europeans(Mourant et al, 1976).

Since the data available on ADA studies in Iran are still very few, complete discussion of the situation within the country is impossible.

Regarding neighbouring areas, the $ADA^2$ frequency of 9.60 percent in the Turks of Turkey (Altay et al, 1974) seems to be lower than that found in Iranians and quite close to those in Italians and Greeks.

The frequency of the $ADA^2$ gene in the populations of Iraq ranges from 9.76 percent in the Kurdish Jews of the south east to 24.39 percent in the Kurdish Jews of the north west(Tills et al, 1977). With an average $ADA^2$ frequency of 17.07 percent, the Iraqi Kurdish Jews seem to show a higher $ADA^2$ frequency than that found in Iranians and higher than the frequency in Europeans.

The $ADA^2$ gene frequency in the populations of Kuwait varies between 2.00 percent in the Suluba tribe and 16.00 per-

cent in the general population of Kuwait (Khaled et al,1981). With an average $ADA^2$ frequency of 8.00 percent, the Kuwaiti population appears to exhibit a lower $ADA^2$ frequency than that found in Iranians and also lower than the frequency in southern Europeans.

A similarly low $ADA^2$ frequency of 6.80 percent is reported by Goedde et al (1979) in Saudi Arabians.

The frequency of the $ADA^2$ gene in the Afghan populations ranges from 10.09 percent in the Uzbeks to 17.39 percent in the Pushtus (Goedde et al,1977). With an average $ADA^2$ frequency of 12.85 percent, the population of Afghanistan seems to show an $ADA^2$ frequency similar to that found in Iranians and higher than the European frequency.

Conclusion

The frequency of the $ADA^2$ gene ranges from 5 to 9 percent in European populations.Moreover,the European data seem to show an eastward increase in the frequency of $ADA^2$ from 5 percent in Ireland and 6 percent in England to 8 percent in Germany and 9 percent in Italy.

The frequency of the $ADA^2$ gene is consistently higher in the Indian region than in Europe.

In Africa the average frequency is under 1 percent(Mourant et al, 1976).

In Iranian and neighbouring populations,with the exception of the Arabs of Kuwait and Saudi Arabia with their relatively lower values, frequencies of the $ADA^2$ gene appear to be higher than those found in Europeans and more similar to the Indian frequencies. Thus as in Europe, the same trend seems to continue in Asia with around 9 percent in Turkey and 13 percent in Iran, Afghanistan and India.

Table 5.3.4.I Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | | Gene frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | ADA 1-1 | ADA 2-1 | ADA 2-2 | ADA 4-1 | ADA 6-1 | $ADA^1$ | $ADA^2$ | $ADA^4$ | $ADA^6$ | | |
| 1-Northern Gorgan | 44 | 38 | 6 | - | - | - | 93.20 | 6.80 | 0.00 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 2-Kurds.Baneh,Marivan | 77 | 66 | 11 | - | - | - | 92.86 | 7.14 | 0.00 | 0.00 | Lehmann,H.,et al. | 1973 |
| 3-Tavalesh,Astara | 61 | 51 | 10 | - | - | - | 91.80 | 8.20 | 0.00 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 4-Gonbad | 155 | 125 | 27 | 3 | - | - | 89.40 | 10.60 | 0.00 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 5-Kurds | 182 | 142 | 37 | 3 | - | - | 88.20 | 11.80 | 0.00 | 0.00 | Vanden Braden.,et al. | 1971 |
| 6-Shahsavar,Rudsar, Rudbar,Rasht,Langa-rud,Lahijan,Band-ar Pahlavi | 86 | 67 | 16 | 3 | - | - | 87.20 | 12.80 | 0.00 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 7-Turks.Rezaieh | 141 | 104 | 30 | 4 | 1 | 2 | 85.46 | 13.48 | 0.35 | 0.71 | Present study. | |
| 8-Jews | 164 | - | - | - | - | - | 85.80 | 14.20 | 0.00 | 0.00 | Simhai,B. | 1974 |
| 9-Kurds.Rezaieh | 147 | 106 | 38 | 3 | - | - | 85.04 | 14.96 | 0.00 | 0.00 | Present study. | |
| 10-Southern Gorgan, Behshahr,Sari | 53 | 37 | 16 | - | - | - | 84.90 | 15.10 | 0.00 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 11-Kurds.Shirvan, Khorasan | 101 | 74 | 23 | 4 | - | - | 84.66 | 15.34 | 0.00 | 0.00 | Present study. | |

Table 5.3.4.I (Cont.) Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | | Gene frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | ADA 1-1 | ADA 2-1 | ADA 2-2 | ADA 4-1 | ADA 6-1 | $ADA^1$ | $ADA^2$ | $ADA^4$ | $ADA^6$ | | |
| 12-Turks.Shirvan, Khorasan | 118 | 83 | 33 | 2 | - | - | 84.33 | 15.67 | 0.00 | 0.00 | Present study. | |
| 13-Kurds. Sanandaj | 105 | 75 | 27 | 3 | - | - | 84.29 | 15.71 | 0.00 | 0.00 | Lehmann,H.,et al. | 1973 |
| 14-Babol,Shahi,Amol | 64 | 43 | 19 | 2 | - | - | 82.00 | 18.00 | 0.00 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 15-Zoroastrians | 46 | 31 | 12 | 3 | - | - | 80.44 | 19.56 | 0.00 | 0.00 | Present study. | |

Table 5.3.4. III Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | ADA 1-1 | ADA 2-1 | ADA 2-2 | $ADA^1$ | $ADA^2$ | |
| 1-Turks | 566 | 462 | 99 | 5 | 90.40 | 9.60 | Altay,C., et al. 1974 |

Table 5.3.4. IV Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | ADA 1-1 | ADA 2-1 | ADA 2-2 | $ADA^1$ | $ADA^2$ | | |
| 1-Kurdish Jews. South east | 41 | 33 | 8 | - | 90.24 | 9.76 | Tills,D.,et al. | 1977 |
| 2-Kurdish Jews. North west | 41 | 22 | 18 | 1 | 75.61 | 24.39 | Tills,D.,et al. | 1977 |

Table 5.3.4. V

Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Kuwait

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | ADA 1-1 | ADA 2-1 | ADA 2-2 | $ADA^1$ | $ADA^2$ | | |
| 1-Suluba tribe | 52 | 50 | 2 | - | 98.00 | 2.00 | Khaled,E.,et al. | 1981 |
| 2-Ajman tribe | 51 | 45 | 6 | - | 94.00 | 6.00 | Khaled,E.,et al. | 1981 |
| 3-General population | 87 | 63 | 24 | - | 84.00 | 16.00 | Khaled,E.,et al. | 1981 |

Table 5.3.4. VI Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | ADA 1-1 | ADA 2-1 | ADA 2-2 | ADA 4-1 | $ADA^1$ | $ADA^2$ | $ADA^4$ | |
| 1- Saudi Arabians | 359 | 318 | 31 | 9 | 1 | 93.00 | 6.80 | 0.20 | Goedde,H.W.,et al. 1979 |

Table 5.3.4. IX Red cell Adenosine deaminase (ADA) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | ADA 1-1 | ADA 2-1 | ADA 2-2 | ADA 4-1 | ADA 4-2 | ADA 6-1 | $ADA^1$ | $ADA^2$ | $ADA^4$ | $ADA^6$ | |
| 1-Uzbeks | 124 | 96 | 21 | 1 | 3 | 2 | 1 | 87.50 | 10.09 | 2.01 | 0.40 | Goedde,H.W.,et al. 1977 |
| 2-Hazaras | 174 | 144 | 25 | 5 | - | - | - | 89.88 | 10.12 | 0.00 | 0.00 | Goedde,H.W.,et al. 1977 |
| 3-Tajiks | 310 | 238 | 63 | 7 | 1 | 1 | - | 87.09 | 12.59 | 0.32 | 0.00 | Goedde,H.W.,et al. 1977 |
| 4-Daris | 177 | 131 | 45 | 1 | - | - | - | 86.70 | 13.30 | 0.00 | 0.00 | Papiha,S.S.,et al. 1977 |
| 5-Pushtus | 103 | 76 | 26 | 1 | - | - | - | 86.40 | 13.60 | 0.00 | 0.00 | Papiha,S.S.,et al. 1977 |
| 6-Pushtus | 210 | 143 | 59 | 7 | 1 | - | - | 82.38 | 17.39 | 0.23 | 0.00 | Goedde,H.W.,et al. 1977 |

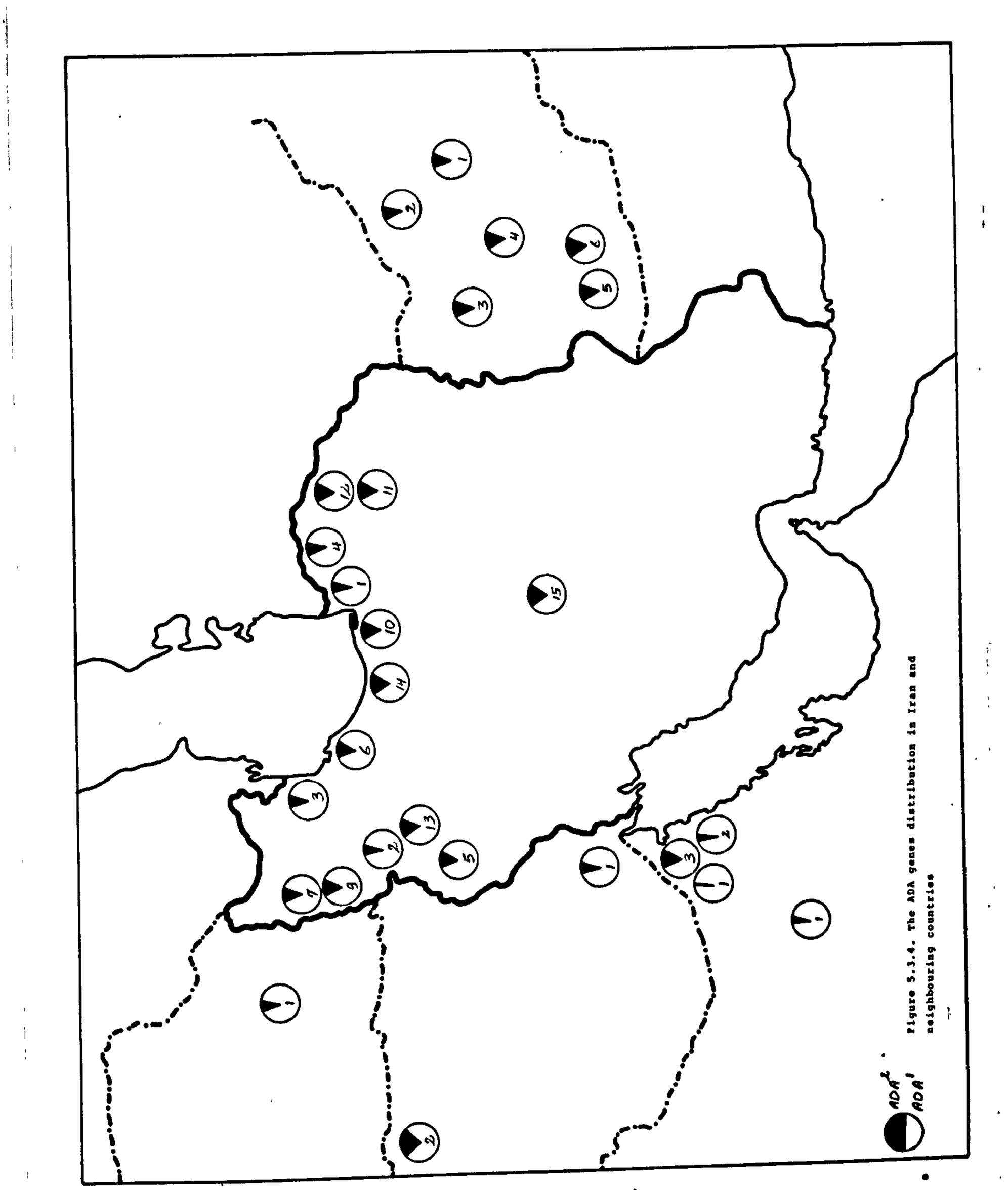



Figure 5.3.4. The ADA genes distribution in Iran and neighbouring countries

## 5.3.5. The esterase D (EsD) system

The distribution of the EsD types and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 5.3.5.

Esterase D is a relatively new polymorphic system, so that knowledge of the allele frequencies in different populations is, as yet, limited.

No observation of the EsD system among Iranians has hitherto been reported. The known heterogenity of the populations of Iran suggested that a searsh for variants there would be more rewarding than in more homogenous populations, so that the present study was under taken in samples from the ten different population groups in Iran.

The frequency of the $EsD^2$ gene varies between 12.61 and 29.55 percent in Iranians, being lowest in the Turks of Shirvan, Khorasan and highest in the Baluchis of Sistan and Baluchistan. With an average $EsD^2$ frequency of 19.23 percent, the Iranian population appears to exhibit a much higher $EsD^2$ frequency than that of about 11 percent in Europeans (Welch and Lee, 1974; Benkmann and Goedde, 1974; Koster et al, 1975).

In the ten Iranian samples, there does not appear to be any consistent pattern in the distribution of EsD genes.

The Turkish sample of Rezaieh in the north western region shows an $EsD^2$ frequency similar to that of the Baluchi sample of Sistan and Baluchistan in the south eastern region while the frequency in the Turks of Shirvan, Khorasan in the north east is similar to that in the Lurs of Luristan in the south western region.

Regarding neighbouring populations, the frequency of the $EsD^2$ gene in the populations of Iraq ranges from 18.90

percent in Baghdad to 24.20 percent in Ramadi(Papiha and Al-Agidi, 1976). With an average $EsD^2$ frequency of 21.62 percent, the population of Iraq, like that of Iran, seems to show a much higher $EsD^2$ frequency than that found in Europeans.

Similar high $EsD^2$ frequencies of 19.68 and 19.50 percent are reported by Cartwright et al(1976)and Goedde et al (1979)in the Kuwaitis and the Saudi Arabians,respectively.

The $EsD^2$ gene frequency in the populations of Afghanistan varies between 10.30 percent in the Afghan sample of papiha and Nahar(1977) and 20.96 percent in the Uzbeks(Goedde et al, 1977). With an average $EsD^2$ frequency of 16.50 percent, though the Afghan population appears to exhibit a lower $EsD^2$ frequency than that found in Iranians but the frequency is still higher than the European frequency.

Conclusion

Distinct geographical variation seems to exist in the frequency of the $EsD^2$ allele, particularly on the continental level.

The frequency of the $EsD^2$ gene in most populations of European origin is around 11 percent.

In eastern Asia it is considerably higher, around 34 percent.

India with an $EsD^2$ frequency of around 22 percent occupies an intermediate position between the European and east Asian levels.

The frequency of $EsD^2$ is only about 9 percent in Africans (Papiha and Nahar, 1977).

In Iranian and neighbouring populations, with the exception of the Afghans with their relatively lower frequency,the frequency of the $EsD^2$ gene appears to be much higher than that found in Europeans but slightly lower than that in Indians.

The position of Afghanistan, with a lower $EsD^2$ frequency than India, may indicate the historical association of the Afghans with the populations from the west.

Thus there is a clear increase in the frequency of the $EsD^2$ allele as one moves from Europe into Asia, and as one moves from peninsular India into the partially Mongoloid regions.

Already it seems clear that the major component of variation in the esterase D polymorphism is ethnic, strongly related to the continental groups to which the sampled populations belong, and this suggests that the frequencies owe little to ecological factors.

Table 5.3.5.I. Red cell Esterase D(EsD) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | EsD 1-1 | EsD 2-1 | EsD 2-2 | $EsD^1$ | $EsD^2$ | |
| 1-Turks.Shirvan,Khorasan | 115 | 88 | 25 | 2 | 87.39 | 12.61 | Present study. |
| 2-Lurs.Luristan | 178 | 136 | 36 | 6 | 86.52 | 13.48 | Present study. |
| 3-Zabolis.Sistan & Baluchistan | 117 | 86 | 28 | 3 | 85.47 | 14.53 | Present study. |
| 4-Kurds.Shirvan,Khorasan | 101 | 71 | 27 | 3 | 83.66 | 16.34 | Present study. |
| 5-Zoroastrians | 111 | 74 | 35 | 2 | 82.43 | 17.57 | Present study. |
| 6-Tehran | 345 | 229 | 105 | 11 | 81.60 | 18.40 | Present study. |
| 7-Kerman | 306 | 204 | 91 | 11 | 81.54 | 18.46 | Present study. |
| 8-Kurds.Rezaieh | 143 | 81 | 57 | 5 | 76.57 | 23.43 | Present study. |
| 9-Turks. Rezaieh | 129 | 66 | 54 | 9 | 72.09 | 27.91 | Present study. |
| 10-Baluchis.Sistan & Baluchistan | 110 | 57 | 41 | 12 | 70.45 | 29.55 | Present study. |

Table 5.3.5. IV Red cell Esterase D (EsD) phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | EsD 1-1 | EsD 2-1 | EsD 2-2 | $EsD^1$ | $EsD^2$ | |
| 1-Baghdad | 122 | 81 | 36 | 5 | 81.10 | 18.90 | Papiha,S.S., & Al-Agidi,S.K. 1976 |
| 2-Basra | 92 | 59 | 30 | 3 | 80.40 | 19.60 | Papiha,S.S., & Al-Agidi,S.K. 1976 |
| 3-Kirkuk | 42 | 24 | 16 | 2 | 76.20 | 23.80 | Papiha,S.S., & Al-Agidi,S.K. 1976 |
| 4-Ramadi | 64 | 34 | 29 | 1 | 75.80 | 24.20 | Papiha,S.S., & Al-Agidi,S.K. 1976 |

Table 5.3.5. V Red cell Esterase D( EsD) phenotypes and gene frequencies distribution in Kuwait

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | EsD 1-1 | EsD 2-1 | EsD 2-2 | $EsD^1$ | $EsD^2$ | | |
| 1-Kuwaitis | 160 | 101 | 55 | 4 | 80.32 | 19.68 | Cartwright,R.A.,et al. | 1976 |

Table 5.3.5. VI Red cell Esterase D (EsD) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | EsD 1-1 | EsD 2-1 | EsD 2-2 | $EsD^1$ | $EsD^2$ | |
| 1-Saudi Arabians | 359 | 229 | 120 | 10 | 80.50 | 19.50 | Goedde,H.W., et al. 1979 |

Table 5.3.5. IX

Red cell Esterase D (EsD) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | EsD 1-1 | EsD 2-1 | EsD 2-2 | EsD 3-1 | $EsD^1$ | $EsD^2$ | $EsD^3$ | | |
| 1-Afghans | 224 | 182 | 38 | 4 | - | 89.70 | 10.30 | 0.00 | Papiha,S.S., & Nahar,A. | 1977 |
| 2-Pushtus | 210 | 153 | 51 | 5 | 1 | 85.24 | 14.53 | 0.23 | Goedde,H.W.,et al. | 1977 |
| 3-Tajiks | 308 | 210 | 86 | 12 | - | 82.26 | 17.74 | 0.00 | Goedde,H.W.,et al. | 1977 |
| 4-Hazaras | 174 | 117 | 48 | 9 | - | 81.04 | 18.96 | 0.00 | Goedde,H.W.,et al. | 1977 |
| 5-Uzbeks | 124 | 74 | 40 | 6 | 4 | 77.41 | 20.96 | 1.63 | Goedde,H.W.,et al. | 1977 |

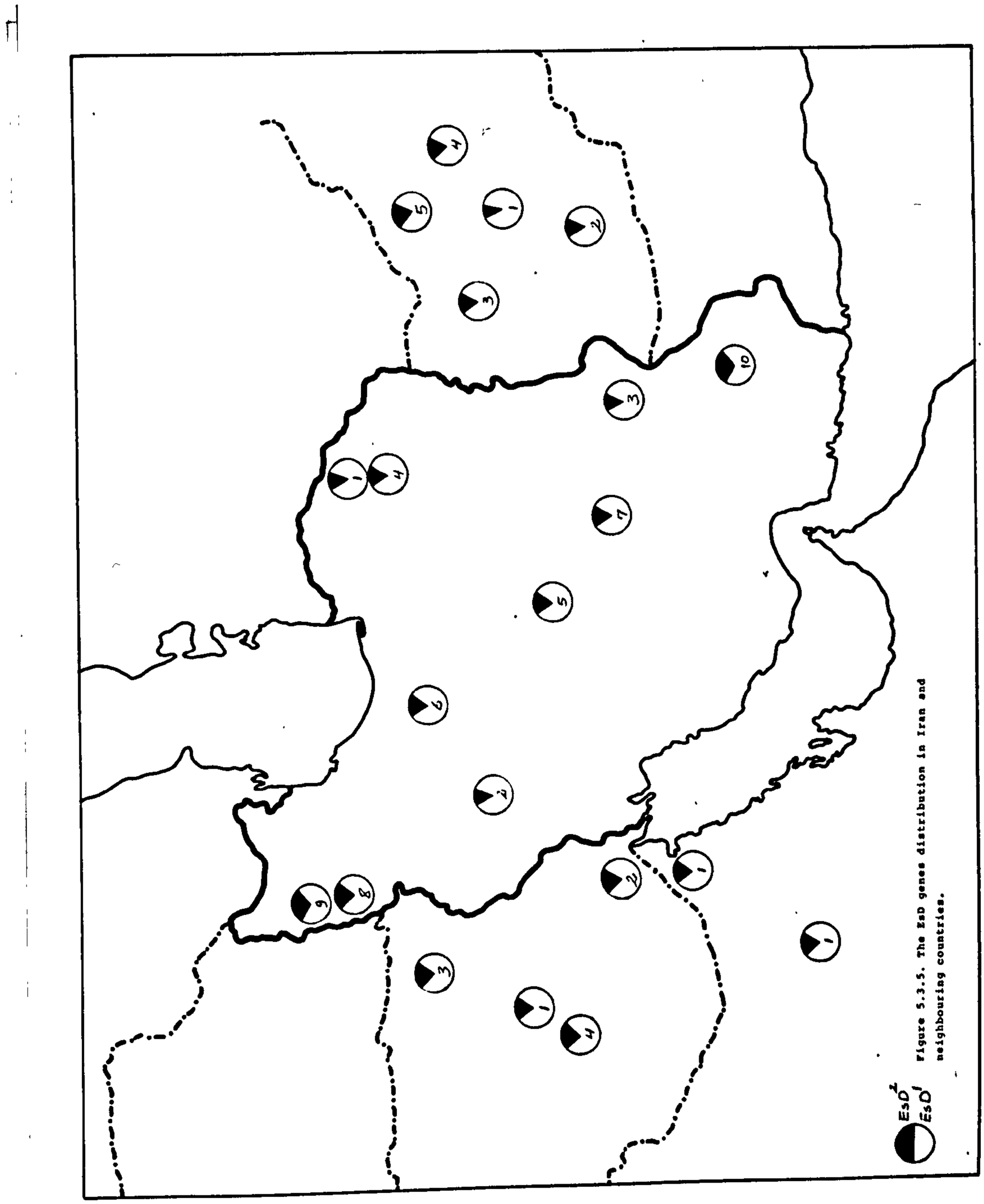



Figure 5.3.5. The EsD genes distribution in Iran and neighbouring countries.

## 5.3.6. The 6-phosphogluconate dehydrogenase(6PGD)system

The distribution of 6-phosphogluconate dehydrogenase types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.3.6.

The frequency of the $PGD^C$ gene ranges from zero to 7.14 percent in Iranians, being lowest in Tavalesh, Astara (Kirk et al, 1977) and highest in the Kurds of Baneh, Marivan(Lehmann et al, 1973). Values obtained in the present investigation are within this range of variation.

On the whole, with an average $PGD^C$ frequency of 3.19 percent, the Iranian population appears to exhibit a $PGD^C$ frequency similar to that varying between 1 and 4 percent found in Europeans (Mourant et al, 1976). However, it seems to be lower than for some other Middle Eastern populations (Tills et al, 1971). It appears from the table that there is an apparent cline for the $PGD^C$ allele in the Caspian sea area of Iran, declining from 5.20 percent in Gonbad ( east Caspian) to zero in Tavalesh, Astara (west Caspian )

The absence of the 6PGD variants in the Iranian Zoroastrians could possibly be due to the small size of the series investigated.

The Kurdish populations of Iran both Jewish and non-Jewish seem to exhibit the highest $PGD^C$ frequencies (average around 5 percent) within the country.

Regarding neighbouring groups, the frequency of the $PGD^C$ allele in the populations of Turkey varies between 0.10 percent in the Turks(Altay et al, 1974) and 4.00 percent in the Kurds ( Richard, 1976). With an average $PGD^C$ frequency of 2.47 percent, the population of Turkey, like that of Iran, seems to show a $PGD^C$ allele frequency similar to that found

in Europeans.

The $PGD^C$ gene frequency in the populations of Iraq ranges from zero in the Karaite Jews (Goldschmidt et al, 1976) to 4.10 percent in the Kurdish Jews of the north west (Tills et al, 1977). With an average $PGD^C$ frequency of 2.47 percent, the Jewish population of Iraq also appears to exhibit a $PGD^C$ allele frequency similar to the European frequency. The loss of the allele $PGD^C$ in the Karaite Jews of Iraq clearly indicates the strong influence of isolation and genetic drift operating in this community.

The frequency of the $PGD^C$ gene in the populations of Kuwait varies between 3.33 percent in the Kuwaiti Arabs (Sawhney, 1975) and 9.00 percent in the Ajman tribe (Khaled et al, 1981). With an average $PGD^C$ frequency of 6.58 percent, the Kuwaiti population seems to show a much higher $PGD^C$ frequency than that found in Iranians and much higher than the frequency in Europeans.

The $PGD^C$ gene frequency in the populations of Saudi Arabia ranges from 3.02 percent in western Saudi Arabia (Saha et al, 1980) to 11.20 percent in the Saudi Arabian sample of Goedde et al (1979). With an average $PGD^C$ frequency of 7.11 percent, the Arab population of Saudi Arabia, like that of Kuwait, appears to exhibit a much higher $PGD^C$ frequency than that found in Iranians and much higher than the European frequency. Similar high frequencies of the $PGD^C$ allele were observed in other Arab populations (Tills et al, 1971). High $PGD^C$ frequencies were also found in several Negro populations (Mourant et al, 1976).

The frequency of the $PGD^C$ allele in the populations of Afghanistan varies between 2.02 percent in the Uzbeks and

10.63 percent in the Hazaras ( Goedde et al,1977). Up to now the high frequency of 10.63 percent found in the Hazaras of Afghanistan has only been surpassed by the results of a survey in Bhutan (Fich and Parr, 1967), Chinese (Shih and Hsia, 1969), some African tribes (Mourant et al, 1976) and Saudi Arabians (Goedde et al, 1979). Thus, apart from the Hazaras, with an average $PGD^C$ frequency of 3.17 percent, the Afghan population appears to exhibit $PGD^C$ allele frequencies more like those in Iran, Turkey, Iraq and other populations to the west.

Conclusion

The frequency of the $PGC^C$ gene varies between 1 and 4 percent in European populations, and tends to be higher in the south.

In Africa frequencies of this gene are mostly around 6 percent but reach about 15 percent in the Ethiopians, the Beja of the Sudan and the south African Bantu.

They are on the whole higher still in Asia, varying from 7 percent in much of the Far East to 10 percent in Nepal and 23 percent in Bhutan (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Arabs of Kuwait and Saudi Arabia with their higher frequencies, the frequency of the $PGD^C$ allele appears to be similar to that found in Europeans. However, it seems to be lower than for some other Middle Eastern populations (Tills et al, 1971). High frequencies of the $PGD^C$ gene, similar to those found in the Arabs of Kuwait and Saudi Arabia, were also observed in other Arabs( Tills et al, 1971), and several Negro populations ( Mourant et al, 1976).

Table 5.3.6.I Red cell 6-phosphogluconate dehydrogenase (6PGD) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PGD AA | PGD AC | PGD CC | PGD Rare (HA) | $PGD^A$ | $PGD^C$ | $PGD^H$ | | |
| 1-Tavalesh,Astara | 61 | 61 | - | - | - | 100.00 | 0.00 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 2-Zoroastrians | 66 | 66 | - | - | - | 100.00 | 0.00 | 0.00 | Present study. | |
| 3-Shahsavar,Rudsar,Rudbar, Rasht,Langarud,Lahijan, Bandar-Pahlavi | 86 | 84 | 2 | - | - | 98.80 | 1.20 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 4-Tehran | 132 | 128 | 4 | - | - | 98.47 | 1.53 | 0.00 | Farhud,D.D.,et al. | 1973 |
| 5-Tehran | 82 | 79 | 3 | - | - | 98.17 | 1.83 | 0.00 | Present study. | |
| 6-Southern Gorgan,Behshahr,Sari | 53 | 51 | 2 | - | - | 98.10 | 1.90 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 7-Armenians | 180 | | | | | 98.00 | 2.00 | 0.00 | Simhai,B. | 1978 |
| 8-Jews | 164 | | | | | 97.70 | 2.30 | 0.00 | Simhai, B. | 1974 |
| 9-Tehran | 165 | 157 | 8 | - | - | 97.58 | 2.42 | 0.00 | Sawhney,K.S. | 1975 |
| 10-Moslems.Shiraz | 322 | 304 | 18 | - | - | 97.20 | 2.80 | 0.00 | Bowman,J.E., & Ronaghy, H. | 1967 |
| 11-Esfahan | 89 | 84 | 5 | - | - | 97.19 | 2.81 | 0.00 | Sawhney,K.S. | 1975 |

Table 5.3.6.I(Cont.) Red cell 6-phosphogluconate dehydrogenase(6PGD)phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PGD AA | PGD AC | PGD CC | PGD Rare(HA) | $PGD^A$ | $PGD^C$ | $PGD^H$ | | |
| 12-Kurds.Sanandaj | 106 | 100 | 6 | - | - | 97.17 | 2.83 | 0.00 | Lehmann,H.,et al. | 1973 |
| 13-Babol,Shahi,Amol | 64 | 60 | 4 | - | - | 96.90 | 3.10 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 14-Northern Gorgan | 51 | 47 | 4 | - | - | 96.10 | 3.90 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 15-Kerman | 301 | 278 | 22 | 1 | - | 96.01 | 3.99 | 0.00 | Present study. | |
| 16-Kurdish Jews | 106 | 96 | 9 | - | 1 | 95.28 | 4.25 | 0.47 | Tills,D.,et al. | 1977 |
| 17-Kurdish Jews | 94 | 85 | 8 | - | 1 | 95.21 | 4.26 | 0.53 | Godber,Marilyn.J., et al. | 1973 |
| 18-Kurds | 182 | 165 | 17 | - | - | 95.33 | 4.67 | 0.00 | Tills,D.,et al. | 1971 |
| 19-Gonbad | 155 | 139 | 16 | - | - | 94.80 | 5.20 | 0.00 | Kirk,R.L.,et al. | 1977 |
| 20-Turks.Shirvan,Khorasan | 90 | 80 | 10 | - | - | 94.44 | 5.56 | 0.00 | Present study. | |
| 21-Kurds.Shirvan,Khorasan | 70 | 61 | 9 | - | - | 93.57 | 6.43 | 0.00 | Present study. | |
| 22-Kurds.Baneh,Marivan | 77 | 66 | 11 | - | - | 92.86 | 7.14 | 0.00 | Lehmann,H.,et al. | 1973 |

Table 5.3.6. III Red cell 6-phosphogluconate dehydrogenase (6PGD) phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | PGD AA | PGD AC | PGD CC | PGD Rare (HA) | $PGD^A$ | $PGD^C$ | $PGD^H$ | |
| 1-Turks | 500 | 499 | 1 | - | - | 99.90 | 0.10 | 0.00 | Altay,C.,et al. 1974 |
| 2-Turks | 198 | 185 | 13 | - | - | 96.70 | 3.30 | 0.00 | Brinkmann,B.,et al.1973 |
| 3-Kurds. | | | | | | 96.00 | 4.00 | 0.00 | Richard,P. 1976 |

Table 5.3.6. IV Red cell 6-phosphogluconate dehydrogenase(6PGD)phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | PGD AA | PGD AC | PGD CC | PGD Rare(HA) | $PGD^A$ | $PGD^C$ | $PGD^H$ | |
| 1-Karaite Jews | 72 | 72 | - | - | - | 100.00 | 0.00 | 0.00 | Goldschmidt, Elizabeth.,et al. 1976 |
| 2-Kurdish Jews | 27 | 26 | 1 | - | - | 98.15 | 1.85 | 0.00 | Godber,Marilyn.J.,et al. 1973 |
| 3-Kurdish Jews.South east | 50 | 47 | 3 | - | - | 97.00 | 3.00 | 0.00 | Tills,D.,et al. 1977 |
| 4-Kurdish Jews.North west | 61 | 56 | 5 | - | - | 95.90 | 4.10 | 0.00 | Tills,D.,et al. 1977 |

Table 5.3.6. V Red cell 6-phosphogluconate dehydrogenase(6PGD) phenotypes and gene frequencies distribution in Kuwait

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors |
|---|---|---|---|---|---|---|---|---|---|
| | | PGD AA | PGD AC | PGD CC | PGD Rare(HA) | $PGD^A$ | $PGD^C$ | $PGD^H$ | |
| 1-Kuwaiti Arabs | 150 | 140 | 10 | - | - | 96.67 | 3.33 | 0.00 | Sawhney,K.S. 1975 |
| 2-Suluba tribe | 52 | 47 | 4 | 1 | - | 94.00 | 6.00 | 0.00 | Khaled,E.,et al. 1981 |
| 3-General population | 86 | 72 | 14 | - | - | 92.00 | 8.00 | 0.00 | Khaled,E.,et al. 1981 |
| 4-Ajman tribe | 51 | 43 | 7 | 1 | - | 91.00 | 9.00 | 0.00 | Khaled,E.,et al. 1981 |

Table 5.3.6. VI Red cell 6-Phosphogluconate dehydrogenase (6PGD) phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PGD AA | PGD AC | PGD CC | PGD rare(HA) | $PGD^A$ | $PGD^C$ | $PGD^H$ | | |
| 1-Western Saudi Arabia | 149 | 141 | 7 | 1 | - | 96.98 | 3.02 | 0.00 | Saha,N.,et al. | 1980 |
| 2-Saudi Arabians | 354 | 280 | 69 | 5 | - | 88.80 | 11.20 | 0.00 | Goedde,H.W.,et al. | 1979 |

Table 5.3.6. IX Red cell 6-Phosphogluconate dehydrogenase(6PGD) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|
| | | PGD AA | PGD AC | PGD CC | PGD rare(HA) | $PGD^A$ | $PGD^C$ | $PGD^H$ | | |
| 1-Uzbeks | 124 | 119 | 5 | - | - | 97.98 | 2.02 | 0.00 | Goedde,H.W.,et al. | 1977 |
| 2-Pushtus | 210 | 200 | 10 | - | - | 97.62 | 2.38 | 0.00 | Goedde,H.W.,et al. | 1977 |
| 3-Tajiks | 310 | 293 | 17 | - | - | 97.26 | 2.74 | 0.00 | Goedde,H.W.,et al. | 1977 |
| 4-Pushtus | 103 | 95 | 8 | - | - | 96.10 | 3.90 | 0.00 | Papiha,S.S.,et al. | 1977 |
| 5-Daris | 177 | 159 | 17 | - | 1 | 94.90 | 4.80 | 0.30 | Papiha,S.S.,et al. | 1977 |
| 6-Hazaras | 174 | 138 | 35 | 1 | - | 89.37 | 10.63 | 0.00 | Goedde,H.W.,et al. | 1977 |

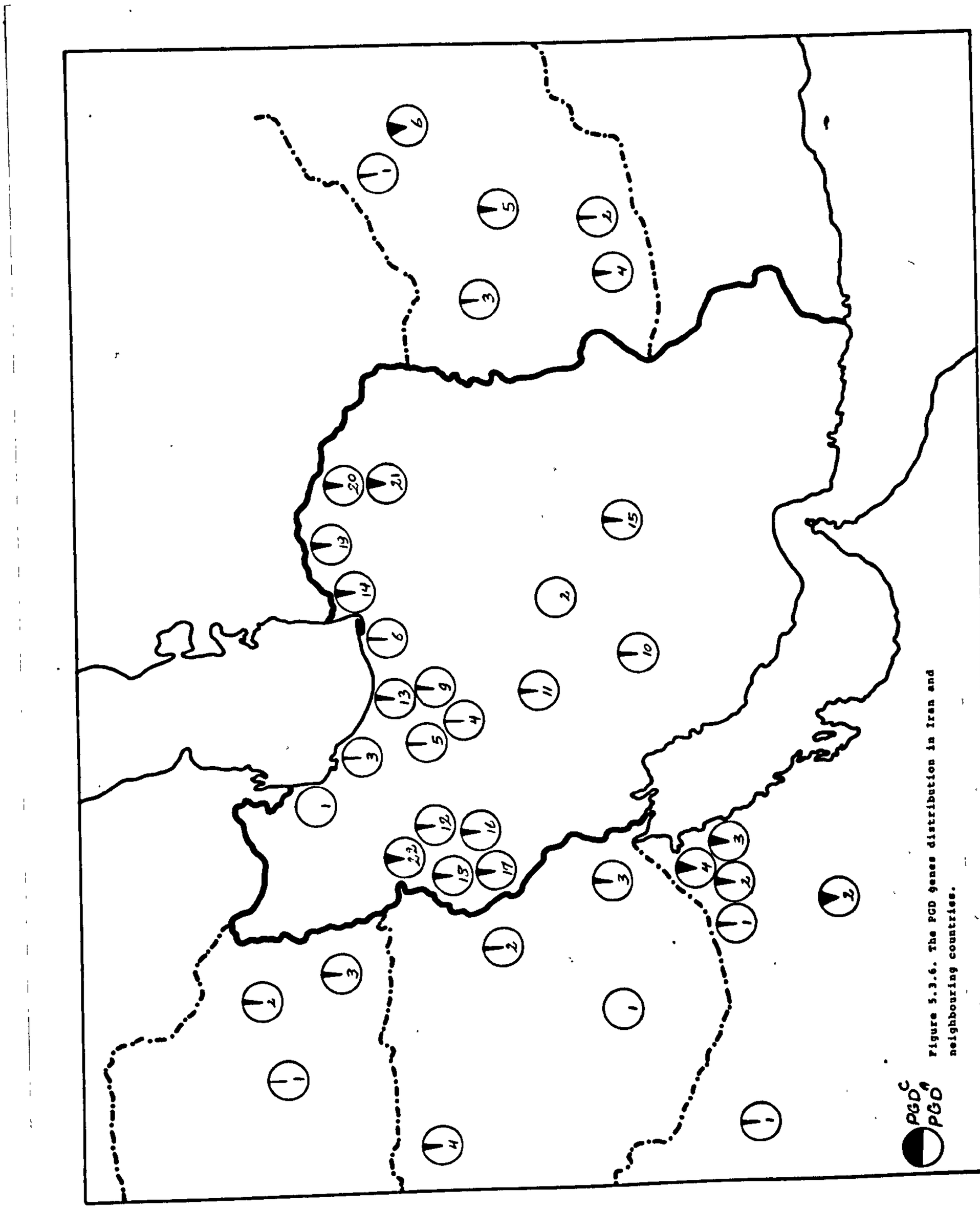



Figure 5.3.6. The PGD genes distribution in Iran and neighbouring countries.

5.3.7. The glucose-6-phosphate dehydrogenase( G6PD) system

Data on the distribution of glucose-6-phosphate dehydrogenase deficiency in Iranian and neighbouring populations are set out in Tables 5.3.7.

The frequency of the $Gd^-$ gene ranges from zero to 58.16 percent in Iranians, being lowest in the Zoroastrians(Bowman and Walker, 1961) and highest in the Kurdish Jews of western Iran ( Sheba et al, 1961).

A number of investigators have reported deficiency of G6PD in Iranians. Among them is a series of studies by Bowman and his collaborators of Moslems, Zoroastrians, Armenians and several tribal populations (summarized by Bowman and Ronaghy,1967 ). For male Moslems in Shiraz they found approximately 9 percent deficiency, a figure similar to that found for Moslems in other areas. Beaconsfield et al (1967) also surveyed the frequency of G6PD deficiency in various populations in Iran. In Yazd and environs, a desert plateau free from malaria, G6PD deficiency was reported to be 1 percent among Zoroastrians and 2.50 percent among Moslems, whereas among Moslems in the more malarial parts of the Caspian Littoral it was 25 percent. High values were also given for ethnic groups in and around the city of Kermanshah where malaria is common, while lower values (9 percent fro Moslems in Esfahan) were found where malaria has been intermittent. Kirk et al (1977) studied the frequency of G6PD deficiency for six areas in the Caspian Littoral. The male G6PD deficiency frequencies range from 1.20 to 17.90 percent, and there is a marked difference in frequency between the low values in Gonbad (1.20 percent) and Tavalesh, Astara (6.50 percent) and the other four areas from northern Gorgan to Bandar-Pahlavi where values are three to

four times higher. These latter areas comprise what used to be the highly malarial parts of the Caspian Littoral or include persons who have moved from such areas in the recent past, and corresponds with that part of the Caspian area included in Beaconsfield's (1967) study referred to above. The frequencies reported by Kirk et al (1977) therefore are in good agreement with those from previous work and add further support to the relationship between the frequency of G6PD deficiency and the level of malaria endemicity in the recent past. The most striking finding is the very high frequency of G6PD deficiency in the Kurdish Jews of western Iran on the one hand and the low frequency in the non-Jewish Kurds of the same region on the other. Thus, the Iranian Kurds carry the gene, but with a much lower frequency than the Kurdish Jews who live among them. It is difficult to account in terms of exposure to malaria for the marked difference between two populations living in such close proximity.

Though quite high frequencies are found in the indigenous populations in many parts of Iran, since one of the main determining conditions is probably a selective advantage of carriers of the gene, in the presence of endemic falciparum malaria, the very much higher frequency in Kurdish Jews than in Kurds poses a special problem.

It is suggested that the deficiency is almost certainly, like Haemoglobin S, protective against falciparum malaria. Malaria does not appear to have been endemic in the mountains of Kurdistan. However, the extreme contrast between the frequencies of the deficiency among the Kurdish Jews on the one hand and among the non-Jewish Kurds on the other makes it almost certain that the very high frequencies in the Kurdish Jews and

other oriental Jews is due mainly either to their ancestry or their way of life, rather than to any natural selection resulting from a common malarial environment.

We must therefore look for a cause for the high frequency in the Jews in some circumstances peculiar to their own ancestry or mode of life. The gene itself may have accompained the Jewish community, which presumably migrated a very long time ago from Israel at the time of Assyrian deportations. It is unlikely, however, that it is solely through inbreeding that the high frequency found in this community has persisted and even increased in the long subsequent period, in the absence of some special environmental influence favouring this normaly slightly deleterious gene. It seems unlikely also that there has been any substantial amount of immigration in recent centuries.

One is led to look for some cultural peculiarity resembling in a general way that found by Undevia (1969) in the parsis of Bombay. These have a significantly higher incidence of G6PD deficiency than their Hindu neighbours, and he shows that this was associated with a former much higher incidence of malaria in the Parsi community. Public health investigations had shown that this was due to the open bathing tanks attached to Parsi dwellings which, though favouring hygiene in other respcts, also enabled malarial mosquitos to breed.

There is, however, no new evidence as to why it has operated strongly on Jews but hardly at all on Kurds. The only suggestion is still that of Godber et al (1973): " some Jewish hygienic practices, as with the Parsis of Bombay, may in some way have promoted the breeding of malaria carrying mosquitoes near their dwellings."

Regarding neighbouring populations, the frequency of the $Gd^-$ gene in the populations of the Caucasus ranges from zero in the Assyrians to 62.30 percent in the Jews (Voronov,1981). It can be seen from the table that in the Caucasus, as in Iran, the non-Jewish populations exhibit lower frequencies of the deficiency compared with the high ones in the Jews.

It should be pointed out that two neighbouring populations of different ethnic origin differ markedly in the frequency of abnormality. This observation refers to the mountain Jews of the Caucasus among whom the deficiency is frequent, and the Circassian Moslems living in the same region among whom it is absent.

If we accept the hypothesis of balanced polymorphism produced by the protective action of the enzyme deficiency against the lethal effect of falciparum malaria, then the findings of significantly different frequencies in neighbouring communities suggest a difference of their ethnic origin.

In a community where the abnormality is present, it could be concentrated by the protective action against a lethal environmental factor, whereas in a community originally free from the enzyme deficiency it do not arise throughout centuries of living in malarial areas.

Thus, these findings support the view, that G6PD deficiency may constitute a significant ethnological marker for the study of the origins of neighbouring communities.

The frequency of the $Gd^-$ gene in the populations of Turkey varies between zero in the Armenians and 11.43 percent in the Eti-Turks ( Say et al, 1965). On the whole, it seems that the overall incidence of G6PD deficiency in Turkish populations is low.

The frequency of the $Gd^-$ gene in the populations of Iraq ranges from zero in the Karaite Jews (Goldschmidt et al,1976) to 7.63 percent in the Kurdish Jews (Szeinberg, 1963). Again, as in Iran, the Iraqi Kurdish Jews exhibit very high frequencies of the enzyme deficiency. The zero frequency of the enzyme deficiency in the Karaite Jews of Iraq is somewhat surprising since non-Karaite Iraqi Jews have a high prevalence of G6PD deficiency; the gene frequency is approximately 25 percent (Sheba et al, 1961; Szeinberg, 1963). This observation confirms the isolation of the Karaite from other Jews in Iraq.

A $Gd^-$ gene frequency of 18.96 percent is reported by Shaker et al (1966) in the Kuwaiti population. This finding indicates that the gene for G6PD deficiency is quite prevalent in Kuwait.

The state of Kuwait, an arid land, is known to be free from malaria, although it is surrounded by several countries where malaria is highly endemic, especially Iraq and Saudi Arabia. This finding of a high frequency of G6PD deficiency would seem to contradict the malaria protection theory of Allison (1963), who in fact stated that the only convincing argument against his hypothesis would be " the finding of high frequencies of abnormal haemoglobin or G6PD deficiency genes among populations known to have lived for long periods in malaria-free environments", and that " no such populations have yet been found to exist". The discrepancy, however, could be explained by the intermingling of the racial groups in the Gulf area over the ages and the fact that the present day inhabitants of Kuwait originally migrated, early in the eighteenth century, from neighbouring Saudi Arabia, where malaria is

heavily endemic (Dickson, 1956), and later from other neighbouring countries, mainly Iraq and Iran, where malaria is also endemic.

The frequency of the $Gd^-$ gene in the populations of Saudi Arabia ranges from 2.94 percent in the Sunnis to 65.38 percent in the Shias ( Gelpi, 1965). These findings indicate that G6PD deficiency is significantly elevated in Saudi Arabians, as it is in other Arab populations, the national average being 31.67 percent.

A relatively lower frequency (10 percent) of the enzyme deficiency is reported by Kamel et al (1980) in the Abu-Dhabians of the United Arab Emirates.

The $Gd^-$ gene frequency in the populations of Pakistan varies between 1.42 percent in the Bengalis ( Ronald et al, 1968) and 7.89 percent in the Pathans (Stern et al, 1968). On the whole, with a national average of 3.30 percent, the overall incidence of the enzyme deficiency seems to be low in the Pakistani population.

Similar low frequencies ranging from 0.97 percent in the Pushtus (Papiha et al, 1977) to 10.34 percent in the Jews (Sheba et al, 1961) characterize the Afghan population, the national average being 4.9 percent.

## Conclusion

Glucose-6-phosphate dehydrogenas deficiency, especially that of the $B^-$ type can, particularly through favism, lead to serious illness with appreciable mortality. The genetical problem which it presents is thus similar to that of the abnormal haemoglobins and the thalassaemias. We need to explain how a gene with deleterious effects can nevertheless maintain very high frequencies in certain populations. In certain areas,

and especially in the Mediterranean region, the distribution of the gene shows a considerable degree of correspondence with that of present or recent malignant tertian malaria.There is also some direct evidence for protection of the individual against the effects of malarial infection(Allison and Clyde, 1961; Bienzle et al, 1972; Harris and Gilles, 1961). The deficiency is found in a belt extending from the Mediterranean area through south west Asia and India to south East Asia including Indonesia(Mourant et al, 1976).

Though quite high frequencies are found in the indigenous populations in many parts of Iran where malaria was formerly endemic, the highest frequencies of deficiency yet recorded are in the Kurdish Jews of Iran and Iraq. The Iranian Kurds carry the gene, but with a much lower frequency than the Kurdish Jews who live among them. It is difficult to account in terms of exposure to malaria for the marked difference between two populations living in such close proximity. Moreover, malaria does not appear to have been endemic in the mountainous area of Kurdistan.

However, the extreme contrast between the frequencies of the deficiency among the Kurdish Jews on the one hand and among the non-Jewish Kurds on the other makes it almost certain that the very high frequencies in the Kurdish Jews and other oriental Jews is due mainly either to their ancestry or their way of life, rather than to any natural selection resulting from a common malarial environment.There is, however, no new evidence as to why it has operated strongly on Jews but hardly at all on Kurds. The only suggestion is still that of Godber et al (1973):" some Jewish hygienic practices, as with the Parsis of Bombay(Undevia, 1969),may in some way have

promoted the breedings of malaria carrying mosquitoes near their dwellings."

The only other population known to have G6PD deficiency frequencies comparable with those of the Kurdish Jews consists of the indigenous Shia Moslems of the formerly malarial eastern Oases of Saudi Arabia. The more recent immigrants of the Sunni Moslem sect appear to have considerably lower frequencies of G6PD deficiency.

Finally, apart from the Kurdish Jews of western Iran and Iraq with their extremely high frequencies of G6PD deficiency and also the Shia Moslem Arabs of Saudi Arabia with high frequencies, comparable with those of the Kurdish Jews, there are substantial frequencies of this condition in most other populations in Iran and in neighbouring countries both in the indigenous populations and in Jews, but in general the condition is commoner in Jews.

Table 5.3.7. I Red cell Glucose-6-Phosphate dehydrogenase(G6PD) deficiency distribution in Iran ( Males only)

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors |
|---|---|---|---|---|---|
| 1-Zoroastrians.Yazd | 146 | 146 | - | 0.00 | Bowman,J.E.,& Walker,D. 1961 G. |
| 2-Armenians.Esfahan | 158 | 157 | 1 | 0.63 | Bowman,J.E.,& Walker,D. 1961 G. |
| 3-Zoroastrians.Yazd | | | | 1.00 | Beaconsfield,P.,et al. 1967 |
| 4-Gonbad | 82 | 81 | 1 | 1.20 | Kirk,R.L.,et al. 1977 |
| 5-Yazd | | | | 2.50 | Beaconsfield,P.,et al. 1967 |
| 6-Kurds.Sanandaj | 106 | 103 | 3 | 2.83 | Lehmann,H., et al. 1973 |
| 7-Kurds.Baneh,Marivan | 77 | 72 | 5 | 6.49 | Lehmann,H., et al. 1973 |
| 8-Tavalesh,Astara | 31 | 29 | 2 | 6.50 | Kirk,R.L., et al. 1977 |
| 9-Chah Bahar,Jask | 142 | 132 | 10 | 7.04 | Hedayat,S.,et al. 1969 |
| 10-Mashhad | | | | 7.50 | Beaconsfield,P.,et al. 1967 |
| 11-Kazeroun | | | | 7.50 | Beaconsfield,P.,et al. 1967 |
| 12-Moslems.Fars | 984 | 906 | 78 | 7.93 | Bowman,J.E.,& Walker,D. 1961 G. |
| 13-Esfahan | | | | 9.00 | Beaconsfield,P.,et al. 1967 |
| 14-Shiraz | | | | 9.00 | Beaconsfield,P.,et al. 1967 |

Table 5.3.7.I (Cont.) Red cell Glucose-6-Phosphate dehydrogenase (G6PD)deficiency distribution in Iran (Males only)

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors | |
|---|---|---|---|---|---|---|
| 15-Moslems.Shiraz | 221 | 201 | 20 | 9.05 | Bowman,J.E., & Ronaghy, H. | 1967 |
| 16-Moslems.Tehran | 557 | 502 | 55 | 9.87 | Hedayat,S.,et al. | 1969 |
| 17-Jews.Esfahan,Shiraz, Tehran | 370 | 330 | 40 | 10.81 | Szeinberg,A. | 1963 |
| 18-Ghashghais | 133 | 118 | 15 | 11.28 | Bowman,J.E., & Walker, D.G. | 1961 |
| 19-Jews | 139 | | | 12.00 | Simhai,B. | 1974 |
| 20-Jews.Tehran | 108 | 95 | 13 | 12.04 | Hedayat,S.,et al. | 1969 |
| 21-Babol,Shahi,Amol | 32 | 28 | 4 | 12.50 | Kirk,R.L.,et al. | 1977 |
| 22-Basseris.Fars | 83 | 72 | 11 | 13.25 | Bowmann,J.E.,& Walker, D.G. | 1961 |
| 23-Jews.Esfahan | | | | 14.00 | Beaconsfield,P.,et al. | 1967 |
| 24-Northern Gorgan | 21 | 18 | 3 | 14.30 | Kirk,R.L.,et al. | 1977 |
| 25-Armenians.Tehran | 102 | 87 | 15 | 14.71 | Hedayat,S.,et al. | 1969 |
| 26-Jews | 557 | 473 | 84 | 15.08 | Sheba,C.,et al. | 1961 |

Table 5.3.7.I (Cont.) Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Iran (Males only).

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors | |
|---|---|---|---|---|---|---|
| 27-Shahsavar,Rudsar,Rud, bar,Rasht,Langarud, Lahijan,Bandar-Pahlavi | 43 | 36 | 7 | 16.30 | Kirk,R.L.,et al. | 1977 |
| 28-Southern Gorgan, Behshahr,Sari | 28 | 23 | 5 | 17.90 | Kirk,R.L.,et al. | 1977 |
| 29-Kermanshah | | | | 19.00 | Beaconsfield,P.,et al. | 1967 |
| 30-Mamassanis.Fars | 91 | 73 | 18 | 19.78 | Bowman,J.E., & Walker,G. | D.1961 |
| 31-Kurds.Kermanshah | | | | 25.00 | Beaconsfield,P.,et al. | 1967 |
| 32-Caspian Littoral area | | | | 25.00 | Beaconsfield,P.,et al. | 1967 |
| 33-Jews.Kermanshah | | | | 29.00 | Beaconsfield,P.,et al. | 1967 |
| 34-Kurdish Jews | 63 | 40 | 23 | 36.51 | Tills,D.,et al. | 1977 |
| 35-Kurdish Jews | 57 | 35 | 22 | 38.60 | Godber,Marilyn.J., et al. | 1973 |
| 36-Kurdish Jews,Kermanshah,Sanandaj | 45 | 25 | 20 | 44.44 | Szeinberg,A. | 1963 |

Table 5.3.7.I (Cont.) Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Iran (Males only)

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors | |
|---|---|---|---|---|---|---|
| 37-Kurdish Jews | 196 | 82 | 114 | 58.16 | Sheba,C.,et al. | 1961 |

Table 5.3.7.b. I Red cell Glucose-6-phosphate dehydrogenase(G6PD) types in Iran ( Males onlY)

| Population | Number Tested | Phenotypes | | | | Gene frequencies | | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | Normal | | Deficient | | | | | | | |
| | | A+ | B+ | A- | B- | $Gd^{A+}$ | $Gd^{B+}$ | $Gd^{A-}$ | $Gd^{B-}$ | | |
| 1-Jurds.Sanandaj | 106 | 103 | | 3 | | 97.17 | | 2.83 | | Lehmann,H.,et al. | 1973 |
| 2-Kurds.Baneh, Marivan | 77 | 72 | | 5 | | 93.51 | | 6.49 | | Lehmann,H.,et al. | 1973 |
| 3-Kurdish Jews | 57 | 35 | | 22 | | 61.40 | | 38.60 | | Godber,Marilyn J., et al. | 1973 |

Table 5.3.7. II Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in the Caucasus (Males only )

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors | |
|---|---|---|---|---|---|---|
| 1-Assyrians | 289 | | | 0.00 | Voronov,A.A. | 1981 |
| 2-Circassians | 57 | | | 0.00 | Sheba,C.,et al. | 1961 |
| 3-Ingushes | 210 | | | 0.00 | Voronov, A.A. | 1981 |
| 4-Imeritins.Georgian | 421 | | | 0.20 | Voronov. A.A. | 1981 |
| 5-Megrels.Georgian | 817 | | | 0.30 | Voronov, A.A. | 1981 |
| 6-Gurians.Georgian | 625 | | | 0.30 | Voronov, A.A. | 1981 |
| 7-Cahetins.Georgian | 212 | | | 0.50 | Voronov, A.A. | 1981 |
| 8-Armenians | 5161 | | | 0.90 | Voronov, A.A. | 1981 |
| 9-Chechens | 691 | | | 1.30 | Voronov, A.A. | 1981 |
| 10-Talyshes.Azerbaijan | 237 | | | 8.00 | Voronov, A.A. | 1981 |
| 11-Azerbaijanians | 18720 | | | 9.90 | Voronov, A.A. | 1981 |
| 12-Avars.Daghestan | 100 | | | 19.00 | Voronov, A.A. | 1981 |
| 13-Udins.Azerbaijan | 75 | | | 21.40 | Voronov, A.A. | 1981 |
| 14-Jews | 25 | 18 | 7 | 28.00 | Sheba,C.,et al. | 1961 |
| 15-Mountain Jews | 90 | | | 62.30 | Voronov, A.A. | 1981 |

Table 5.3.7.III Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Turkey ( Males only )

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors | |
|---|---|---|---|---|---|---|
| 1-Armenians.Istanbul | 44 | 44 | - | 0.00 | Say,B.,et al. | 1965 |
| 2-Jews.Istanbul | 29 | 29 | - | 0.00 | Say,B.,et al. | 1965 |
| 3-Istanbul | 37 | 37 | - | 0.00 | Say,B.,et al. | 1965 |
| 4-Asia Minor.Rize. Kurdish speakers | 109 | 109 | - | 0.00 | Say,B.,et al. | 1965 |
| 5-Asia Minor,Ankara.Neonates | 1000 | 995 | 5 | 0.50 | Say,B.,et al. | 1965 |
| 6-Asia Minor,Izmir | 212 | 210 | 2 | 0.94 | Say,B.,et al. | 1965 |
| 7-Asia Minor,Diarbakir. Kurdish speakers | 208 | 204 | 4 | 1.92 | Say,B.,et al. | 1965 |
| 8-Jews | 256 | 251 | 5 | 1.95 | Sheba,C.,et al. | 1961 |
| 9-Jews | 93 | 91 | 2 | 2.15 | Sheba,C.,et al. | 1961 |
| 10-Turks abroad | 71 | 69 | 2 | 2.82 | Kattamis,C.A.,et al. | 1969 |
| 11-Kurds | | | | 4.00 | Richard,P. | 1976 |
| 12-Turks. Antalya | 73 | | | 5.40 | Aksoy,M.,et al. | 1980 |
| 13-Eti-Turks | 637 | 593 | 44 | 6.50 | Altay,C.,et al. | 1978 |

Table 5.3.7. III (Cont.) Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Turkey (Males only )

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors | |
|---|---|---|---|---|---|---|
| 14-Eti-Turks. Adana,Tarsus | 105 | 93 | 12 | 11.43 | Say,B.,et al. | 1965 |

Table 5.3.7. IV Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Iraq ( Males only)

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors | |
|---|---|---|---|---|---|---|
| 1-Karaite Jews | 29 | 29 | - | 0.00 | Goldschmidt,E.,et al. | 1976 |
| 2-Moslems | 305 | 267 | 38 | 12.40 | Hamamy,H.A.& Saeed,T. Kh. | 1981 |
| 3-Jews.Baghdad | 286 | 216 | 70 | 24.48 | Szeinberg, A. | 1963 |
| 4-Jews | 902 | 678 | 224 | 24.83 | Sheba,C.,et al. | 1961 |
| 5-Kurdish Jews | 59 | 38 | 21 | 35.59 | Szeinberg, A. | 1963 |
| 6-Kurdish Jews | 14 | 6 | 8 | 57.14 | Godber,Marilyn.J., et al. | 1973 |
| 7-Kurdish Jews.South east | 19 | 8 | 11 | 57.89 | Tills,D.,et al. | 1977 |
| 8-Kurdish Jews.North west | 36 | 11 | 25 | 69.44 | Tills,D.,et al. | 1977 |
| 9-Kurdish Jews | 126 | 37 | 89 | 70.63 | Szeinberg, A. | 1963 |

Table 5.3.7. V Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Kuwait (Males only)

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors |
|---|---|---|---|---|---|
| 1-Kuwaitis | 211 | 171 | 40 | 18.96 | Shaker, Yehia.,et al. 1966 |

Table 5.3.7. VI Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Saudi Arabia ( Males only )

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors | |
|---|---|---|---|---|---|---|
| 1-Sunnis .Eastern Provinces (except Al Qatif & Al Hasa) | 34 | 33 | 1 | .2.94 | Gelpi,A.P. | 1965 |
| 2-Western Provinces | 97 | 93 | 4 | 4.12 | Gelpi,A.P. | 1965 |
| 3-Shias & Sunnis. Al .Hasa | 64 | 61 | 3 | 4.69 | Gelpi,A.P. | 1965 |
| 4-Shias. Al Hasa,Qarah | 104 | 79 | 25 | 24.04 | Gelpi,A.P. | 1965 |
| 5-Shias.Al Qatif | 111 | 73 | 38 | 34.23 | Gelpi,A.P. | 1965 |
| 6-Shias.Al Hasa, Mansurah | 92 | 43 | 49 | 53.26 | Gelpi,A.P. | 1965 |
| 7-Shias. Al Qatif,Safwah | 51 | 18 | 33 | 64.71 | Gelpi,A.P. | 1965 |
| 8-Shias. Al Qatif, Al Ajman | 104 | 36 | 68 | 65.38 | Gelpi,A.P. | 1965 |

Table 5.3.7. VII Red cell Glucose-6-Phosphate dehydrogenase(G6PD) deficiency distribution in the United Arab Emirates (Males only)

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors |
|---|---|---|---|---|---|
| 1-Abu-Dhabians | 100 | 90 | 10 | 10.00 | Kamel, K., et al. 1980 |

Table 5.3.7. VIII Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Pakistan ( Males only )

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors | |
|---|---|---|---|---|---|---|
| 1-Bengalis.West | 141 | 139 | 2 | 1.42 | Ronald,A.R.,et al. | 1968 |
| 2-Pathans.West | 60 | 59 | 1 | 1.67 | McCurdy,P.R., et al. | 1970 |
| 3-Former residents of India | 45 | 44 | 1 | 2.22 | Ronald,A.R.,et al. | 1968 |
| 4-Punjabis.West | 133 | 130 | 3 | 2.25 | McCurdy,P.R.,et al. | 1970 |
| 5-Punjabis.West | 185 | 180 | 5 | 2.70 | Ronald,A.R.,et al. | 1968 |
| 6-Other than Pathans and Punjabis.West | 28 | 27 | 1 | 3.57 | McCurdy,P.R.,et al. | 1970 |
| 7-Pathans.West | 85 | 81 | 4 | 4.71 | Ronald,A.R.,et al. | 1968 |
| 8-Pathans.West | 114 | 105 | 9 | 7.89 | Stern,M.A., et al. | 1968 |

Table 5.3.7.IX Red cell Glucose-6-Phosphate dehydrogenase (G6PD) deficiency distribution in Afghanistan (Males only )

| Population | Number Tested | Normal | Deficient | $Gd^-$ | Authors |
|---|---|---|---|---|---|
| 1-Pushtus | 103 | 102 | 1 | 0.97 | Papiha,S.S.,et al. 1977 |
| 2-Daris | 177 | 171 | 6 | 3.39 | Papiha,S.S.,et al. 1977 |
| 3-Jews | 29 | 26 | 3 | 10.34 | Sheba,C.,et al. 1961 |

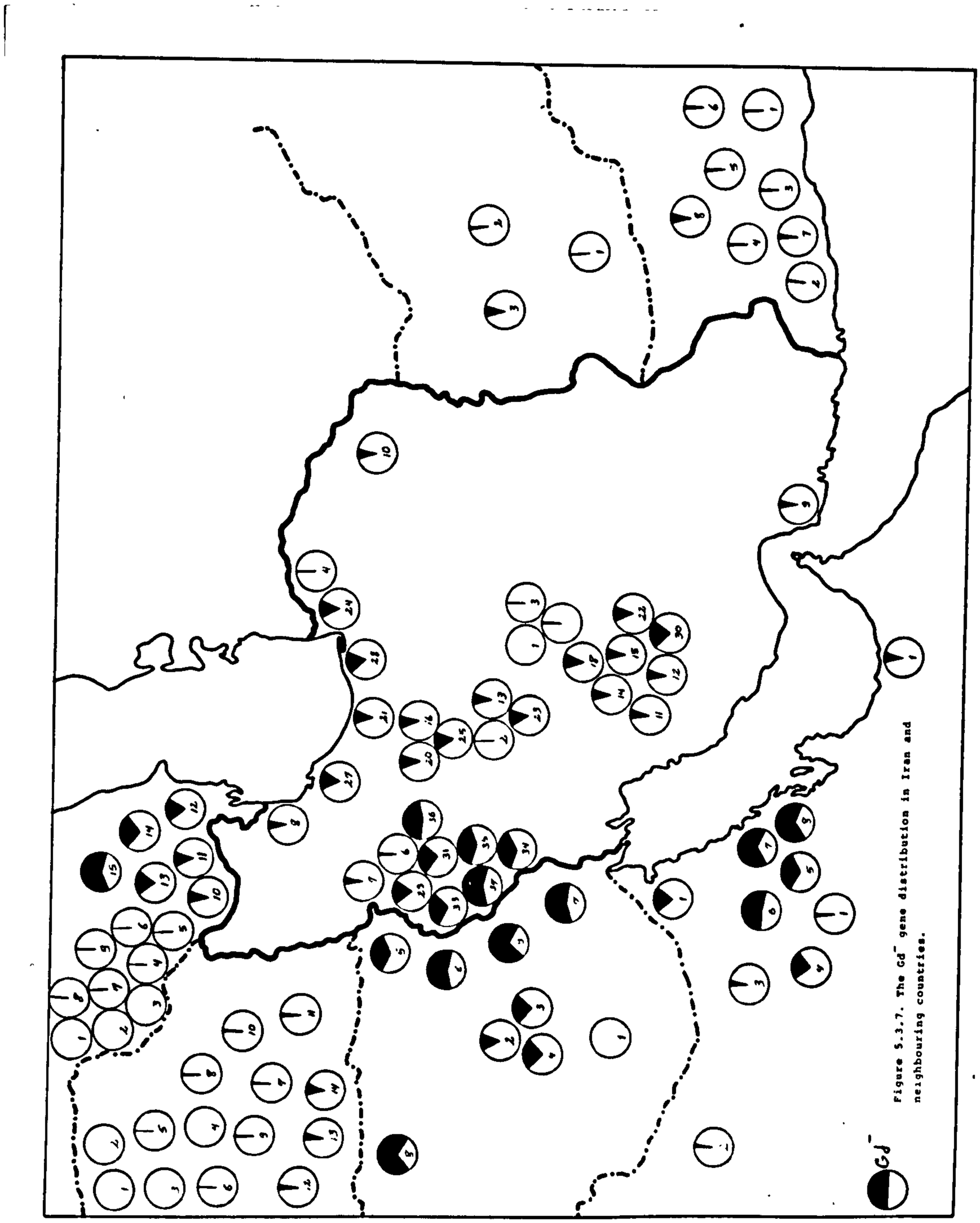



Figure 5.3.7. The $Gd^-$ gene distribution in Iran and neighbouring countries.

5.3.8 The lactate dehydrogenase ( LDH) system

The distribution of lactate dehydrogenase types in Iranian and neighbouring populations is presented in Tables 5.3.8.

With the exception of one Calcutta 1 type in the 463 specimens from the Caspian sea area of Iran (Kirk et al,1977), all the samples from Iran and neighbouring countries showed the common LDH type.

Conclusion

Variants of Lactate dehydrogenase are all very rare, occurring with a frequency of the order of 1 in 1000 in most populations. Type Calcutta 1 has a frequency somewhat over 1 percent in various Indian populations(Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of one Calcutta 1 type found in the Caspian sea area of Iran, the only type was found to be the Normal LDH type.

Table 5.3.8.I. Red cell Lactate dehydrogenase (LDH) types in Iran

| Population | Number Tested | Phenotypes | | %Variant | Authors |
|---|---|---|---|---|---|
| | | LDH.N(Normal) | LDH Variant | | |
| 1-Kurds.Sanandaj | 105 | 105 | - | - | Lehmann,H.,et al. 1973 |
| 2-Tehran | 132 | 132 | - | - | Farhud,D.D.,& Walter, H.1973 |
| 3-Tehran | 165 | 165 | - | - | Sawhney,K.S. 1975 |
| 4-Esfahan | 89 | 89 | - | - | Sawhney,K.S. 1975 |
| 5-Caspian Sea area | 463 | 462 | 1Calcutta 1 | 0.22 | Kirk,R.L.,et al. 1977 |

Table 5.3.8. V. Red cell Lactate dehydrogenase (LDH) types in Kuwait

| Population | Number Tested | Phenotypes | | %Variant | Authors | |
|---|---|---|---|---|---|---|
| | | LDH.N(Normal) | LDH Variant | | | |
| 1-Kuwaiti Arabs | 150 | 150 | - | - | Sawhney,K.S. | 1975 |
| 2-General population | 89 | 89 | - | - | Khaled,E.,et al. | 1981 |
| 3-Suluba tribe | 52 | 52 | - | - | Khaled,E.,et al. | 1981 |
| 4-Ajman tribe | 52 | 52 | - | - | Khaled,E.,et al. | 1981 |

Table 5.3.8. VI Red cell Lactate dehydrogenase (LDH) types in Saudi Arabia

| Population | Number Tested | Phenotypes | | %Variant | Authors |
|---|---|---|---|---|---|
| | | LDH.N(Normal ) | LDH Variant | | |
| 1-Western Saudi Arabia | 245 | 245 | - | - | Saha,N.,et al. 1980 |

Table 5.3.8. IX Red cell Lactate dehydrogenase (LDH) types in Afghanistan

| Population | Number Tested | Phenotypes | | % Variant | Authors |
|---|---|---|---|---|---|
| | | LDH.N (Normal) | LDH Variant | | |
| 1-Pushtus | 103 | 103 | - | - | Papiha,S.S.,et al. 1977 |
| 2-Daris | 177 | 177 | - | - | Papiha,S.S.,et al. 1977 |

## 5.3.9. The malate dehydrogenase (MDH) system

The distribution of malate dehydrogenase types in Iranian and neighbouring populations is shown in Tables 5.3.9.

With the exception of one MDH5-1 type in the 463 specimens from the Caspian sea area of Iran (Kirk et al, 1977),all the samples from Iran and neighbouring countries showed only the common MDH1-1 phenotype.

The MDH variant detected in a single individual in the Caspian sea sample was described by Blake (1977). It does not correspond with any previously reported MDH variant and the Caspian phenotype has been designated s-MDH5-1.

Conclusion

Many thousands of persons from Europe, the Near East and Asia have shown only the common MDH1-1 homozygous type (Mourant et al, 1976).

In Iranian and neighbouring populations, as in Europeans and in other Asiatic populations, the only type was found to be the normal MDH1-1 phenotype.

Table 5.3.9.I Red cell Malate dehydrogenase (MDH) types in Iran

| Population | Number Tested | Phenotypes MDH.N(Normal) 1-1 | Phenotypes MDH Variant | % Variant | Authors | |
|---|---|---|---|---|---|---|
| 1-Kurds | 182 | 182 | - | - | Leakey,T.E.B.,et al. | 1972 |
| 2-Tehran | 165 | 165 | - | - | Sawhney,K.S. | 1975 |
| 3-Esfahan | 89 | 89 | - | - | Sawhney,K.S. | 1975 |
| 4-Caspian Sea area | 463 | 462 | 1s-MDH5-1 | 0.22 | Kirk,R.L.,et al. | 1977 |

Table 5.3.9. V. Red cell Malate dehydrogenase(MDH) types in Kuwait

| Population | Number Tested | Phenotypes | | %Variant | Authors |
|---|---|---|---|---|---|
| | | MDH.N(Normal) 1-1 | MDH Variant | | |
| 1-Kuwaiti Arabs | 150 | 150 | - | - | Sawhney,K.S. 1975 |

Table 5.3.9. VI Red cell Malate dehydrogenase (MDH) types in Saudi Arabia

| Population | Number Tested | Phenotypes | | %Variant | Authors |
|---|---|---|---|---|---|
| | | MDH.N (Normal ) 1-1 | MDH Variant | | |
| 1- Western Saudi Arabia | 93 | 93 | - | - | Saha,N.,et al. 1980 |

Table 5.3.9. IX Red cell Malate dehydrogenase (MDH) types in Afghanistan

| Population | Number Tested | Phenotypes | | %Variant | Authors |
|---|---|---|---|---|---|
| | | MDH.N(Normal) 1-1 | MDH Variant | | |
| 1-Pushtus | 103 | 103 | - | - | Papiha,S.S.,et al. 1977 |
| 2-Daris | 177 | 177 | - | - | Papiha,S.S.,et al. 1977 |

## 5.3.10. The Phosphohexose isomerase (PHI) system

The distribution of phosphohexose isomerase types and respective gene frequencies in Iranian populations is presented in Table 5.3.10.

Information on the distribution of the PHI genes in neighbouring populations is lacking.

The Iranian sample consists of four population groups. Comparing the four groups, Farhud et al (1973) and Sawhney (1975) found no PHI3-1 phenotype in the 125 Tehrani and the 88 Esfahani Iranians studied by them, but Sawhney (1975) and Kirk et al (1977) found two PHI3-1 phenotypes in the 165 Tehrani sample and seven in the 463 sample from the Caspian sea area, respectively. Detter et al (1968) found that the Asiatic populations exhibit an appreciable frequency of the allele $PHI^3$.

$PHI^3$ frequencies of 0.61 and 0.76 percent in Iranians confirm this tendency. Also, these frequencies are comparable with the values given by Blake et al (1971) for the North Indians.

Conclusion

The PHI system is of potential interest because the $PHI^3$ allele frequency is found to be higher in the Asiatic populations. Like the Asiatics, the Iranians also show an appreciable frequency of the $PHI^3$ allele. This finding suggests the importance of conducting further studies in Iranian and neighbouring populations.

Table 5.3.10. I Red cell Phosphohexose isomerase (PHI) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | PHI 1-1 | PHI 3-1 | $PHI^1$ | $PHI^3$ | | |
| 1-Tehran | 125 | 125 | - | 100.00 | 0.00 | Farhud,D.D.,et al. | 1973 |
| 2-Esfahan | 88 | 88 | - | 100.00 | 0.00 | Sawhney,K.S. | 1975 |
| 3-Tehran | 165 | 163 | 2 | 99.39 | 0.61 | Sawhney,K.S. | 1975 |
| 4-Caspian Sea area | 463 | 456 | 7 | 99.24 | 0.76 | Kirk,R.L.,et al. | 1977 |

## 5.3.11. The glutamic pyruvic transaminase (GPT) system

The distribution of GPT types and respective gene frequencies in Iranian and neighbouring populations is set out in Tables 5.3.11.

There are a few reports on GPT studies in Iranian and neighbouring populations.

The only report on Iranians is that of Kirk et al (1977) who studied six areas in the Caspian Littoral of Iran. The frequency of the $GPT^2$ allele ranges from 38.50 to 50.00 percent in northern Iran, being lowest in Tavalesh, Astara and highest in northern Gorgan. With an average $GPT^2$ frequency of 42.62 percent, the population of northern Iran appears to exhibit a lower $GPT^2$ frequency than that of 47.67 percent in Europeans (Kompf and Ritter, 1979).

Regarding neighbouring areas, the $GPT^2$ frequency of 45.60 percent in the Turks of Turkey ( Brinkmann et al,1973) is higher than the average for northern Iran but lower than the European frequency.

The $GPT^2$ frequency of 41.10 percent in the Iraqi Jews is slightly lower than that in Iranians and lower than the frequency in Europeans.

The frequency of the $GPT^2$ gene being 38.20 percent in Saudi Arabians (Goedde et al, 1979) is also lower than that in Iranians and much lower than the European frequency.

The $GPT^2$ allele frequency in the populations of Afghanistan ranges from 50.41 percent in the Uzbeks to 55.81 percent in the Tajiks (Goedde et al, 1977). With an average $GPT^2$ frequency of 53.05 percent, the Afghan population appears to exhibit a much higher $GPT^2$ frequency than that found in Iranians and even higher than the frequency in Europeans.

Conclusion

The European populations frequencies of the $GPT^2$ gene lie between 46.10 and 49.66 percent (Kompf and Ritter,1979). Frequencies of the $GPT^2$ allele, ranging from 55.15 to 64.63 percent, are higher in the Indian region than in Europe (Blake, 1979).

In Chinese the $GPT^2$ gene frequencies are around 50 percent (Chen et al, 1973; Blake, 1976).

In south African Bantus it is only 12.71 percent(Blake,1976).

In Iranian and neighbouring populations, with the exception of the Afghans with their higher values, frequencies of the $GPT^2$ gene appear to be lower than those in Europeans.

Table 5.3.11. I. Red cell Glutamic Piruvic transaminase(GPT) phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | GPT 1-1 | GPT 2-1 | GPT 2-2 | $GPT^1$ | $GPT^2$ | | |
| 1-Tavalesh,Astara | 61 | 28 | 19 | 14 | 61.50 | 38.50 | Kirk,R.L.,et al. | 1977 |
| 2-Babol,Shahi,Amol | 64 | 23 | 32 | 9 | 60.90 | 39.10 | Kirk,R.L.,et al. | 1977 |
| 3-Southern Gorgan Behshahr,Sari | 53 | 19 | 26 | 8 | 60.40 | 39.60 | Kirk,R.L.,et al. | 1977 |
| 4-Shahsavar,Rudsar,Rudbar, Langarud,Lahijan,Bandar-Pahlavi | 86 | 31 | 37 | 18 | 57.60 | 42.40 | Kirk,R.L.,et al. | 1977 |
| 5-Gonbad | 155 | 47 | 73 | 35 | 53.90 | 46.10 | Kirk,R.L.,et al. | 1977 |
| 6-Northern Gorgan | 44 | 9 | 26 | 9 | 50.00 | 50.00 | Kirk,R.L.,et al. | 1977 |

Table 5.3.11.III Red cell Glutamic Piruvic transaminase (GPT) phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | GPT 1-1 | GPT 2-1 | GPT 2-2 | $GPT^1$ | $GPT^2$ | |
| 1-Turks | 213 | | | | 54.40 | 45.60 | Brinkmann,B.,et al. 1973 |

Table 5.3.11. IV Red cell Glutamic Piruvic transaminase (GPT) phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | GPT 1-1 | GPT 2-1 | GPT 2-2 | $GPT^1$ | $GPT^2$ | |
| 1- Jews | 192 | | | | 58.90 | 41.10 | Lahav,M.,et al. 1972 |

Table 5.3.11. VI Red cell Glutamic Piruvic transaminase (GPT)phenotypes and gene frequencies distribution in Saudi Arabia

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|---|
| | | GPT 1-1 | GPT 2-1 | GPT 2-2 | $GPT^1$ | $GPT^2$ | |
| 1-Saudi Arabians | 359 | 123 | 198 | 38 | 61.80 | 38.20 | Goedde,H.W.,et al. 1979 |

Table 5.3.11. IX Red cell Glutamic Piruvic transaminase(GPT) phenotypes and gene frequencies distribution in Afghanistan

| Population | Number Tested | Phenotypes | | | | | Gene frequencies | | | | Authors |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | GPT 1-1 | GPT 2-1 | GPT 2-2 | GPT 3-2 | GPT 6-2 | $GPT^1$ | $GPT^2$ | $GPT^3$ | $GPT^6$ | |
| 1-Uzbeks | 124 | 29 | 65 | 30 | - | - | 49.59 | 50.41 | 0.00 | 0.00 | Goedde,H.W.,et al.1977 |
| 2-Hazaras | 174 | 37 | 94 | 42 | - | 1 | 48.27 | 51.45 | 0.00 | 0.28 | Goedde,H.W.,et al.1977 |
| 3-Pushtus | 210 | 44 | 103 | 63 | - | - | 45.47 | 54.53 | 0.00 | 0.00 | Goedde,H.W.,et al.1977 |
| 4-Tajiks | 310 | 59 | 155 | 95 | 1 | - | 44.03 | 55.81 | 0.16 | 0.00 | Goedde,H.W.,et al.1977 |

## 5.3.12. Other red cell enzyme systems

In this part, data on some other red cell enzyme systems are presented. Since data available on these systems are not representative for the region, no discussion will be given.

Table 5.3.12. a. I Red cell phosphoglucomutase Locus 2($PGM_2$) in Iran

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | $PGM_2$ 1-1 | $PGM_2$ 9-1 | $PGM_2^1$ | $PGM_2^9$ | |
| Caspian Sea area | 463 | 459 | 4 | 99.57 | 0.43 | Kirk, R.L., et al. 1977 |

Table 5.3.12.a.IV. Red cell phosphoglucomutase Locus 2($PGM_2$) in Iraq

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | $PGM_2$ 1-1 | $PGM_2$ rare | $PGM_2^1$ | $PGM_2^{rare}$ | |
| Jews | 69 | 69 | - | 100.00 | 0.00 | Hopkinson,D.A.,et al. 1966 |

Table 5.3.12.a. IX Red cell phosphoglucomutase Locus 2($PGM_2$) in Afghanistan

| Population | Number tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | $PGM_{1\text{-}1}^{2}$ | $PGM_2^{rare}$ | $PGM_2^1$ | $PGM_2^{rare}$ | | |
| 1-Daris | 178 | 178 | - | 100.00 | 0.00 | Papiha,S.S.,et al. | 1977 |
| 2-Pushtus | 103 | 103 | - | 100.00 | 0.00 | Papiha,S.S.,et al. | 1977 |

Table 5.3.12.b.I. Red cell peptidase A ( PeP A) in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|---|
| | | PePA 1-1 | PePA 2-1 | PePA 2-2 | $PePA^1$ | $PePA^2$ | | |
| Caspian Sea area | 463 | 459 | 3 | 1 | 99.46 | 0.54 | Kirk,R.L., et al. | 1977 |

Table 5.3.12.C.I. Red cell peptidase B( PePB) in Iran

| Population | Number Tested | Phenotypes | | | Gene frequencies | | | Authors | |
|---|---|---|---|---|---|---|---|---|---|
| | | PePB 1-1 | PePB 2-1 | PePB 3-1 | $PePB^1$ | $PePB^2$ | $PeP^3$ | | |
| Caspian Sea area | 463 | 459 | 2 | 2 | 99.56 | 0.22 | 0.22 | Kirk,R.L.,et al. | 1977 |

Table 5.3.12.d.I. Red cell peptidase C (PePC) in Iran

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | PePC 1-1 | PePC 3-1 | $PePC^1$ | $PePC^3$ | | |
| Caspian Sea area | 463 | 462 | 1 | 99.89 | 0.11 | Kirk,R.L.,et al. | 1977 |

Table 5.3.12.e.I. Red cell peptidase D ( PePD) in Iran

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | PePD 1-1 | PePD 3-1 | $PePD^1$ | $PePD^3$ | | |
| Caspian Sea area | 463 | 460 | 3 | 99.68 | 0.32 | Kirk, R.L.,et al. | 1977 |

Table 5.3.12.f.I. Red cell Isocitrate dehydrogenase ( ICD) in Iran

| Population | Number Tested | Phenotypes | Gene frequencies | Authors |
|---|---|---|---|---|
| | | ICD 1-1 | $ICD^1$ | |
| 1- Tehran | 132 | 132 | 100.00 | Farhud,D.D.,et al. 1973 |
| 2- Caspian Sea area | 463 | 463 | 100.00 | Kirk,R.L., et al. 1977 |

Table 5.3.12.g.I. Red cell phosphoglycerate Kinase(PGK) in Iran

| Population | Number Tested | Phenotypes | Gene frequencies | Authors |
|---|---|---|---|---|
| | | PGK 1-1 | $PGK^1$ | |
| Caspian Sea area | 463 | 463 | 100.00 | Kirk, R.L., et al. 1977 |

Table 5.3.12.h.I. Red cell Superoxide dismutase (SoD) in Iran

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | SoD 1-1 | SoD 2-1 | $SoD^1$ | $SoD^2$ | |
| Caspian Sea area | 463 | 459 | 4 | 99.57 | 0.43 | Kirk, R.L., et al. 1977 |

Table 5.3.12.h.VI. Red cell Superoxide dismutas ( SoD) in Saudi Arabia

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | SoD 1-1 | SoD 2-1 | $SoD^1$ | $SoD^2$ | |
| Western Saudi Arabia | 149 | 149 | - | 100.00 | 0.00 | Saha,N., et al. 1980 |

Table 5.3.12.i.I. Red cell Nicotinamide Adenine Dinucleotide (NADH) Diaphorase in Iran

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Dia 1-1 | Dia 2-1 | $Dia^1$ | $Dia^2$ | |
| Caspian Sea area | 463 | 461 | 2 | 99.78 | 0.22 | Kirk,R.L.,et al. 1977 |

Table 5.3.12.j. I. Red cell glutamic oxaloacetic transaminase(GoT) in Iran

| Population | Number Tested | Phenotypes | Gene frequencies | Authors |
|---|---|---|---|---|
| | | GoT 1-1 | $GoT^1$ | |
| Tehran | 134 | 134 | 100.00 | Farhud,D.D.,et al. 1973 |

Table 5.3.12.k. IX Red cell phosphogluco-isomerase( PGI) in Afghanistan

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | PGI 1-1 | PGI 1-3 | $PGI^1$ | $PGI^3$ | |
| 1-Daris | 178 | 177 | 1 | 99.70 | 0.30 | Papiha,S.S.,et al. 1977 |
| 2-Pushtus | 102 | 102 | - | 100.00 | 0.00 | Papiha,S.S.,et al. 1977 |

Table 5.3.12. 1. V. Red cell Catalase in Kuwait

| Population | Number Tested | Normal type | variant | Authors |
|---|---|---|---|---|
| 1- General population | 89 | 89 | - | Khaled,E.,et al. 1981 |
| 2-Ajman tribe | 52 | 52 | - | Khaled,E.,et al. 1981 |
| 3-Suluba tribe | 52 | 52 | - | Khaled,E.,et al. 1981 |

## 5.4. Genetic distance

### 5.4.1. Introduction

The study of genetic distance is now a major growth industry, and new measures of distance come off the assembly line almost as frequently as new car models (Kirk,1976).And like the automobile industry all distance measures have something in common: they manipulate variations in gene frequencies, package them in various styles and come up with a product that performs the same functions and appeals to the consumer on the basis of a variety of hard and soft 'sells'. What is of importance, however, is customer satisfaction, that is whether the product performs according to the demands made on it by the user.

Excellent reviews of the theory underlying genetic distance measurement have been provided by Balakrishnan et al (1975),Cavalli-Sforza(1973), Cannings and Cavallisforza(1974), Chakraborty et al (1976,1980,1982), Edwards and Cavalli-Sforza(1972), Goodman and Laseer (1974), Morton(1974) and Nei (1975,1978). Many of these authors have pointed out the close mathematical relationship between the various distance indices.

Several measures of genetic distance are now in common use. Those distance coefficients which make use of gene frequency results may appropriately be designated as ' genetic distances' (Constandse-westermann,1972). If two populations have precisely the same gene frequencies they will be considered to be at Zero' distance' apart. The greater the distance between the gene frequencies the greater the distance (C.A.B. Smith, 1977). Genetic distance is used for two reason: Firstly, to reduce a complicated mass of data to easily manageable and visuable form. The study of variation in gene fre-

quency at a single locus is comparatively easy. The difficulty of comprehending such variation at a large number of loci increases very rapidly, and it is necessary to have some composite measure to suggest relationships between population based on all the characteristics under consideration. This is the rational for having a measure of genetic distance( Sanghvi and Balakrishnan, 1972), and secondly to try to reconstruct something of the evolutionary history of the populations concerned (Smith, 1977). Most genetic distances care calculated from qualitative traits, which have been developed mostly over the last ten years. A massive amount of information about the different populations has been gathered and studied using other methods.

According to Constandse-westermann(1972) the coefficients which can be calculated from qualitative data (gene frequencies) may be divided into four categories.

The first category including those coefficients based on squared differences between percentages of frequency values,initially reported by Spuhler(1954).

The second category includes those distance coefficients based on the same principles as $X^2$, namely $GS^2$ considered by Edwards and Cavalli-Sforza(1972) and $DK^2$, which can be considered as a transition to a coefficient belonging to the third category.

The third category includes the coefficient by which the differences in frequency values between populations are expressed in terms of the elements of the pooled dispersion matrix of all investigated groups.

The fourth category contains those distance coefficients whose calculation is based on the angular transformation of the

original percentages of frequencies; this category was used in the present investigation for calculation of distance coefficient.Edwards and Cavalli-Sforza(1972) suggested that the angular transformation provides a measure of distance, which has some advantages over other measures, namely,that the angular transformation, being non-linear, standardises the variances and takes care of the correlations by reducing the number of dimensions and gives a better estimate of large distances. The genetic distance E is strictly comparable between different sets of data based on information from the same loci.

The study of genetic relationships between populations in Iran, either on the broad basis of comparisons across the sub-continent, or at the local level, is still in its infancy. For such an analysis, the first practical step is to collect data on gene frequencies of various polymorphic systems. This is as far as most of the work so far completed on the Iranian population goes, and indeed even at the present time no comprehensive and countrywide survey has yet been undertaken.

5.4.2. Materials and method

In the present investigation because of the large number of the population groups and the serological systems involved, finding of a considerable number of loci common to all the populations considered and calculating of a single genetic distance analysis, was impossible. Therefore, eleven groups of populations were selected on the basis of variability of gene frequency information on particular loci common to all of them. The resulting eleven sets of genetic distance

analysis together with the population groups and the loci considered in each set of analysis will be presented later.

The method employed in the present study for distance coefficient was Edwards and Cavalli-Sforza's ' new $E^2$'. The genetic distance statistic Edwards $E^2$ is a distance coefficient produced between groups for frequency data( Edwards and Cavalli-Sforza, 1972).

The Statistic $E^2$ is calculated from the following formula:

$$E^2 = 8\left(1 - \sum_{K=1}^{S_J+1} \sqrt{P_1 JK \cdot P_2 JK}\right) \Bigg/ \left(\left(1 + \sum_{K=1}^{S_J+1} \sqrt{\frac{P_1 JK}{S_J+1}}\right)\left(1 + \sum_{K=1}^{S_J+1} \sqrt{\frac{P_2 JK}{S_J+1}}\right)\right)$$

Where $P_1$ JK is the frequency of the K th class of the Jth character in the population.

SJ is the number of classes minus one.

The $E^2$ distance for the various population groups from Iran and neighbouring countries were examined on the basis of this formula, the gene frequencies data was analysed using the Fortran Program.

The output from the program consisted of:

1- A data input reprint.

2- Individual $E^2$ values for each locus separately.

3- The total $E^2$ values.

4- The standardised $E^2$ values.

Two types of analysis were used in the aid to interpretation:

1- Non-Metric Multidimensional Scaling the Program was called ' MINISSAN' from the ' MDS(X)' package.

2- Cluster Analysis-using maximum/complete link clustering.

5.4.3. Results

The results of the two types of the genetic distance analysis, based on Edwards $E^2$ standard genetic distance , for the eleven sets of data are presented in figures 5.4.3. 1-11.

Since in the eleven sets of analysis the population groups and the loci considered differ one from another, therefore, the Non-Metric two dimensional Scaling and the cluster analysis outputs (figures a and b respectively) together with the populations codesand the loci considered for each set of analysis are given separately below:

Set 1.

The 7 loci and the respective 17 alleles

| Loci | Alleles |
|---|---|
| The ABO locus | A, B, O |
| The Rh locus | D, d |
| The HP locus | $HP^1$, $HP^2$ |
| The Tf locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP locus | $AcP^A$, $AcP^B$, $AcP^C$ |
| The AK locus | $AK^1$, $AK^2$ |
| The $PGM_1$ locus | $PGM_1^1$, $PGM_1^2$ |

Set 2.

The 7 loci and the respective 18 alleles

| Loci | Alleles |
|---|---|
| The $A_1A_2BO$ locus | $A_1$, $A_2$, B, O |
| The Rh locus | D, d |
| The HP locus | $HP^1$, $HP^2$ |
| The Tf locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP locus | $AcP^A$, $AcP^B$, $AcP^C$ |
| The AK locus | $AK^1$, $AK^2$ |
| The $PGM_1$ locus | $PGM_1^1$, $PGM_1^2$ |

Set 3.

The 8 loci and the respective 19 alleles

| Loci | Alleles |
|---|---|
| The ABO locus | A, B, O |
| The Rh locus | D, d |
| The HP locus | $HP^1$, $HP^2$ |
| The Tf locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP locus | $AcP^A$, $AcP^B$, $AcP^C$ |
| The AK locus | $AK^1$, $AK^2$ |

| Loci | | Alleles |
|---|---|---|
| The $PGM_1$ | locus | $PGM_1^1$, $PGM_1^2$ |
| The PGD | locus | $PGD^A$, $PGD^C$ |

Set 4.

The 8 loci and the respective 19 alleles

| Loci | | Alleles |
|---|---|---|
| The ABO | locus | A, B, O |
| The Rh | locus | D, d |
| The HP | locus | $HP^1$, $HP^2$ |
| The Tf | locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP | locus | $AcP^A$, $AcP^B$, $AcP^C$ |
| The AK | locus | $AK^1$, $AK^2$ |
| The $PGM_1$ | locus | $PGM_1^1$, $PGM_1^2$ |
| The ADA | locus | $ADA^1$, $ADA^2$ |

Set 5.

The 8 loci and the respective 19 alleles

| Loci | | Alleles |
|---|---|---|
| The ABO | locus | A, B, O |
| The Rh | locus | D, d |
| The HP | locus | $HP^1$, $HP^2$ |
| The Tf | locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP | locus | $AcP^A$, $AcP^B$, $AcP^C$ |
| The AK | locus | $AK^1$, $AK^2$ |
| The $PGM_1$ | locus | $PGM_1^1$ , $PGM_1^2$ |
| The EsD | locus | $EsD^1$, $EsD^2$ |

Set 6.

The 9 loci and the respective 22 alleles

| Loci | Alleles |
|---|---|
| The $A_1A_2BO$ locus | $A_1$, $A_2$, B, O |

| Loci | | Alleles |
|---|---|---|
| The Rh | locus | D, d |
| The MN | locus | M, N |
| The Kell | locus | K, k |
| The Hp | locus | $HP^1$ , $HP^2$ |
| The Tf | locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP | locus | $AcP^A$, $AcP^B$, $AcP^C$ |
| The AK | locus | $AK^1$ , $AK^2$ |
| The $PGM_1$ | locus | $PGM_1^1$ , $PGM_1^2$ |

Set 7.

The 9 loci and the respective 24 alleles

| Loci | | Alleles |
|---|---|---|
| The $A_1A_2BO$ | locus | $A_1$, $A_2$, B, O |
| The Rh | locus | D, d |
| The MNSs | locus | M, N, S, s |
| The Kell | locus | K, k |
| The HP | locus | $HP^1$, $HP^2$ |
| The Tf | locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP | locus | $AcP^A$, $AcP^B$, $AcP^C$ |
| The AK | locus | $AK^1$, $AK^2$ |
| The $PGM_1$ | locus | $PGM_1^1$ , $PGM_1^2$ |

Set 8.

The 10 loci and the respective 26 alleles

| Loci | | Alleles |
|---|---|---|
| The $A_1A_2$ BO | locus | $A_1$, $A_2$, B, O |
| The Rh | locus | D, d |
| The MNSs | locus | M, N, S, s |
| The Kell | locus | K, k |
| The Duffy | locus | $FY^a$, $Fy^b$+ FY |

| Loci | | Alleles |
|---|---|---|
| The HP | locus | $HP^1$, $HP^2$ |
| The Tf | locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP | locus | $AcP^A$ , $AcP^B$, $AcP^C$ |
| The AK | locus | $AK^1$, $AK^2$ |
| The $PGM_1$ | locus | $PGM_1^1$ , $PGM_1^2$ |

Set 9.

The 10 loci and the respective 26 alleles

| Loci | | Alleles |
|---|---|---|
| The $A_1A_2$ BO | locus | $A_1$, $A_2$, B, O |
| The Rh | locus | D, d |
| The MNSs | locus | M, N, Ŝ, s |
| The Kell | locus | K, k |
| The Kidd | locus | $JK^a$, $JK^b$+Jk |
| The HP | locus | $HP^1$, $HP^2$ |
| The Tf | locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP | locus | $AcP^A$ , $AcP^B$, $AcP^C$ |
| The AK | locus | $AK^1$, $AK^2$ |
| The $PGM_1$ | locus | $PGM_1^1$ , $PGM_1^2$ |

Set 10.

The 9 loci and the respective 26 alleles

| Loci | | Alleles |
|---|---|---|
| The $A_1A_2$ BO | locus | $A_1$, $A_2$, B, O |
| The Rh | locus | C, c, D, d, E, e |
| The MN | locus | M, N |
| The Kell | locus | K, k |
| The HP | locus | $HP^1$, $HP^2$ |
| The Tf | locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP | locus | $AcP^A$, $AcP^B$, $AcP^C$ |

| Loci | Alleles |
|---|---|
| The AK locus | $AK^1$, $AK^2$ |
| The $PGM_1$ locus | $PGM_1^1$, $PGM_1^2$ |

Set 11.

The 12 loci and the respective 30 alleles

| Loci | Alleles |
|---|---|
| The $A_1A_2BO$ locus | $A_1$, $A_2$, B, O |
| The Rh locus | D, d |
| The MNSs locus | M, N, S, s |
| The Kell locus | K, k |
| The Duffy locus | $Fy^a$, $Fy^b$, + Fy |
| The Kidd locus | $JK^a$, $JK^b$, + JK |
| The HP locus | $HP^1$, $HP^2$ |
| The Tf locus | $Tf^C$, $Tf^B$, $Tf^D$ |
| The AcP locus | $AcP^A$, $AcP^B$, $AcP^C$ |
| The AK locus | $AK^1$, $AK^2$ |
| The $PGM_1$ locus | $PGM_1^1$, $PGM_1^2$ |
| The EsD locus | $EsD^1$, $EsD^2$ |

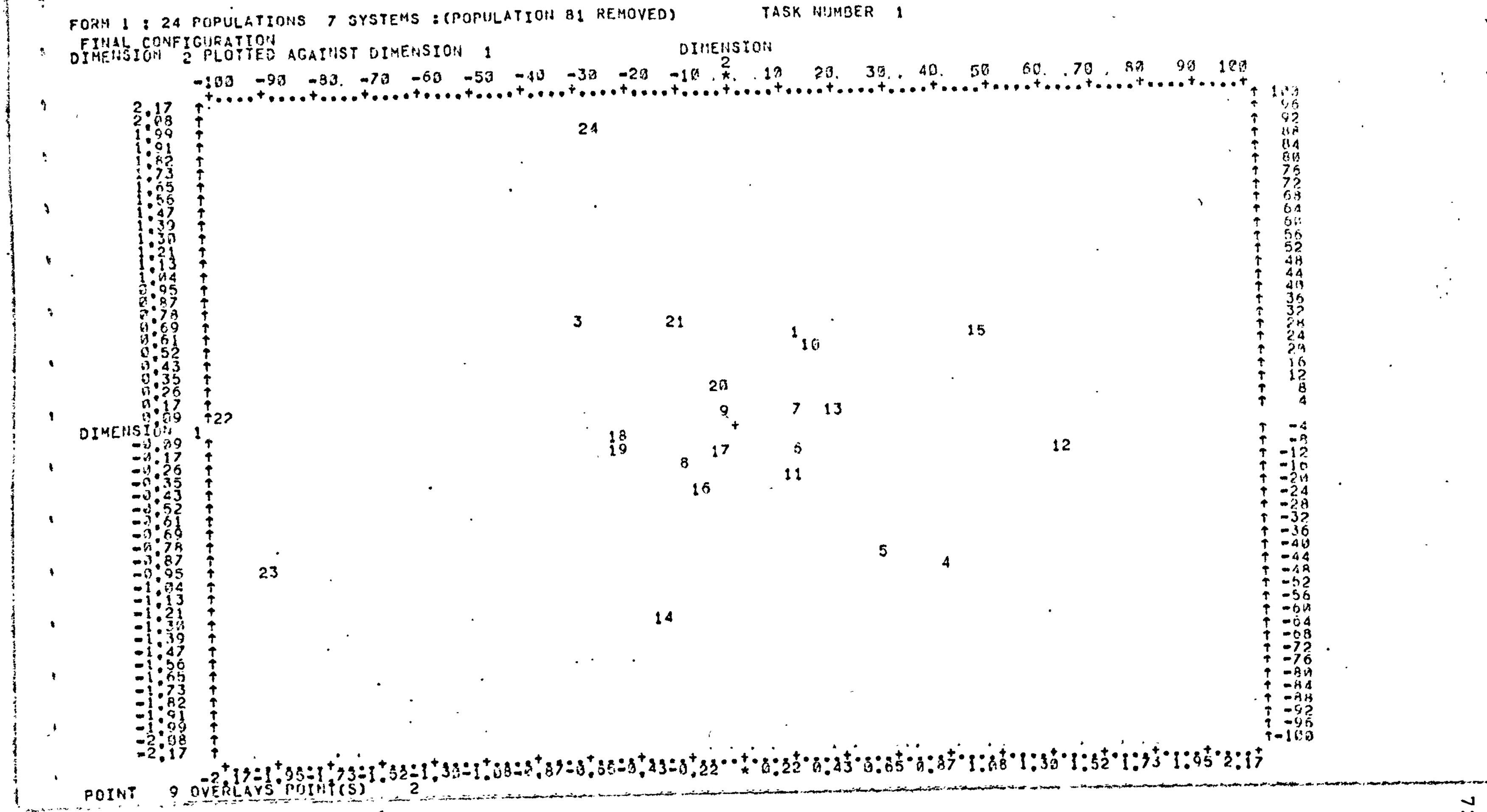



Fig. 5.4.3. 1a. Two- dimensional map of genetic distance for 24 Iranian and neighbouring populations.

Based on gene frequency data for 7 polymorphic loci.

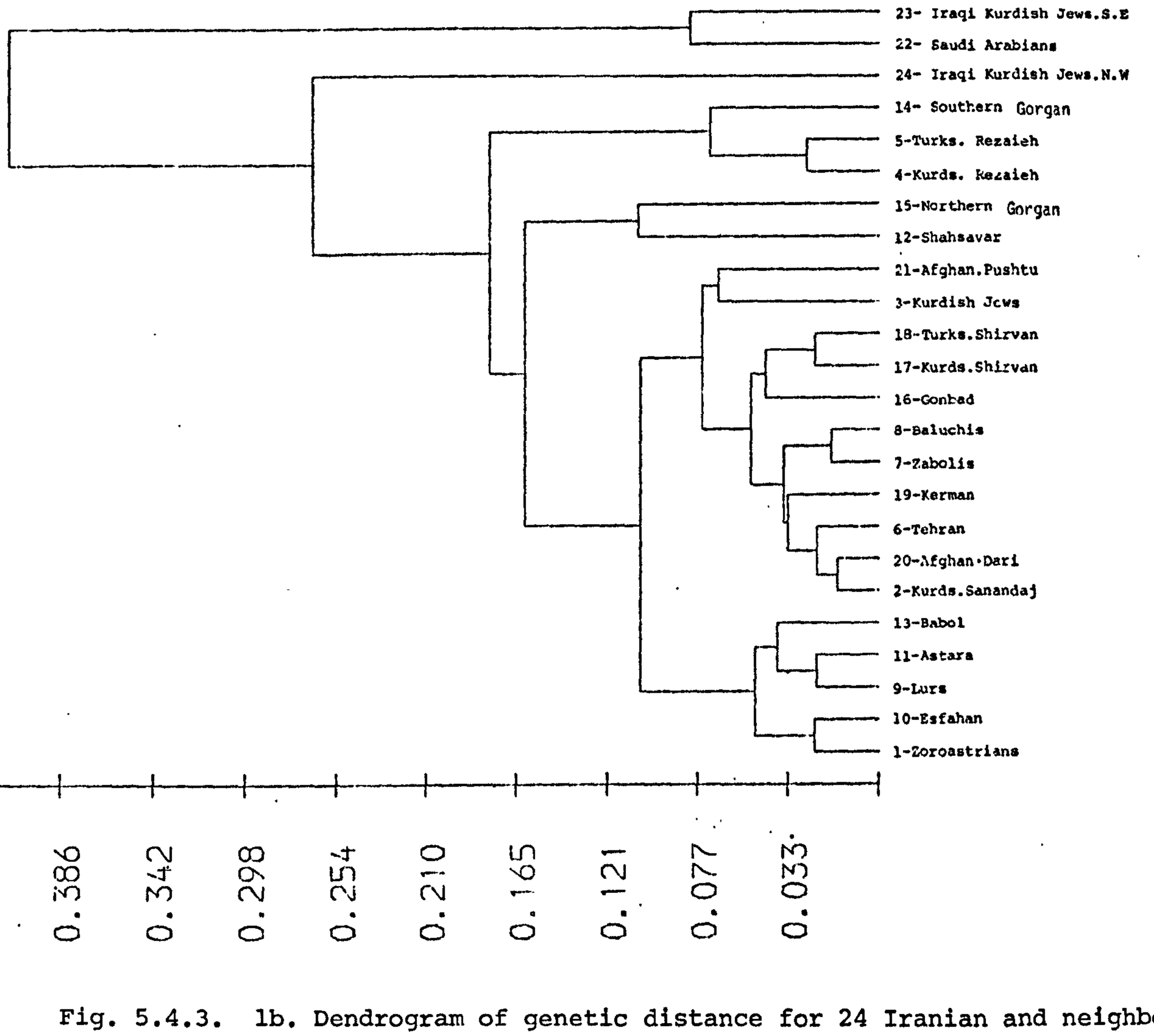



Fig. 5.4.3. 1b. Dendrogram of genetic distance for 24 Iranian and neighbouring populations. Based on gene frequency data for 7 polymorphic loci.

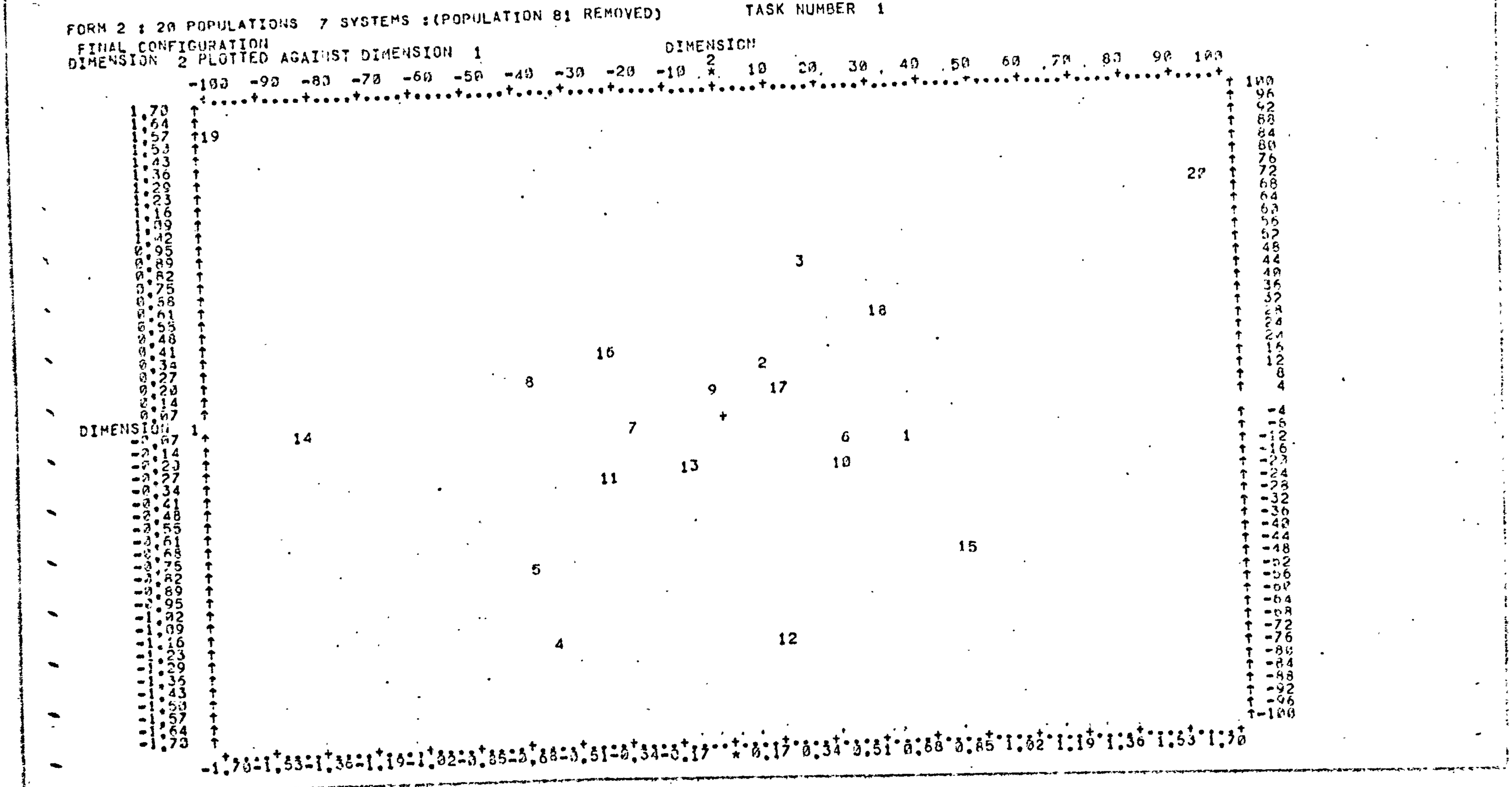



Fig. 5.4.3. 2a. Two- dimensional map of genetic distance for 20 Iranian and neighbouring populations.

Based on gene frequency data for 7 polymorphic loci.

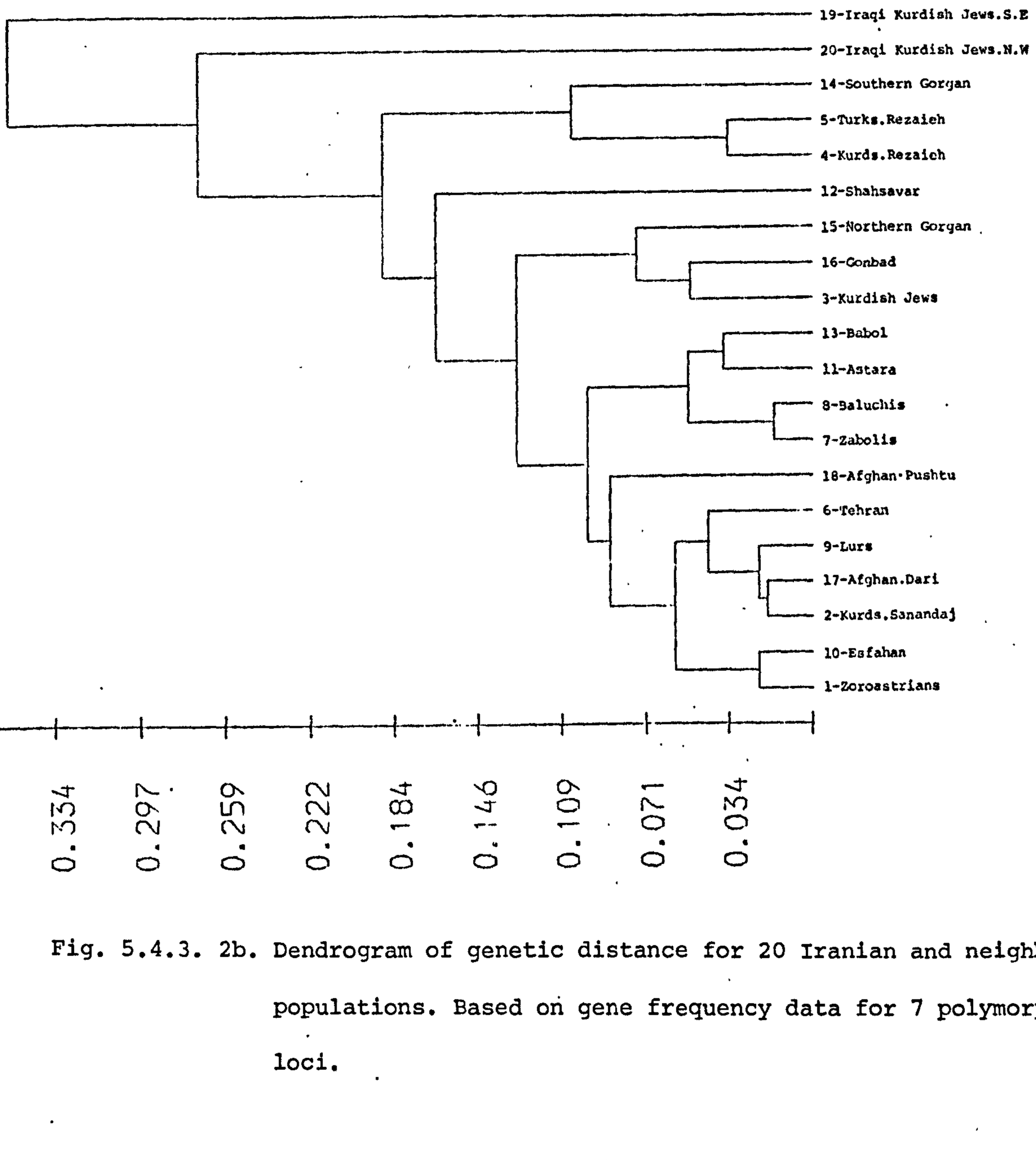



Fig. 5.4.3. 2b. Dendrogram of genetic distance for 20 Iranian and neighbouring populations. Based on gene frequency data for 7 polymorphic loci.

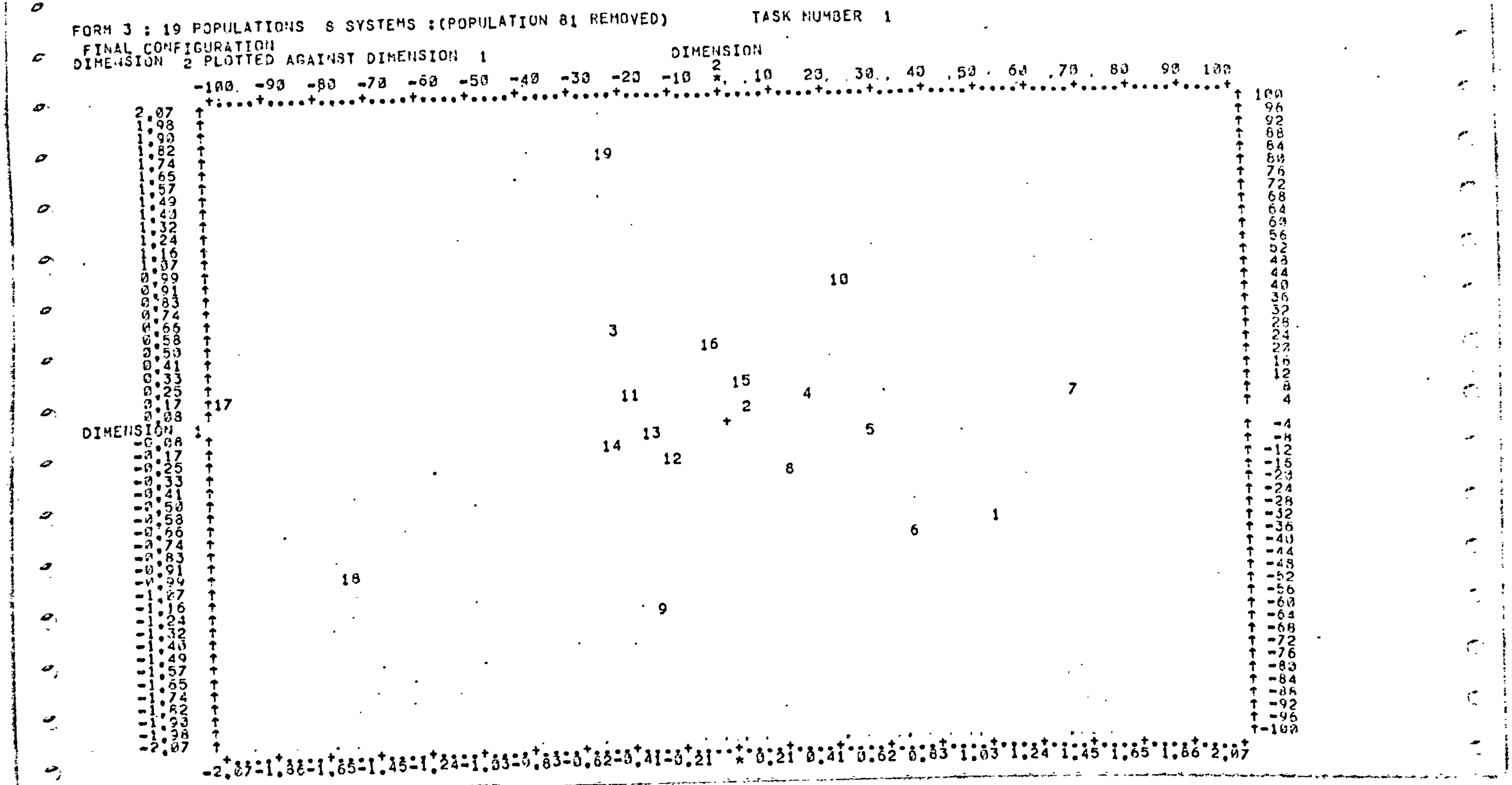



Fig. 5.4.3. 3a. Two- dimensional map of genetic distance for 19 Iranian and neighbouring populations.

Based on gene frequency data for 8 polymorphic loci.

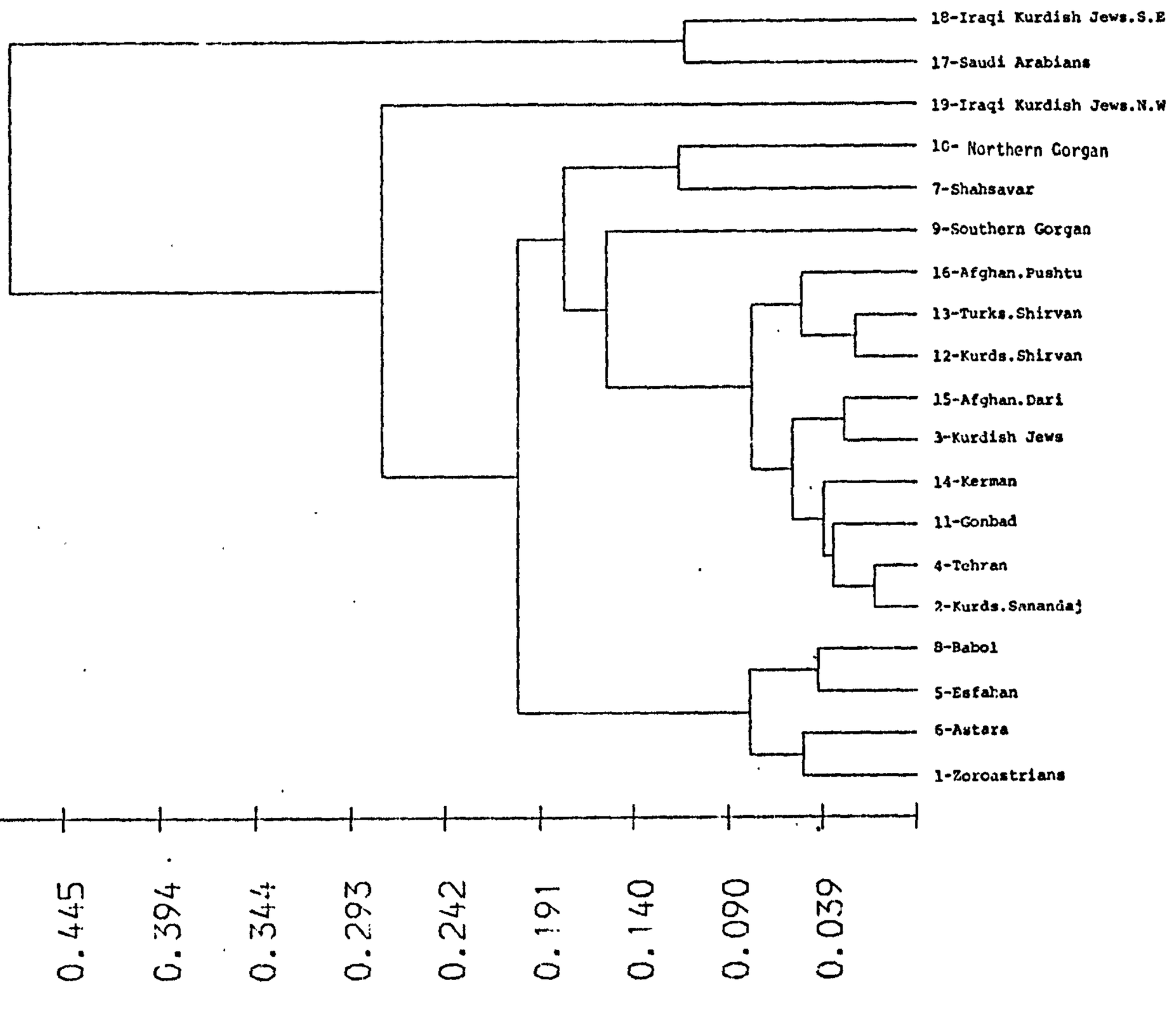



Fig. 5.4.3. 3b. Dendrogram of genetic distance for 19 Iranian and neighbouring populations. Based on gene frequency data for 8 polymorphic loci.

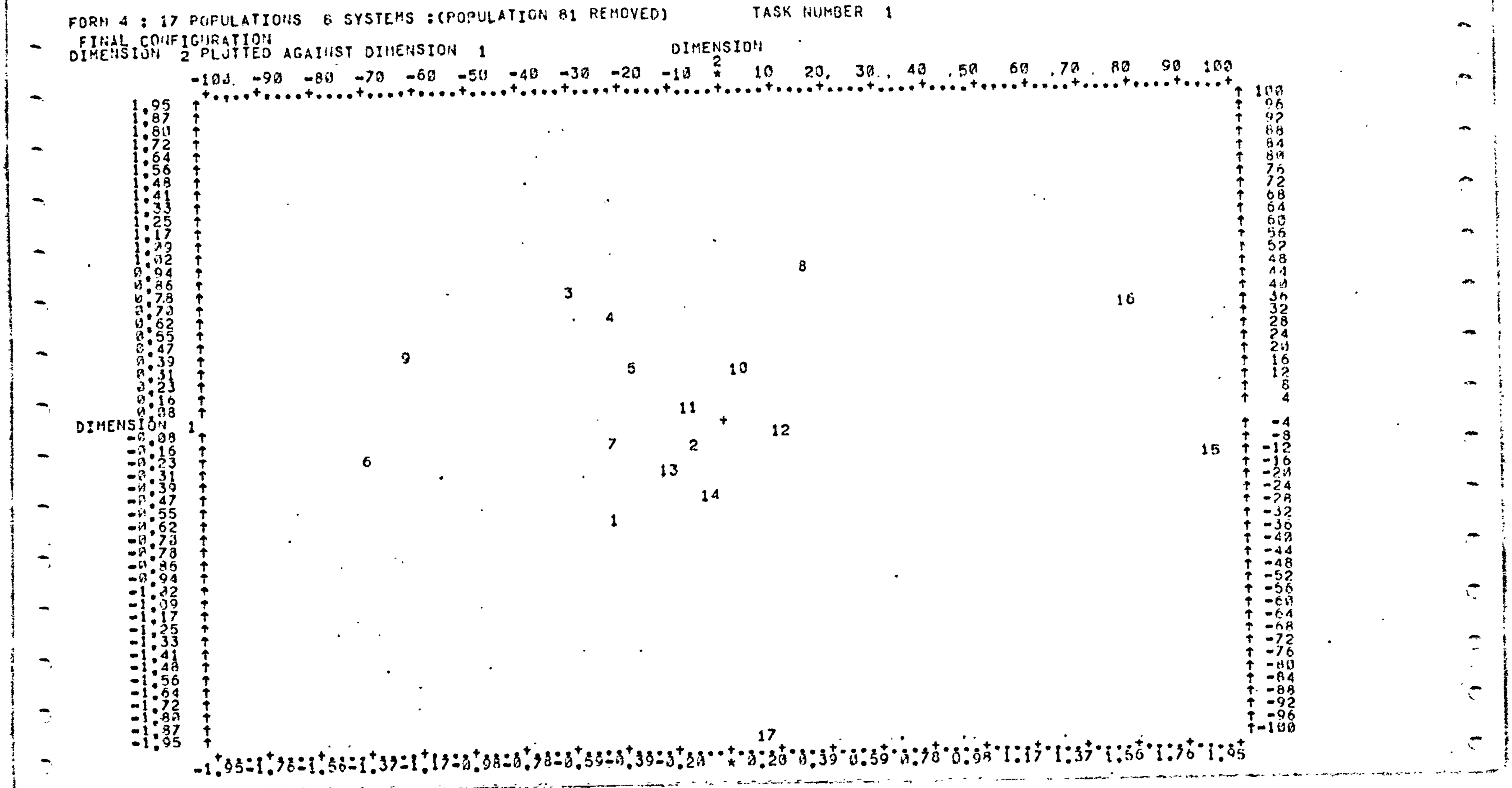



Fig. 5.4.3. 4a. Two-dimensional map of genetic distance for 17 Iranian and neighbouring populations.

Based on gene frequency data for 8 polymorphic loci.

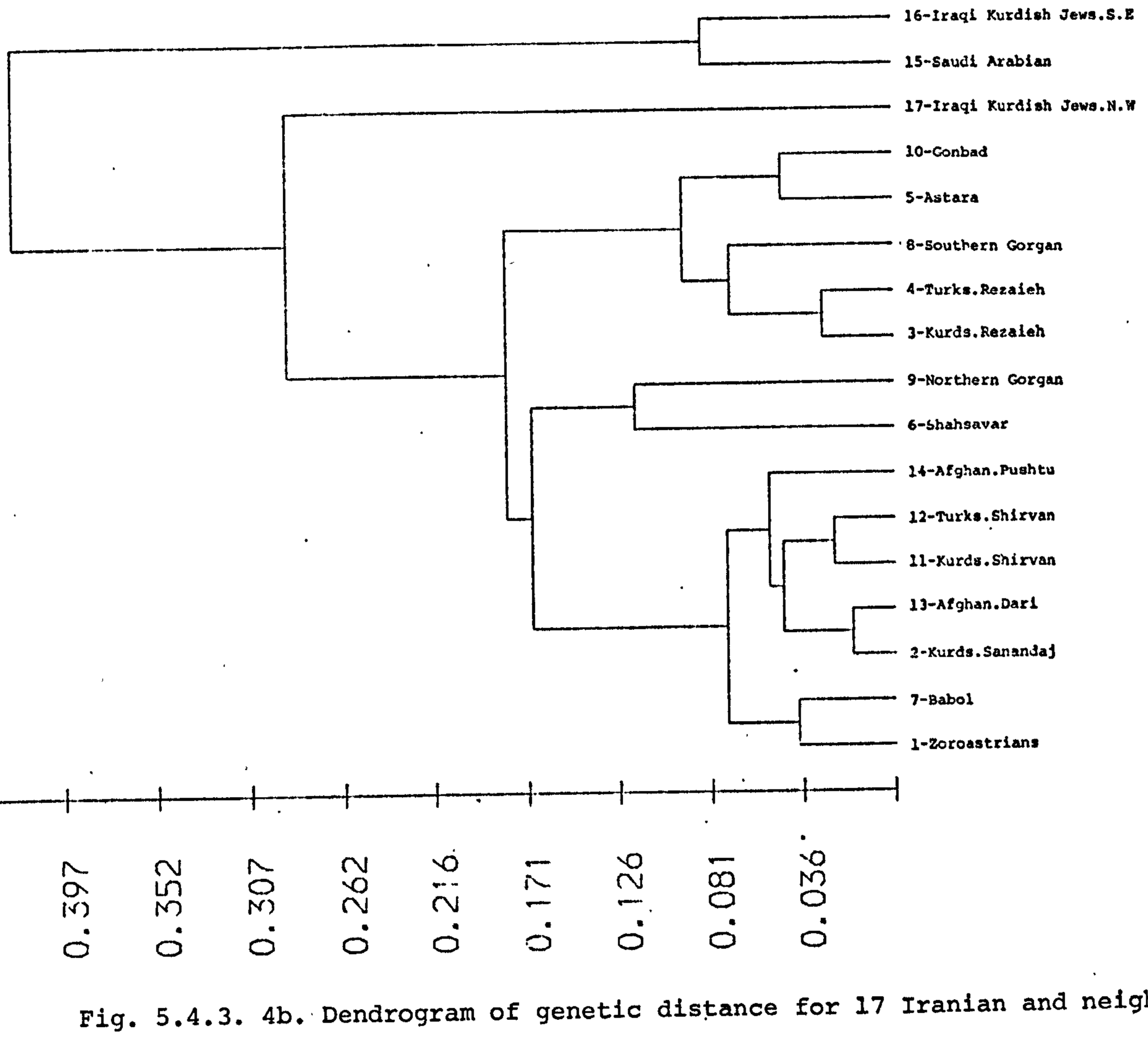



Fig. 5.4.3. 4b. Dendrogram of genetic distance for 17 Iranian and neighbouring populations. Based on gene frequency data for 8 polymorphic loci.

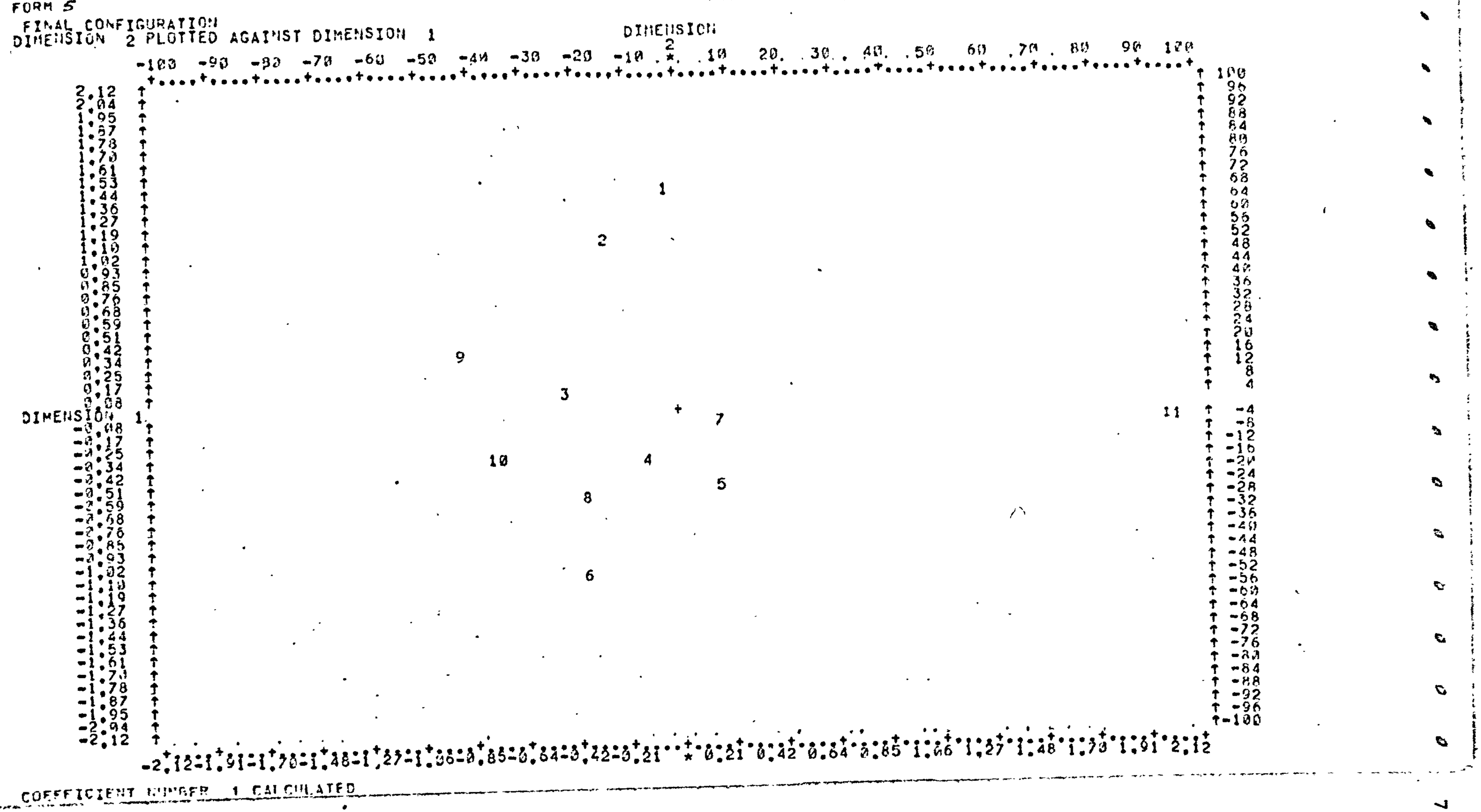



Fig. 5.4.3. 5a. Two-dimensional map of genetic distance for 11 Iranian and neighbouring populations.

Based on gene frequency data for 8 polymorphic loci.

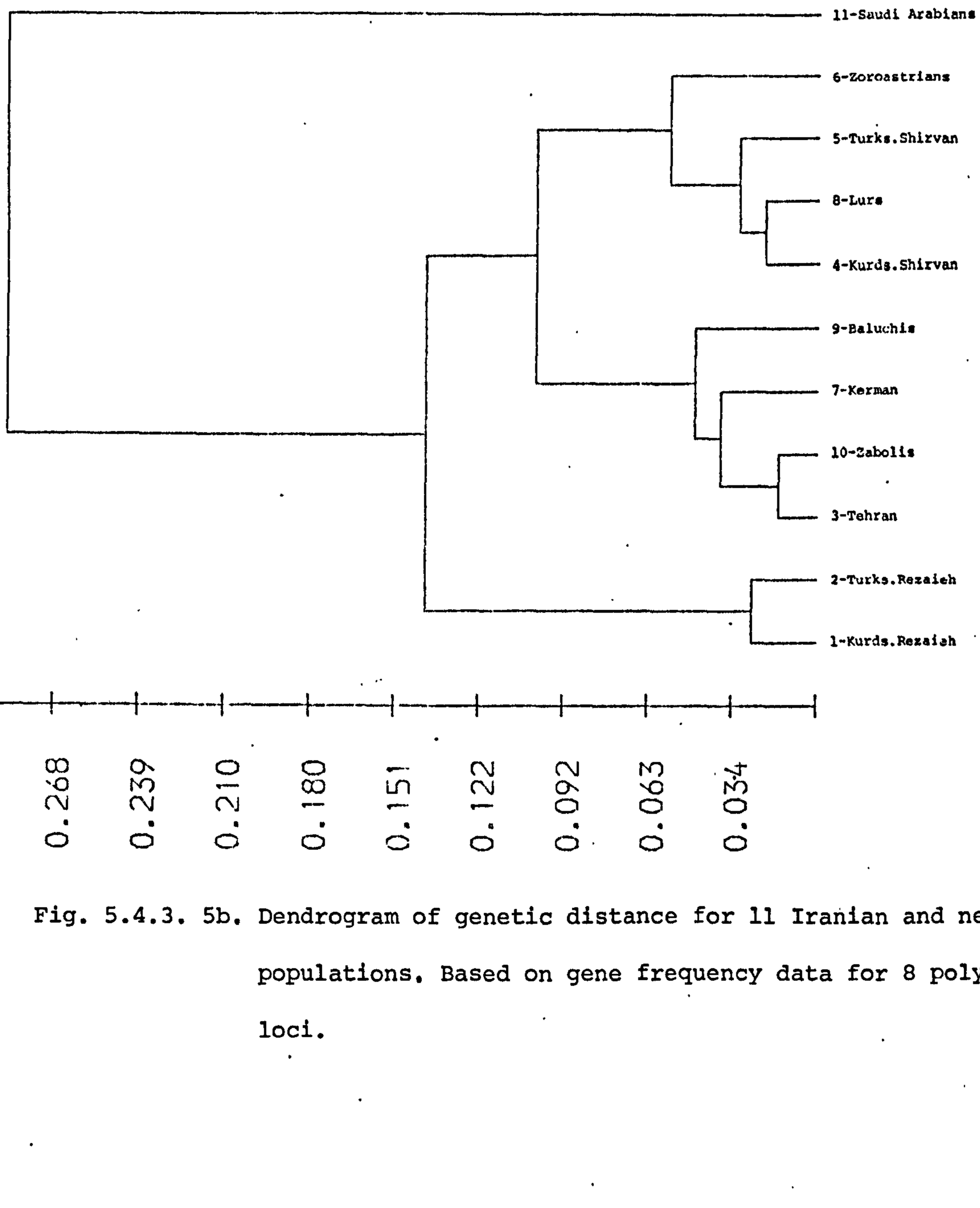



Fig. 5.4.3. 5b. Dendrogram of genetic distance for 11 Iranian and neighbouring populations. Based on gene frequency data for 8 polymorphic loci.

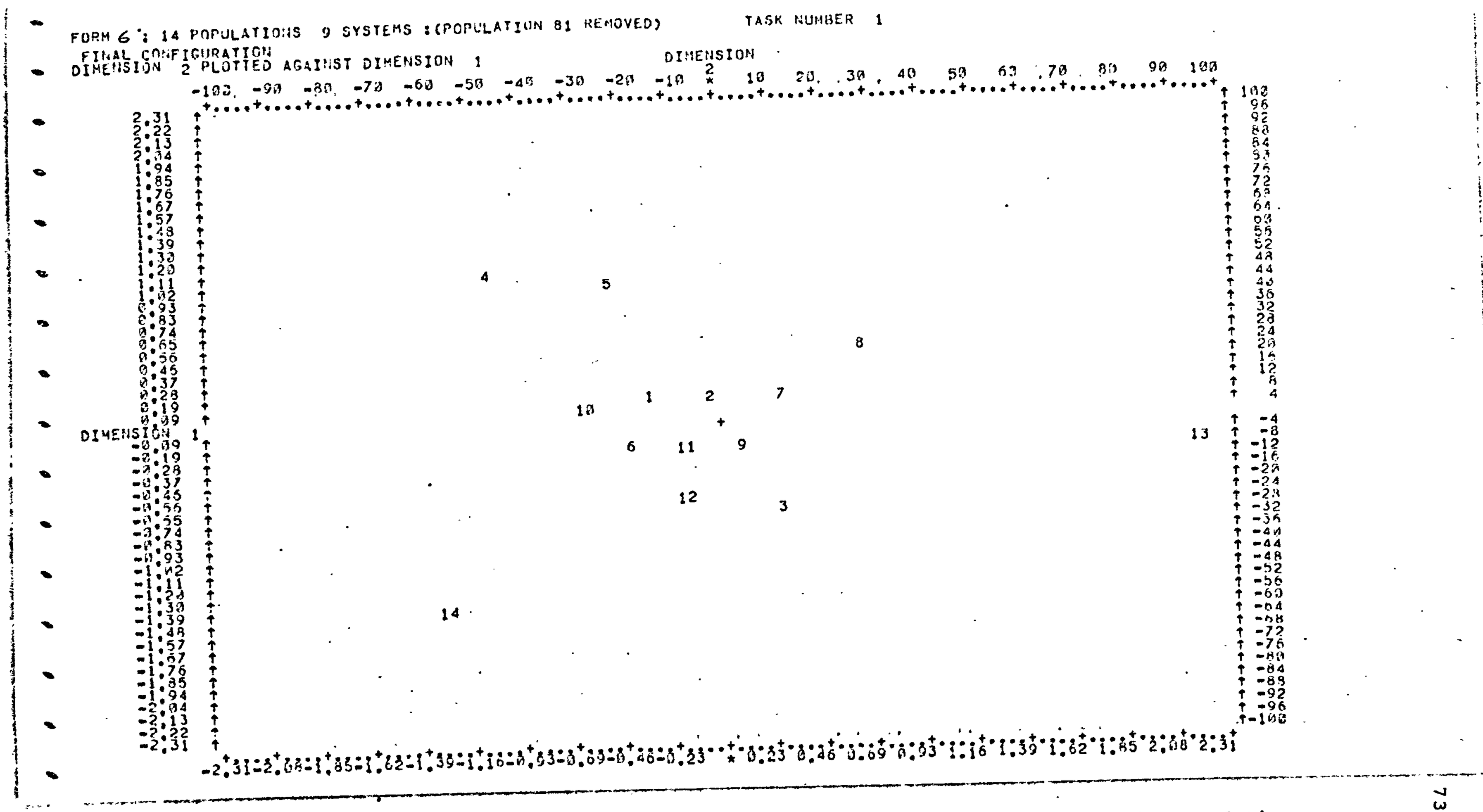



Fig. 5.4.3. 6a. Two- dimensional map of genetic distance for 14 Iranian and neighbouring populations.

Based on gene frequency data for 9 polymorphic loci.

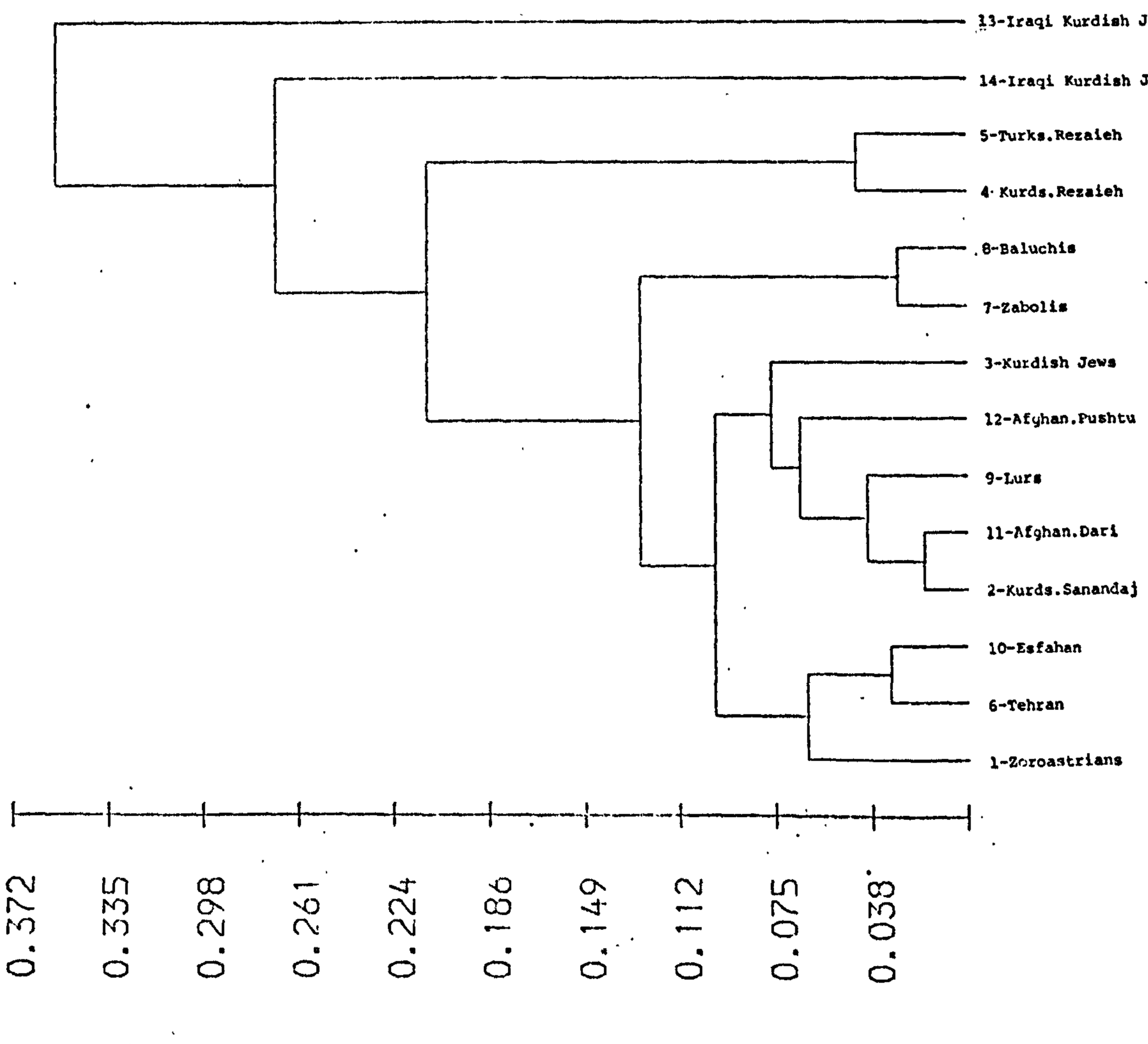



Fig. 5.4.3. 6b. Dendrogram of genetic distance for 14 Iranian and neighbouring populations. Based on gene frequency data for 9 polymorphic loci.

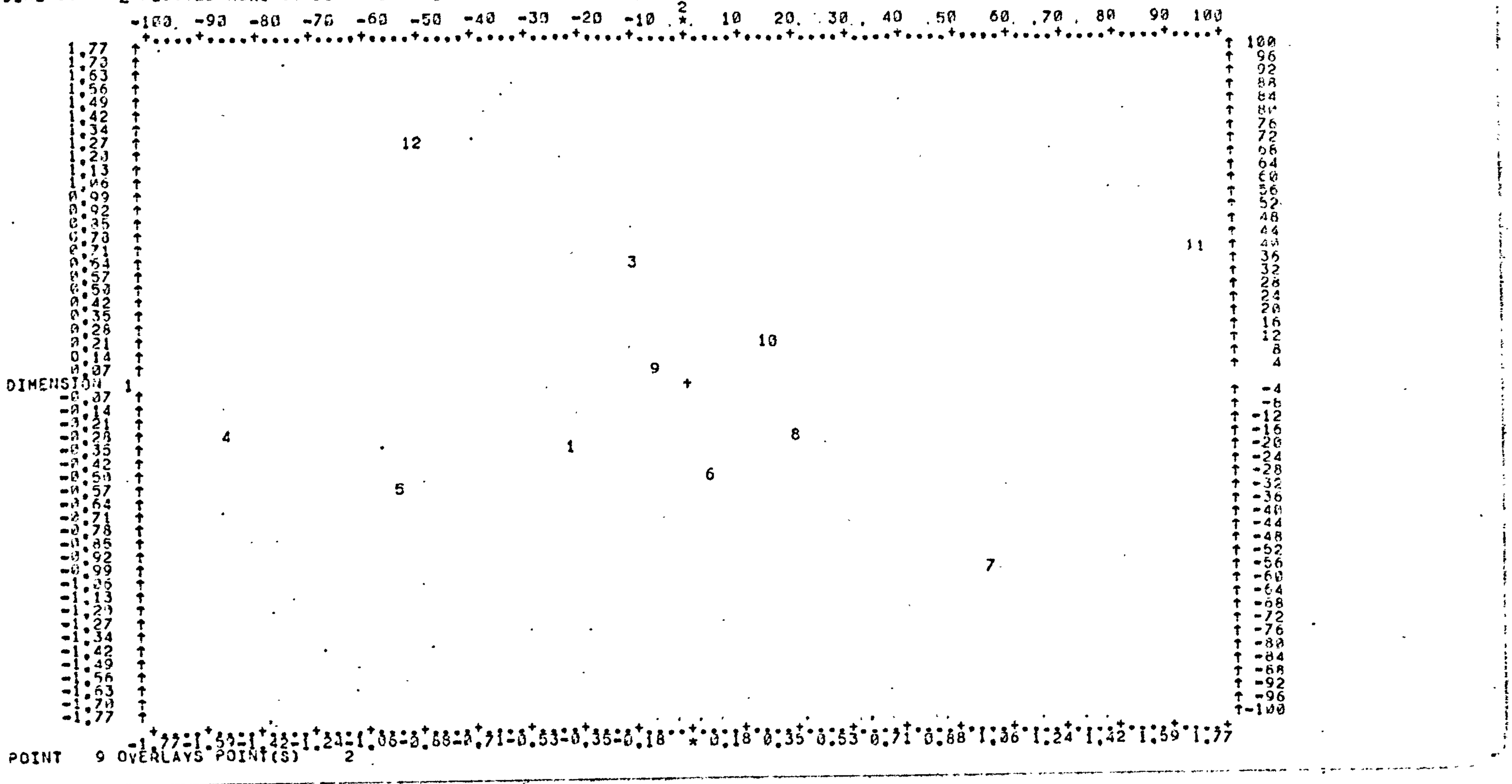



Fig. 5.4.3. 7a. Two- dimensional map of genetic distance for 12 Iranian and neighbouring populations.

Based on gene frequency data for 9 polymorphic loci.

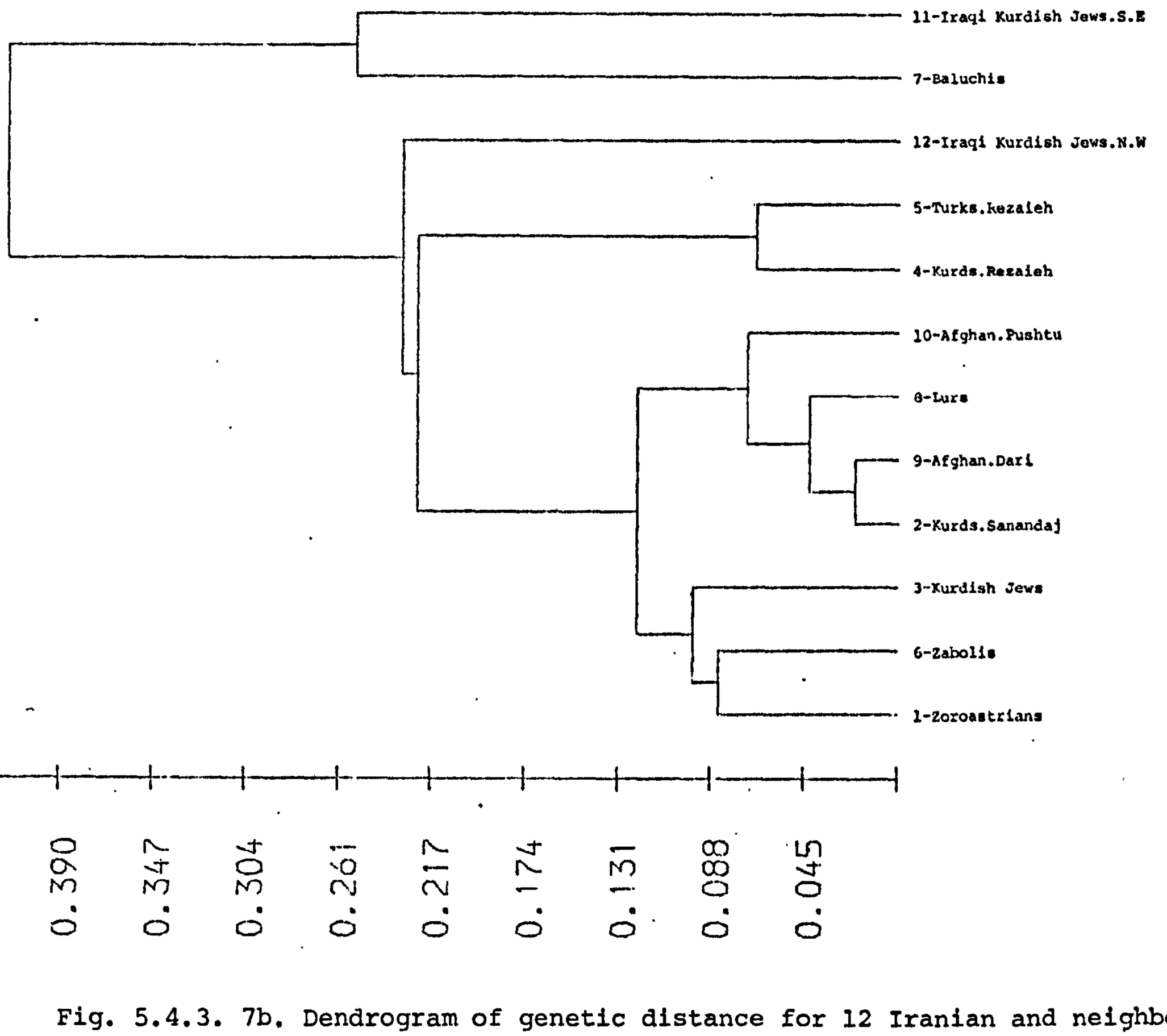



Fig. 5.4.3. 7b. Dendrogram of genetic distance for 12 Iranian and neighbouring populations. Based on gene frequency data for 9 polymorphic loci.

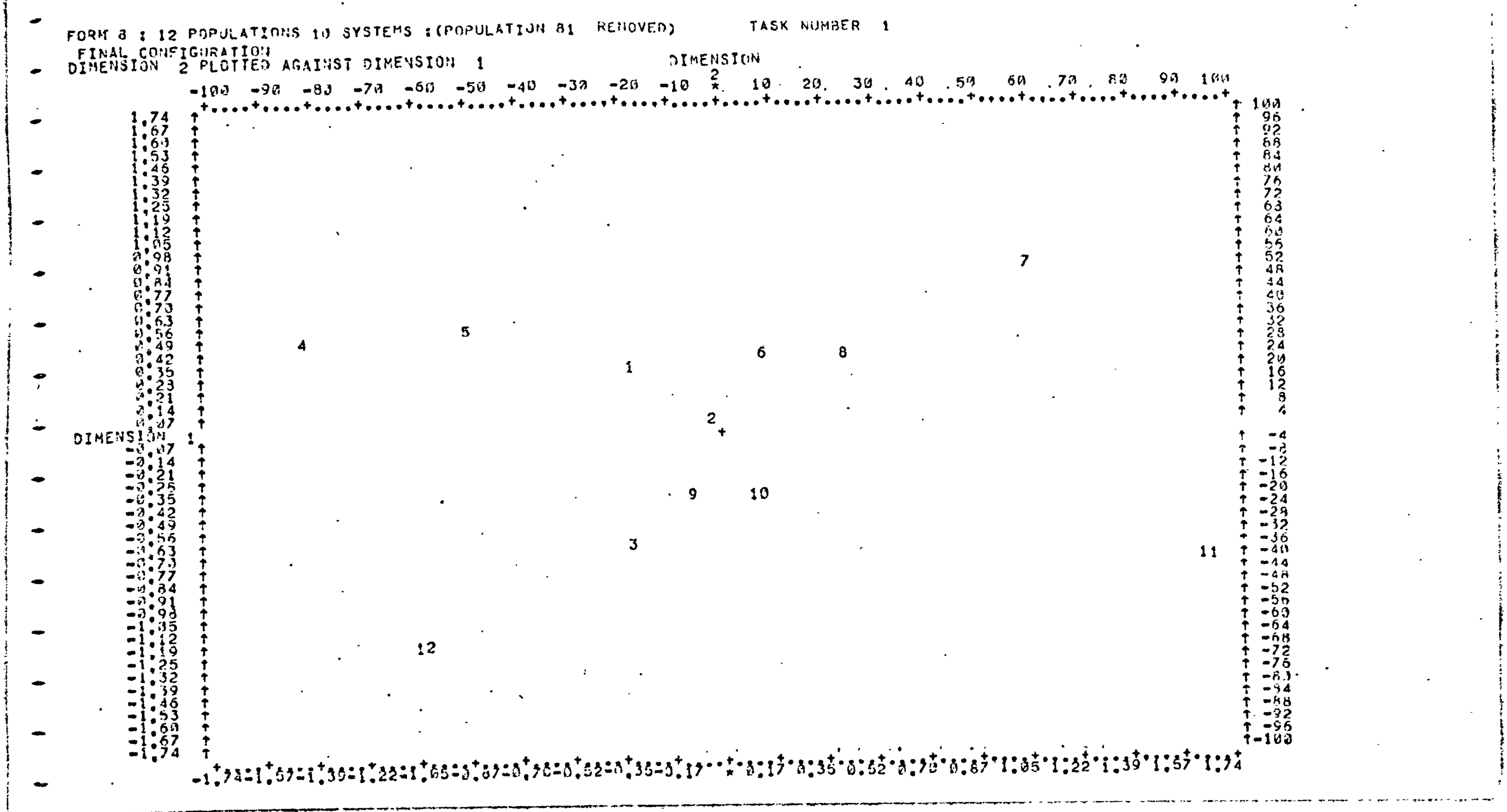



Fig. 5.4.3. 8a. Two-dimensional map of genetic distance for 12 Iranian and neighbouring populations.

Based on gene frequency data for 10 polymorphic loci.

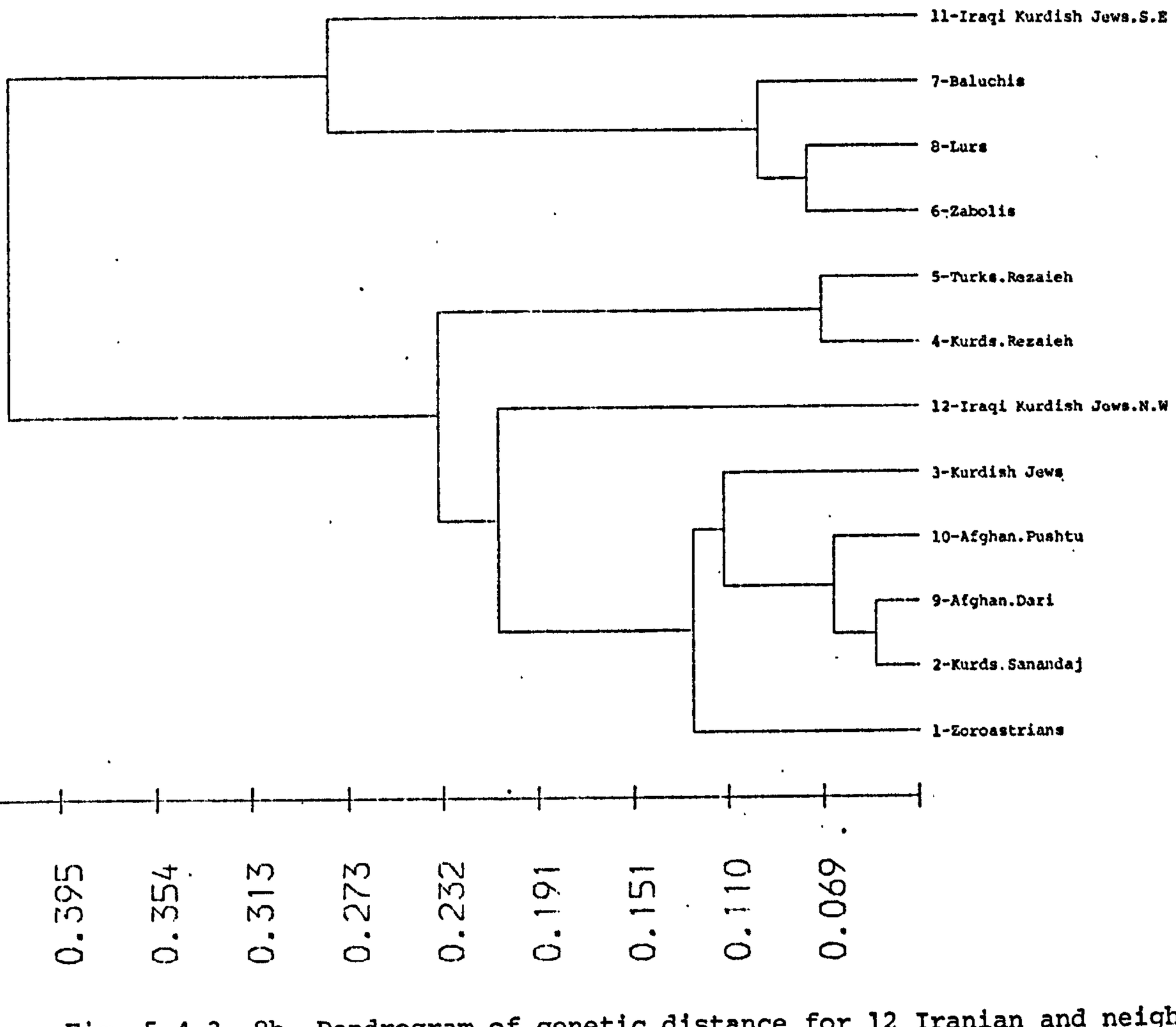



Fig. 5.4.3. 8b. Dendrogram of genetic distance for 12 Iranian and neighbouring populations. Based on gene frequency data for 10 polymorphic loci.

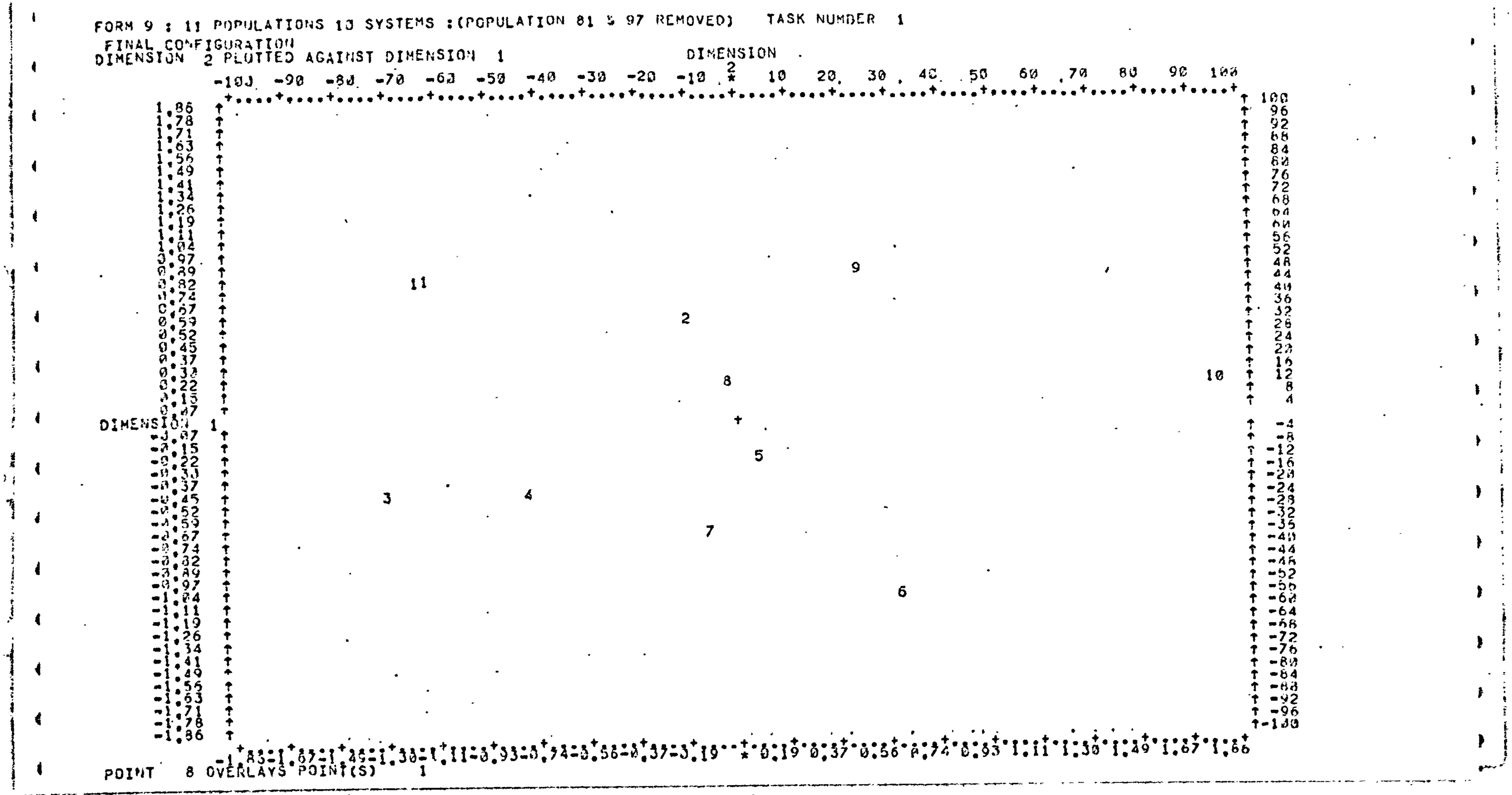



Fig. 5.4.3. 9a. Two- dimensional map of genetic distance for 11 Iranian and neighbouring populations.

Based on gene frequency data for 10 polymorphic loci.

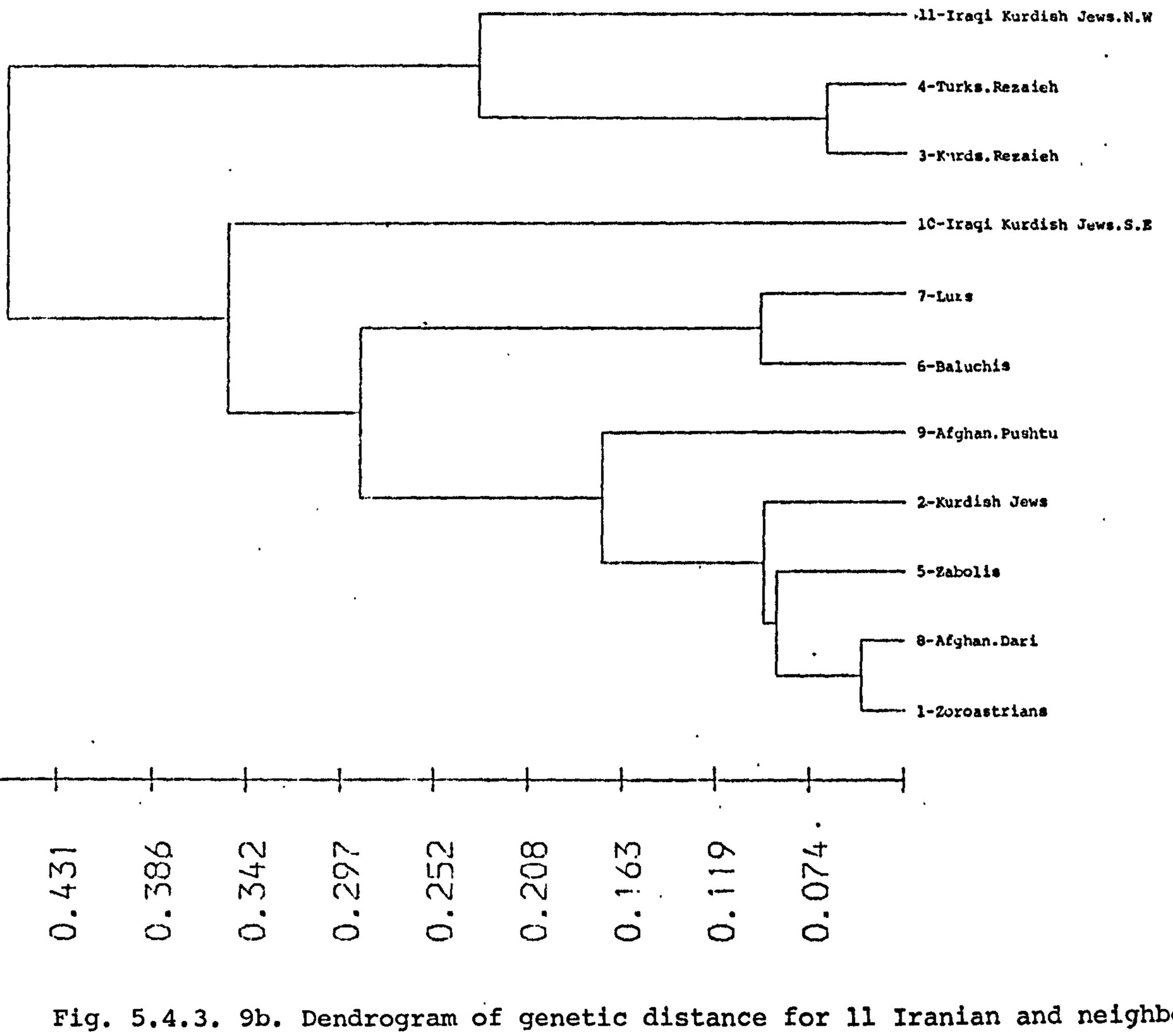



Fig. 5.4.3. 9b. Dendrogram of genetic distance for 11 Iranian and neighbouring populations. Based on gene frequency data for 10 polymorphic loci.

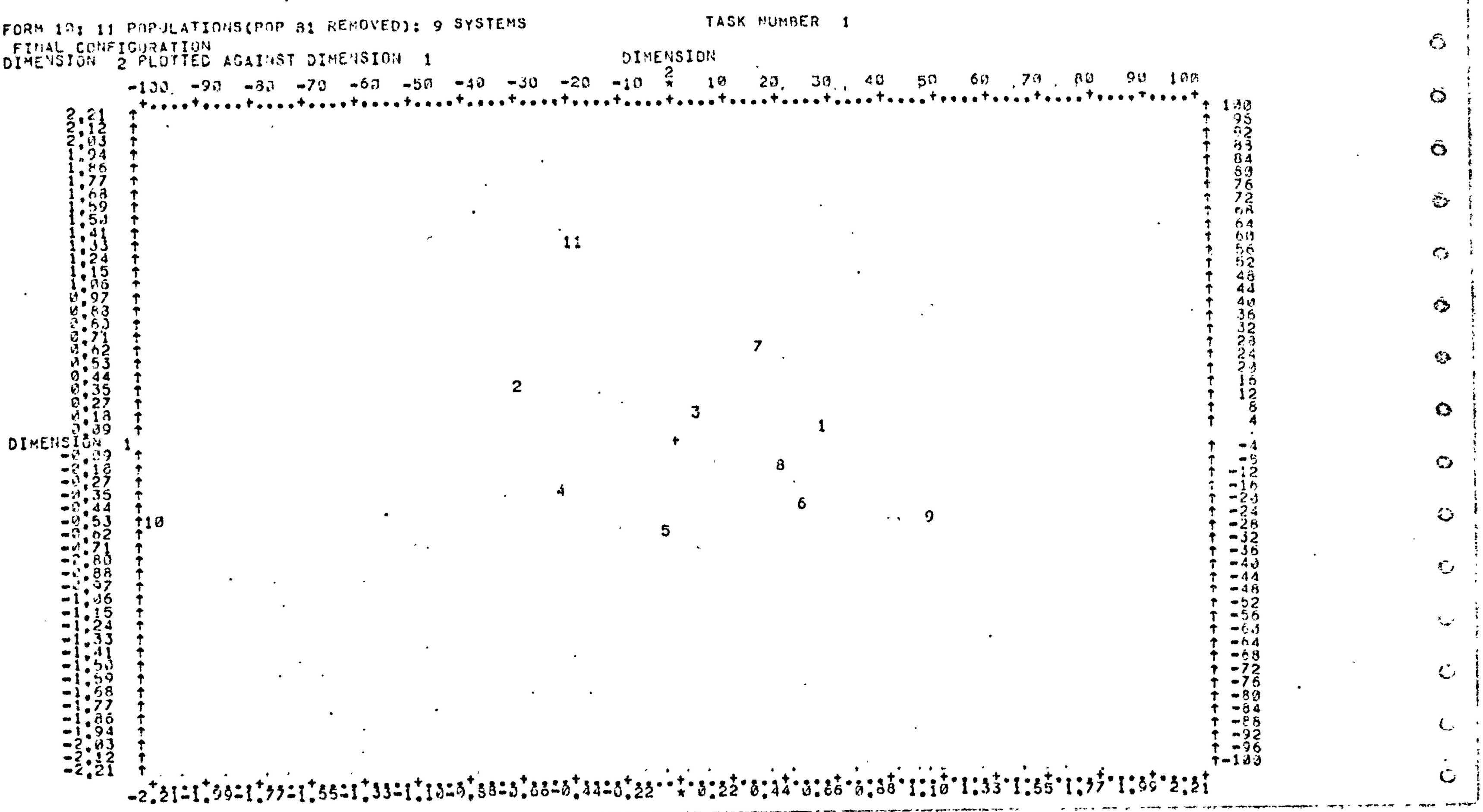



Fig. 5.4.3. 10a. Two- dimensional map of genetic distance for 11 Iranian and neighbouring populations.

Based on gene frequency data for 9 polymorphic loci.

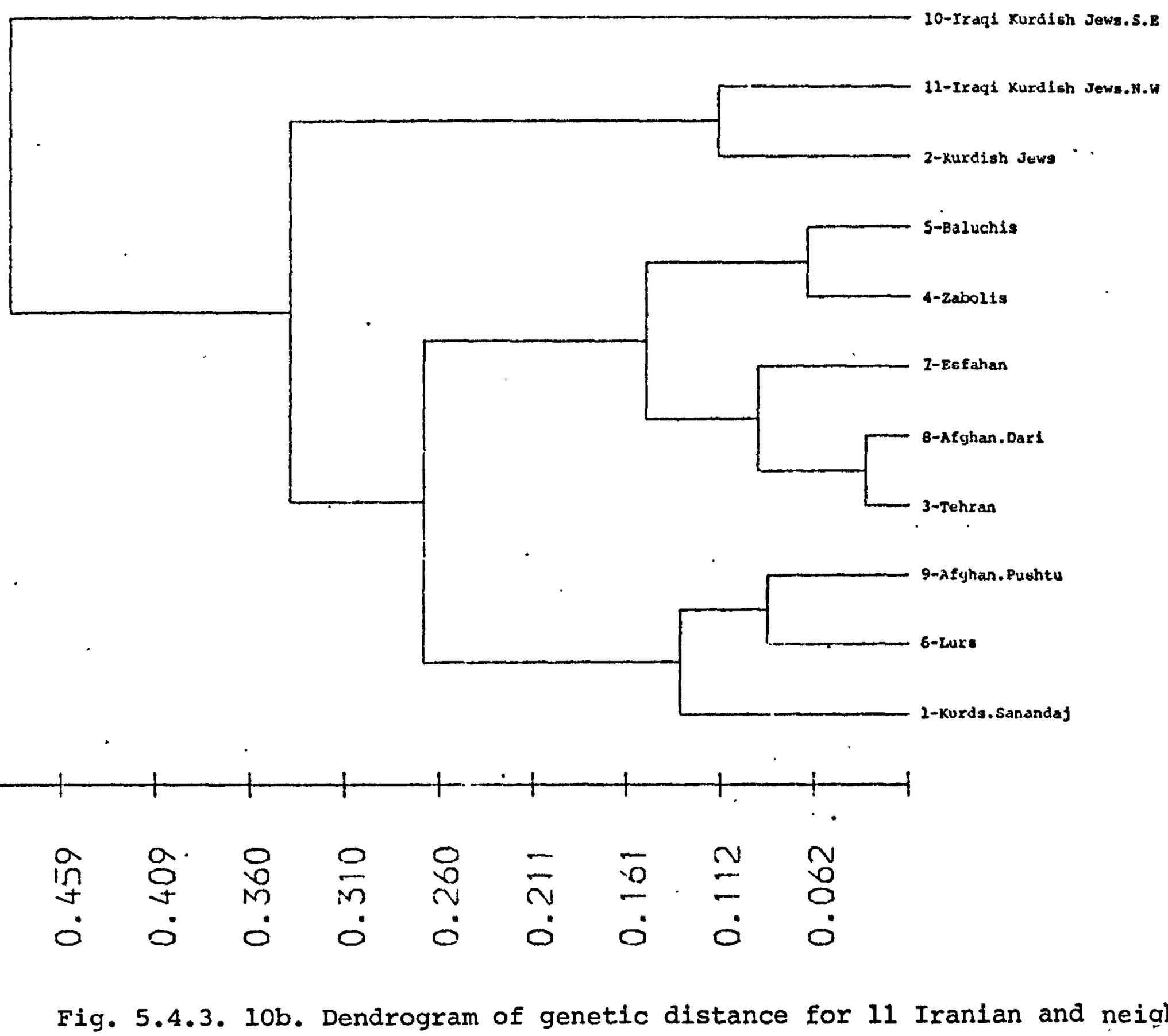



Fig. 5.4.3. 10b. Dendrogram of genetic distance for 11 Iranian and neighbouring populations. Based on gene frequency data for 9 polymorphic loci.

COEFFICIENT NUMBER 1 CALCULATED

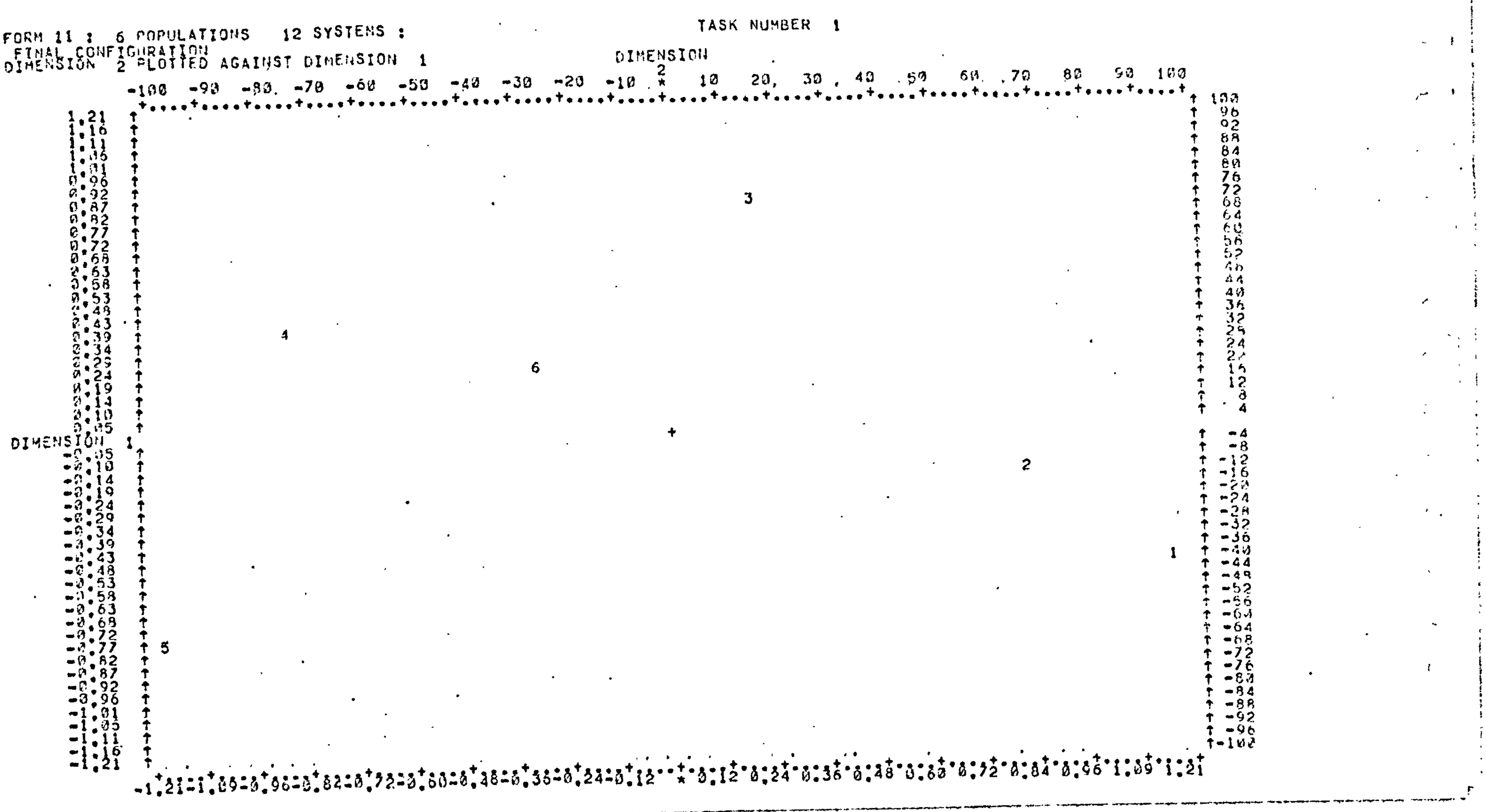



Fig. 5.4.3. 11a. Two- dimensional map of genetic distance for 6 Iranian populations, Based on gene frequency data for 12 polymorphic loci.

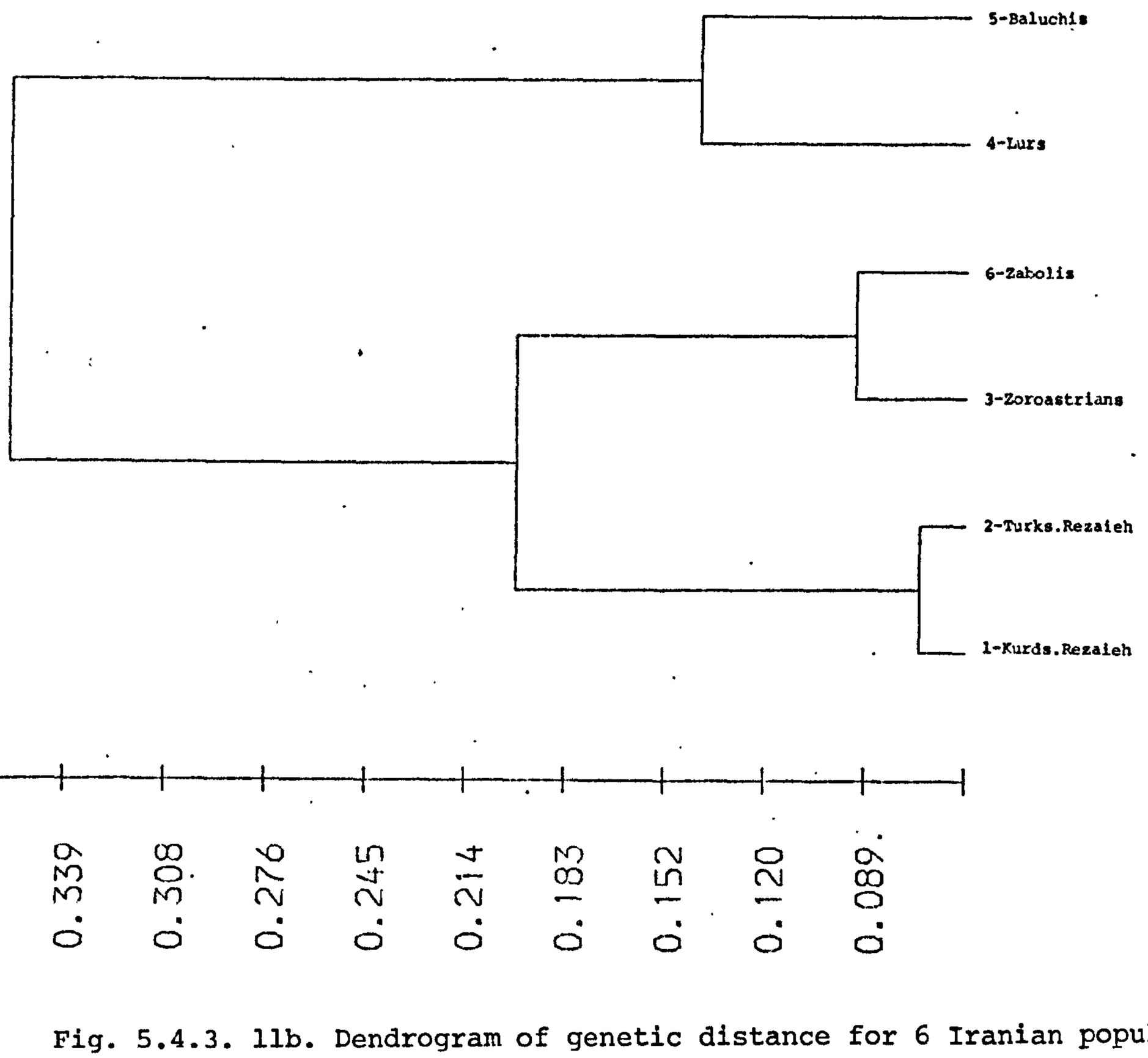



Fig. 5.4.3. 11b. Dendrogram of genetic distance for 6 Iranian populations.

Based on gene frequency data for 12 polymorphic loci.

## 5.4.4. Discussion

The various population groups from Iran and neighbouring countries have been compared with each other. The neighbouring groups consisted of the Kurdish Jews of the N.W and S.E Iraq, the Daris and Pushtus of Afghanistan and the Saudi Arabian Arabs.

No data, on the serological systems considered in the present analysis, was available for the other neighbouring populations.

Figures 1-11 a and b present the Non-Metric two dimensional scaling and the cluster analysis outputs, respectively, for the eleven sets of analysis produced using Edwards $E^2$ statistics.

As mentioned before, in the present study two types of analysis were used for the eleven possible combinations of the investigated populations and serological systems.It should be pointed out that on the one hand, the Non-Metric two dimensional scaling analysis and the cluster analysis may give slightly different relationships, so they must be used together and the outlying populations show relationships which agree in both types of analysis. On the other hand, the actual relationships generated by the eleven sets of analysis differ depending on the loci considered, thus, strict relationships should not be emphasised too much unless they appear in more than one set of analysis. Therefore, there will be no clearcut answer from any single analysis, and they have to be taken together.

In this sense, regarding all the eleven sets of analysis, those relationships which are confirmed in more than

one set of analysis will be discussed and the remaining doubtful ones will not be considered.

The results of the present analysis show that the Kurds and Turks of Rezaieh( north western Iran) are closely related to each other and both populations are distantly related to the Turks of Astara living in the same region. These findings seem to indicate that the populations of north western Iran are genetically homogeneous. All these three populations are related at a higher level to the Iranian Kurdish Jews and the Iraqi Kurdish Jews of the north west.

The Kurdish Jews of western Iran and the Iraqi Kurdish Jews of the N.W. are closer to each other than they are to the non-Jewish Kurds of western Iran, they show a moderate genetic resemblance to the indigenous Kurds, while those from south eastern Iraq differ considerably.

As mentioned above, while there is not a close resemblance between the Kurdish Jews and the Kurds, there is a recognizable similarity. This is more particularly so for the Kurdish Jews from western Iran and north western Iraq than for those of south eastern Iraq. The latter indeed differ so widely from any other populations in or near Iraq that is must be assumed that they are an extremely high inbred isolate(Tills et al, 1977).

The Arabs of Saudi Arabia seem to be related only to the Iraqi Kurdish Jews of the south east and unrelated at all to the remaining large number of populations included in the eleven sets of analysis. Generaly, the people of Saudi Arabia have some typical genetic characteristics which suggest a considerable amount of African admixture.

The Zoroastrians are closely related to the Esfahanis, living in nearly the same geographical location, and both populations seem to be distantly related to the Tehranis and Lurs.

The Zabolis and the Baluchis of Sistan and Baluchistan are closely related to each other and both populations seem to be distantly related to the Kermanis and at a higher level to the Zoroastrians and the Lurs.

Again, as in Rezaieh (north western Iran), the Kurds and Turks of Shirvan, Khorasan( north eastern Iran) are closely related to each other and both populations are distantly related to the Gonbadis living in the same location. It should be noted here that the Kurds and Turks of Rezaieh (north west) show no resemblance to the originally same two population groups of Shirvan, Khorasan (north east), the latter two groups being more closely related to the Gonbadis living in the same geographical location. These findings clearly indicate the strong influence of environmental factors on the genetic structure of populations.

The Afghan Daris and Pushtus, in general, both fit into Iranian clusters and are most closely related to the populations of Khorasan (north eastern Iran). The two Afghan groups are considered to be ethnically allied with Indo-Europeans, they are descendents of an Iranian-Aryan tribe.

Finally, the populations of the Caspian Littoral are not homogeneous, either in the geographical Zones they occupy or in their ethnic affiliations. The results of the present analysis seem to indicate they are also genetically heterogeneous.

## 5.5.Overall conclusion

The Iranian population is among the most heterogeneous in the world. Iran has been invaded by many peoples(i.e., Assyrians, Greeks, Arabs, Turks and Mongols), and each has left its imprint on the gene pool of the pre-existing population and together have contributed to the genetic diversity of present-day Iranian populations. Physical, religious and linguistic barriers have helped to maintain the genetic distinction of many ethnic groups of Iran.

The average gene frequencies for the whole country,of about 22 percent A; 17 percent B; 48 percent CDe($R_1$); 28 percent cde(r); 63 percent M; 44 percent $Fy^a$; 27 percent $HP^1$; 13 percent $C3^F$; 66 percent $AcP^B$; 2 percent $AcP^C$; 6 percent $AK^2$; 31 percent $PGM_1^2$; 13 percent $ADA^2$; and 19 percent $EsD^2$, all show a departure from the values found in the countries to the west and an approach to those of the Indian region.

The connections of the dominant Iranians are to be sought largely among the relatively little-studied peoples to the north of them, but we might expect connections with the Arab peoples to the west and south-west across the Persian Gulf. The Arab population of south-western Iran, however, show little resemblance to the Arabs of Arabia. The Arabic speakers of Iran have the very high B gene frequency of average about 22 percent, relating them rather to the Arabs of Iraq, but suggesting possible affinities with India. The Iranian Arabs also have average about 14 percent of cDe(Ro), showing African admixture to be slight, though rather more than in most other Iranian populations

in whom cDe(Ro) is only a little above European levels. Of the other Arab characteristics, the Iranian Arabs have rather more K genes than other Iranian populations. Their M gene frequency is average about 61 percent, near the average for Iran. This is much lower than the average for Arabia. On the whole the serological evidence suggests that over a long period there has been little migration into Iran across the Persian Gulf. If this is so it implies that the high frequencies of M in Peninsular Arabia and those in the rest of southern Asia are distinct phenomena, since they are separated on the land connection between the two regions by populations with lower M levels.

The Iranian Armenians, who are recent immigrants,have, as everywhere else, high A and cde( r ) frequencies.

The small community of Zoroastrians has attracted considerable attention since its member must be descended from the original Iranians whose religion before the Moslem invasions and conversions was Zoroastrianism. However, at that time and subsequently, many Zoroastrians have migrated to India to give rise to the Parsi community. In general, a high incidence of the gene B exists in all the Zoroastrian populations studied to date. It is known historically that the Zoroastrians of Iran have practiced endogamy since the Arab conquest in the 7th century. The high value of the gene B in the Zoroastrians, while possibly due to genetic drift, is thus more probably the result of the introduction of genes from India or due to the fact that the Zoroastrians were reservoirs of the B gene. On the whole, the pattern of the gene frequencies among Zoroastrians would not be very dissimilar from that of the Iranian Moslems, regarding a

number of serological systems. These may be attributed to the fact that the Zoroastrians of Iran once have been part of a large population in Iran which was later converted to Islam. However, the Iranian Moslems suffered from incursions of various peoples, leading to the introduction of new genetic characteristics.

The Kurds form a numerous and distinct ethnic group in the north west and west of Iran. Serologically they differ little from the Iranians proper and it has been suggested on serological grounds that the latter are the results of mixing of populations similar respectively to the present Kurds and to the Turkish speaking Ghashghais.

One reason for the present interest in the Kurds is the fact that the Kurdish Jews, who lived among them but have now mostly migrated to Israel, have the highest frequencies known of genetically determined glucose-6-phosphate dehydrogenase deficiency. There are substantial frequencies of this condition in most countries of the Near East, both in the indigenous populations (especially the Shia Arabs of Saudi Arabia) and in Jews, but in general the condition is commoner in Jews.

Quite high frequencies of G6PD deficiency are found in the indigenous populations in many parts of Iran, but since one of the main determining conditions is probably a selective advantage of carriers of the gene, in the presence of endemic falciparum malaria, the very much higher frequency in the Kurdish Jews than in Kurds poses a special problem.

The Jews of Iran show considerable genetic heterogeneity, but nearly all the communities have two features in common,

high frequencies of B and Glucose-6-phosphate dehydrogenase deficiency.

Regarding neighbouring populations, the average gene frequencies for the inhabitant of the Caucasus, of about 24 percent A; 11 percent B; 41 percent CDe($R_1$); 35 percent cde (r); 50 percent $P_1$; and 32 percent $HP^1$, all show relationships rather with the west than with the east.

The average gene frequencies for the population of Turkey, of about 29 percent A; 13 percent B; 32 percent cde(r); 56 percent M; 4 percent $AK^2$; 10 percent $ADA^2$; and 2.5 percent $PGD^C$, all suggest western rather than eastern connections.

The average gene frequencies for the inhabitant of Iraq, of about 24 percent A; 19 percent B; 48 percent CDe($R_1$); 27 percent cde(r); 60 percent M; 68 percent $AcP^B$; 3 percent $AcP^C$; 17 percent $ADA^2$; and 22 percent $EsD^2$, all show relationships rather with the east than with the west.

The Arab populations of Kuwait, Saudi Arabia and the United Arab Emirates with the average gene frequencies, of about 16 percent A; 11 percent B; 71 percent O; 65 percent M; 6 percent K; 21 percent $Fy^a$; 27 percent Fy null; 6 percent $JK^a$; 39 percent $HP^1$; 9 percent $C3^F$; 15 percent $Gc^2$; 20 percent $AcP^A$; 78 percent $AcP^B$; 1.5 percent $AcP^C$; 3 percent $AK^2$; and 7 percent $PGD^C$, in general, resemble North Africans. The factors so far mentioned can be regarded as original features of the desert populations, and the isogenic lines for them form closed Curves around Arabia. There are, however, a number of other blood group genes which reach considerably higher frequencies in Arabia than anywhere in Europe, but which reach much higher frequencies still in Africa south of

Sahara. These are Henshaw (He) in the MNSs system, cDe(Ro) and V in the Rh system, $JS^a$ in the Kell system. For some of these, the data are still inadequate but in every case where the data are sufficient the isogenic lines show an area of high frequencies in Africa extending into Arabia.

The high frequencies of O,M,S, and K are clearly original characteristics of the Arabs, maintained by the isolation of the desert dwellers. They may to some extent have been maintained also by natural selection in the desert environment. The high frequencies of the typical Negroid genes must certainly be attributed in the main to incorporation of Negro slaves into the population, a process which seems to have gone on for thousands of years. Raised frequencies of these genes extend into all the Arab peoples, well beyond the limits of the Peninsular isolate, and obviously for the same reason. The long-settled Shia Moslems of the eastern Oases of Saudi Arabia, which were until recently highly malarious also have very high frequencies of glucose-6-phosphate dehydrogenase deficiency, a condition which, like sickling, due to Haemoglobin $s$,is known to protect against falciparum malaria. In some villages the frequency of the enzyme deficiency is almost as high as the extreme values found in the Kurdish Jews. The frequency of G6PD deficiency in the members of Sunni sect who entered the area more recently is almost certainly considerably lower than the Shia.

The average gene frequencies for the population of Pakistan, of about 19 percent A; 25 percent B; 55 percent 0;6@ percent $CDe(R_1)$; 24 percent cde(r); 63 percent M; 58 percent $Fy^a$; 25 percent $HP^1$; and 13 percent $AK^2$, all suggest connections with India rather than with neighbouring countries to the

west.

In Pakistan, there is a rapid increase of B gene frequency from below 23 percent to above 29 percent, and the latter level is maintained throughout almost the whole of west Pakistan, northern and Central India. Changes in A and 0 gene frequencies are less striking but there is a gradual fall in the frequency of the A gene from above 28 percent in Pakistan to values below 15 percent in India.

The 0 gene fluctuates mostly around 55 percent in Pakistan rising above 65 percent in India. The frequency of the M gene is high( average 63%) in Pakistan and this is one of the main ways in which its peoples differ serologically from the Mediterranean peoples in the strict sence. However, the presence of a relatively high frequency of the MS combination is typically Caucasoid. Rh frequencies scarcely differ from those of typical Mediterranean populations, with about 60 percent CDe($R_1$), 24 percent of cde(r) and other combinations mostly below 10 percent. Both CDE($R_Z$) and Cde(r') are present to the extent of about 2 percent each, and such frequencies are typical of the Indian region. Southward and eastward CDe rises and cde falls in frequency.

Generally, as in the Indian region, the frequency of B is high in Pakistan. The high B frequency in Pakistanis and Indians is in some way a response to the environment, and a result of natural selection acting over the last 4000 years.

The average gene frequencies for the Afghan population, of about 20 percent A; 25 percent B; 53 percent CDe($R_1$); 23 percent cde(r); 64 percent M; 57 percent $FY^a$; 26 percent $HP^1$; 12 percent $C3^F$; 69 percent $AcP^B$; 1 percent $AcP^C$;

8.5 percent $AK^2$; 24 percent $PGM_1^2$ ; and 13 percent $ADA^2$, all show both Caucasoid and Mongoloid characteristics. They have average 20 percent of A genes, including 5 percent of A2 which is within the European range, they have also 25 percent of B which is far from being so. Their M gene frequency of 64 percent is typically Asiatic, but the presence of 24 percent of MS shows Caucasoid associations. In the Rh system the cde frequency of 23 percent is very low for a Caucasoid population, whilst cDE level of 19 percent suggests central Asiatic relationships, and the presence of $Di^a$ positive is clearly a Mongoloid feature. They thus resemble many of the peoples living among the mountains on the northern boundary of the Indian region, in showing characteristics of both major races.

CHAPTER 6

# The ability to taste phenylthiocarbomide (PTC)

## 6.1.Introduction

A chance observation by Fox in 1932 was the first to demonstrate the polymorphism with respect to taste sensitivity to the simple chemical compound phenylthiocarbamide (phenylthiourea or PTC). He showed that some people are unable to taste this substance, which others describe as very bitter. Being a chemist he also showed that a considerable number of other closely related compounds, all containing the chemical grouping H-N-C= S, were tasted by the PTC tasters but not by the non-tasters.

It has been shown that this dimorphism in sensitivity is inherited as a simple Mendelian character, the ability to sense the bitterness of PTC being predominant. (Blackeslee and Salmon, 1931; Snyder, 1932).Thus, the inability to taste high concentrations of this group of substances caused by the presence of an autosomal recessive gene in homozygous form. There is however, incomplete penetrance of the taster gene. However, the simple genetic basis of this trait is that non-tasters are homozygous recessive(tt), and tasters either homozygous or heterozygous dominants (TT, or Tt).

Since at this time very few genetical polymorphisms were known in man, this pair of characters was rapidly and extensively applied by geneticists to the study of families and of populations. This is one of the few commonly investigated polymorphisms in man which does not depend upon blood tests.

Unfortunately the early workers, as well as some more recent ones, treated the distinction between tasters and non-tasters as absolute.Most early workers used a rather strong solution, whilst others used the pure crystals, or pieces of filter paper soaked in the solution and dried.Results tended to be erratic, with apparent frequencies of tasters varying widely in the same or closely related populations, or between males

and females in one population.Harris and Kalmus(1950)showed that the distinction was by no means an absolute one, and that reliable results could be obtained only by the use of solutions of known concentration. They devised a method for ascertaining the lowest concentration which could be tasted by each person. They prepared a saturated solution of PTC in tap water, and from this a series of twofold dilutions. Starting with the weakest solution, the various dilutions are successively presented to the subject until he claims to be able to taste one. Two glasses of this dilution and two of tap water are then presented, and he is asked to say which is which. If he answers correctly the dilution is taken to mark his threshold, but if he gives the wrong answer the experiment is repeated with the next strongest solution, and so on.

Nearly every population shows a bimodal distribution of thresholds with a clear-cut intermediate dilution level at which few or no thresholds fall. Those who can taste solutions more dilute than this critical value are classed as tasters, and those thresholds fall at higher concentrations, are non-tasters.

Thus, purely as a taxonomic character the taster phenomenon has not so far proved to be of great value. It is, nevertheless, of great potential importance in population studies because of its relation to thyroid function and its possible involvement in detectable natural selective effects.

The history of this line of investigation goes back to a date before Fox discovered the taster character in man. In 1928 Chesney et al studied goitres in rabbits fed exclusively

on cabbage. Subsequent work showed that the disturbance of thyroid function must have been due to the presence of 1-5-vinyl-2-thio-oxazolidone(goitrin) in these plants,and that a number of other related compounds had a similar effect on the thyroid: this led to the adoption of thiouracil as an antithyroid drug for treating cases of human hyperthyroidism. It later became clear that it was precisely those compounds which showed the taster effect which had antithyroid activity and that, there is a considerable group of substances which are repressors of thyroid activity and to which individuals respond similarly either as tasters of all or as non-tasters of all.(Harris and Kalmus,1949).They mention the importance of the occurence of such substances in food plants and conclude, 'Differences in taste sensivity may reflect differences between individuals with respect to these substances. Such metabolic differences could presumably affect the biological fitness of the three genotypes (TT, Tt, tt) and hence lead to a balanced polymorphism.

Work on the association between chemical constitution and physiological activity was summarized by Harris and Kalmus (1949). This work led to the investigations by Harris and Kalmus, and Trotter (1949) and subsequently by Kitchin et al (1959) of the distribution of taste sensitivity among patients suffering from thyroid disease. Both groups of workers agreed in finding a lower frequency of tasters in cases of nodular-non-toxic goitre, and Kitchin et al also found a significantly raised frequency of tasters in patients with diffuse toxic goitre. Several subsequent investigations in different countries have confirmed these results, but in the

Japanese who normally have a low frequency of non-tasters and in south American Indians who are nearly all tasters, no significant association between goitre and tasting could be found.

Most of the early series of non-toxic goitres were sporadic rather than endemic, but in the Netherlands and in Israel persons with endemic goitre have shown a lowered frequency of tasters.

Moreover, in persons without thyroid disease, Widstrom and Henschen(1963) have shown that there is a statistical correlation between the level of protein-bound iodine in the serum and the ability to taste PTC, persons with a high iodine level (indicating high thyroid activity) being more frequently tasters than those with a low level. This work appears convincing and is consistent with observations on persons with goitres, suggesting that the goitrous cases represent the clinically abnormal extremes of the normal thyroid function range. Unfortunately this work has never been repeated on adequate numbers of subjects, but the results of Becker et al (1966) who investigated relatively small numbers of patients for a possible association between PTC tasting and glaucoma, are in agreement with those of Widstrom and Henschen. But an extensive unpublished survey by Tills and Lehmann(1975), shows no significant differences in thyroxine levels between tasters and non-tasters.

While the situation demands much further work in order to clarify it, many of the above mentioned facts would accord with the suggestion by Harris and Kalmus(1949) of a balanced polymorphism related to the amount of iodine and of thyroid-

inhibiting substances in diet, the frequency of tasters, who fix iodine more efficiently, becoming raised by natural selection to cope at a population level with a low environmental iodine supply or an abundance of thyroid inhibitors,and lowered to meet the opposite conditions. To revert to the hypothesis of a balanced polymorphism, one set of observations which gives some support to it is that of Cartwright and Sunderland(1967) who have shown that the higher incidence of endemic goitre is accompained by a higher incidence of non-tasters.

The implication of the above hypothesis is that the products of the taster and non-taster genes affect not only the ability to taste phenylthiocarbamide and related substances,but also in some way regulate thyroid function. They must therefore be active both in the tongue (for a direct action on the nerves of taste is most unlikely) but also in the thyroid gland(or, rather improbably, the pituitary). The combination of effects on the tongue and the thyroid is not surprising in view of their embryological association.

An indication that the taster phenomenon is of importance in relation to the endocrine balance of healthy persons is the finding by Johnston et al (1966) of a tendency of earlier pubertal development in tasters than in non-tasters. This is perhaps related to a higher level of thyroid activity.The lower incidence of dental caries in tasters than in non-tasters found by Chung et al (1964) is perhaps a related phenomenon.

Ever since the discovery of thyroxine, if not before, it has been realized that goitre distribution is inversely re-

lated to the amount of iodine in the diet (especially the drinking water). Other inorganic (and possibly organic ) substances may be involved. A considerable number of the above themes are brought together in a comprehensive review paper by Fischer(1967).

Taking all the evidence into account it may be suggested as a working hypothesis that the occurence of goitre depends, probably among other factors, on the amount of iodine and of antithyroid substances in the diet, and on the PTC taster status of each individual.

It is already clear that taster status is a biochemical character the effects of which extend far beyond the taste papillae of the tongue. It would be most satisfactory if the cumbersome taste test could be replaced by a biochemical test on blood, or perhaps a metabolic test with ingestion of a thiocarbamide derivative and measurement of a metabolic product in the urine.

As a long-term result one might hope to prove the existence of a balanced polymorphism, with an equilibrium betwe-en the taster and non-taster genes depending upon the amounts of iodine and of thiocarbamide derivatives in the diet, and also probably upon the demands of the physical environment for a high or a low level of general metabolism and hence of thyroid activity.

While the presence of a relation between the taster phenomenon and thyroid function is beyond doubt, whatever may be its explanation, the same cannot be said of the suggested relation of tasting to glaucoma. There are a number of papers, mostly of rather early date, suggesting an association between goitre and glaucoma. Becker and Morton (1964 a, b) have found a raised frequency of non-tasters in primary open-angle glaucoma,and of tasters in angle-closure glaucoma, both in Caucasoids and Negroids.

Other conditions which, on adequate numbers, show a strong association with tasting are tuberculosis and leprosy while, on rather small numbers, there are very strong associations with carcinomas of the breast, cervix and corpus uteri, and ovary, and with menopausal metrorrhagia. There exists a highly significant association between diabetes mellitus and non-tasting. Apparent associations of cystic fibrosis and schizophrenia with non-tasting may be due to the patients failing to taste PTC, or to report tasting it, for reasons other than being genetic non-tasters.

6.2.Material and method

Tests for ability to taste PTC were made on six out of the ten Iranian population groups. A total of 1114 individuals was examined.

The method used was a modification of the Harris-Kalmus two stage ( a subjective followed by an objective test ) technique (Harris and Kalmus 1949).

The test is in two parts, the first giving the approximate and the second the true threshold.

All solution from number 1-11 were used. The dilutions and

controls were made up with local tap water and administered at room temperature. Fresh solutions were made regularly and all the tests were performed in the same way. The strongest solution used,number one, contains 1300 mgs per litre and this is then progressively diluted as follows:

Concentration of PTC solutions

| Solution number | PTC mg /litre |
|---|---|
| 1 | 1300.00 |
| 2 | 650.00 |
| 3 | 325.00 |
| 4 | 162.50 |
| 5 | 81.25 |
| 6 | 40.63 |
| 7 | 20.31 |
| 8 | 10.16 |
| 9 | 5.08 |
| 10 | 2.54 |
| 11 | 1.27 |

Typically, in populations tested so far, there is a bimodal distribution of tasting acuity.

At whatever solution number the antimodal value falls, let as assume at solution 4, half the frequency of individuals tasting at that solution number are allocated to the taster category with solution numbers 5,6,7,8,9,10 and 11, and the other half to the non-taster category including solutions 1, 2 and 3 as well as complete non-tasters(Sunderland 1966; Mitchell 1972).

The method of determining PTC taste thresholds is very suitable for field conditions.

## 6.3. Results

Table 6.3. demonstrates the distribution of PTC taster and non-taster phenotypes and respective gene frequencies in the six Iranian samples.

The frequency of the allele t ranges from 23.50 percent in the Zoroastrians to 49.36 percent in the Turks of Shirvan, Khorasan.

## 6.4. Discussion and conclusion

The distribution of PTC taster and non-taster phenotypes and respective gene frequencies in Iranian and neighbouring populations is presented in Tables 6.4.

The frequency of the allele t ranges from 23.50 to 52.40 percent in Iranians, being lowest in the Zoroastrians (present study) and highest in the Kurds of Sanandaj(Lightman et al, 1970). With an average t gene frequency of 41.14 percent, the Iranian population appears to exhibit a lower t gene frequency than that of around 50 percent found in Europeans( Mourant et al, 1976).

Regarding neighbouring groups, the frequency of the gene t in the populations of the Caucasus varies between 41.32 percent in the Kakhetians, Gurdjaani(Voronov, 1973) and 61.03 percent in the Imeretians of Georgia (Bunak, 1960). With an average t gene frequency of 49.43 percent, the population of the Caucasus seems to show a higher t gene frequency than that found in Iranians but slightly lower than the European frequency.

The t gene frequency of 44.67 percent in the Turks of Turkey (Say,1966) is also higher than the average for Iran but lower than the frequency in Europe.

The frequency of the gene t in the populations of Iraq ranges from 41.55 percent in the Jews to 54.31 percent in the Moslems of Baghdad( Boyd and Boyd, 1941). With an average t gene frequency of 48.77 percent, the Iraqi population also appears to exhibit a higher t gene frequency than that found in Iranians but lower than the European frequency.

The t gene frequency in the populations of Pakistan varies between 48.23 percent in the Chitrali(Graziosi,1964 ) and 75.59 percent in the East of Indus sample of Bernhard (1967). With an average t gene frequency of 60.55 percent, the Pakistani population seems to show a much higher t gene frequency than that found in Iranians and even much higher than the European frequency. High frequencies of the gene t in Pakistan seem to be more similar to the higher values found in India.

Conclusion

Throughout Europe the frequency of non-tasters is not far from 30 percent, corresponding to a frequency of the non-taster gene of a little above 50 percent. The relatively few results from other regions show that there are significantly fewer non-tasters among Africans than among Europeans and that a very large proportion of American Indians are tasters (Mourant et al, 1976).

In Iranian and neighbouring populations, with the exception of the Pakistanis with their much higher frequencies which are more similar to the high values in Indians,frequencies of the gene t appear to be a little under European levels.

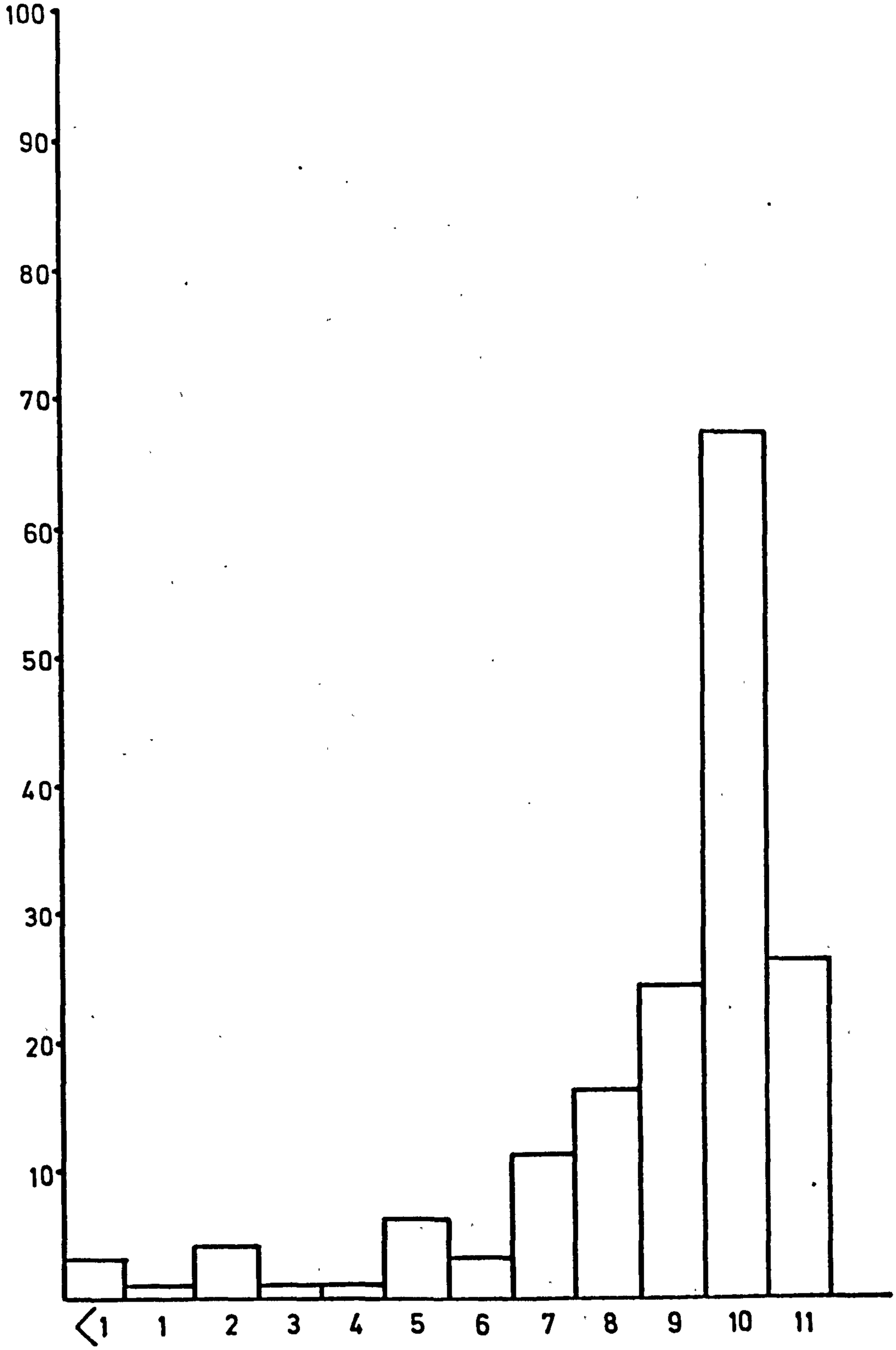



Fig. 6.3.1. The distribution of taste thresholds for P.T.C of the Zoroastrians.

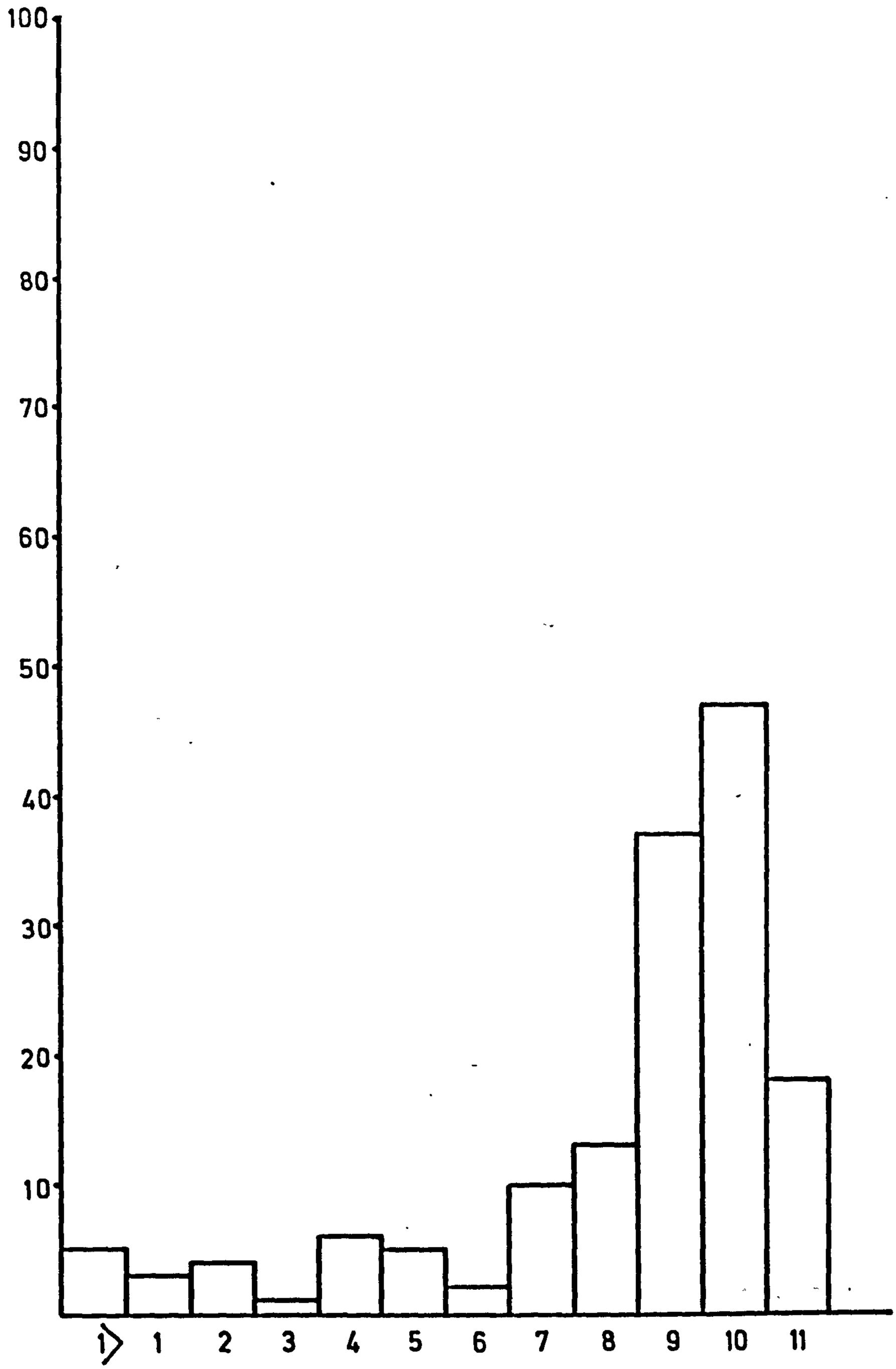



Fig. 6.3.2. The distribution of taste thresholds for P.T.C of the Kurds of Rezaieh.

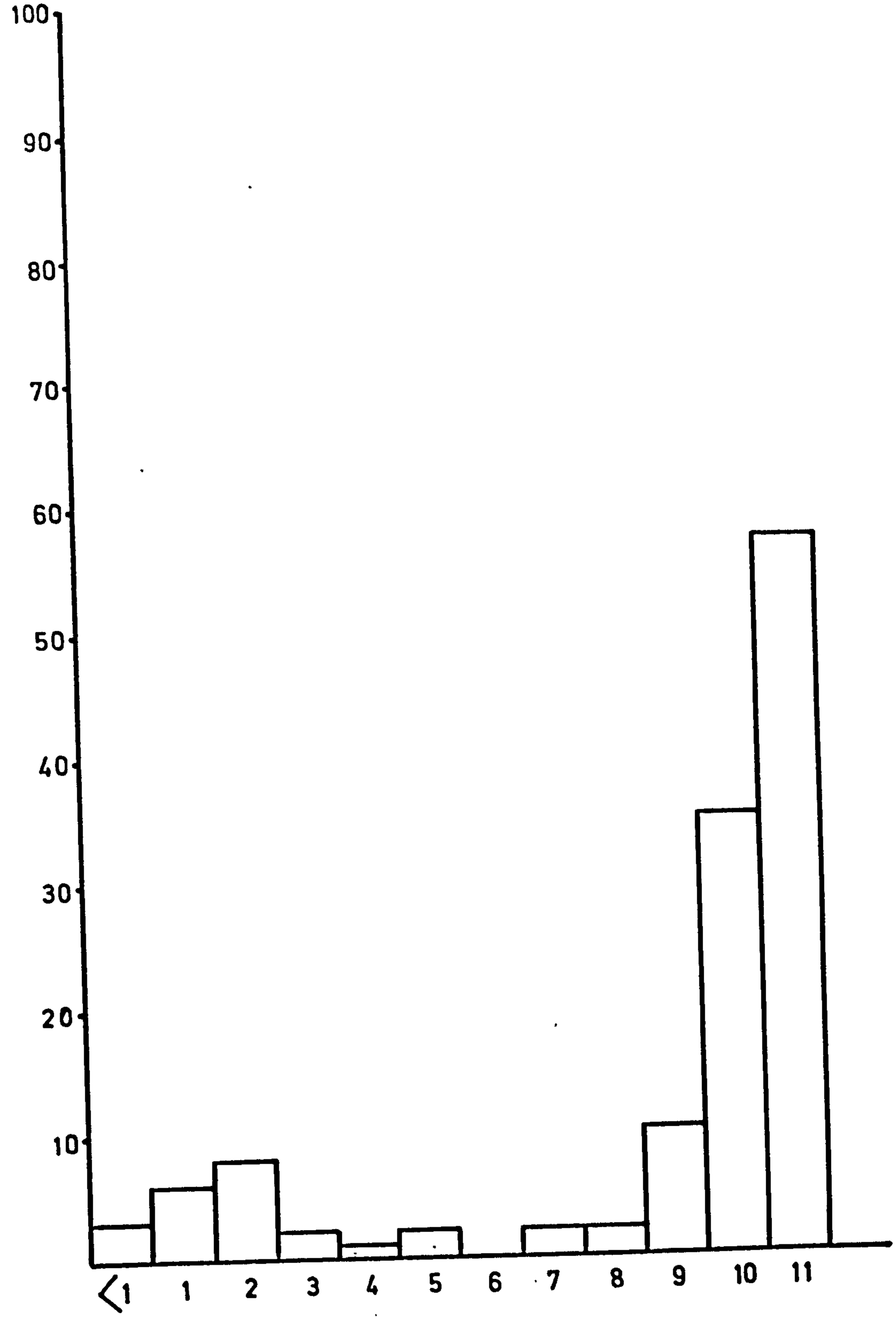



Fig. 6.3.3. The distribution of taste thresholds for P.T.C of the Kermanis.

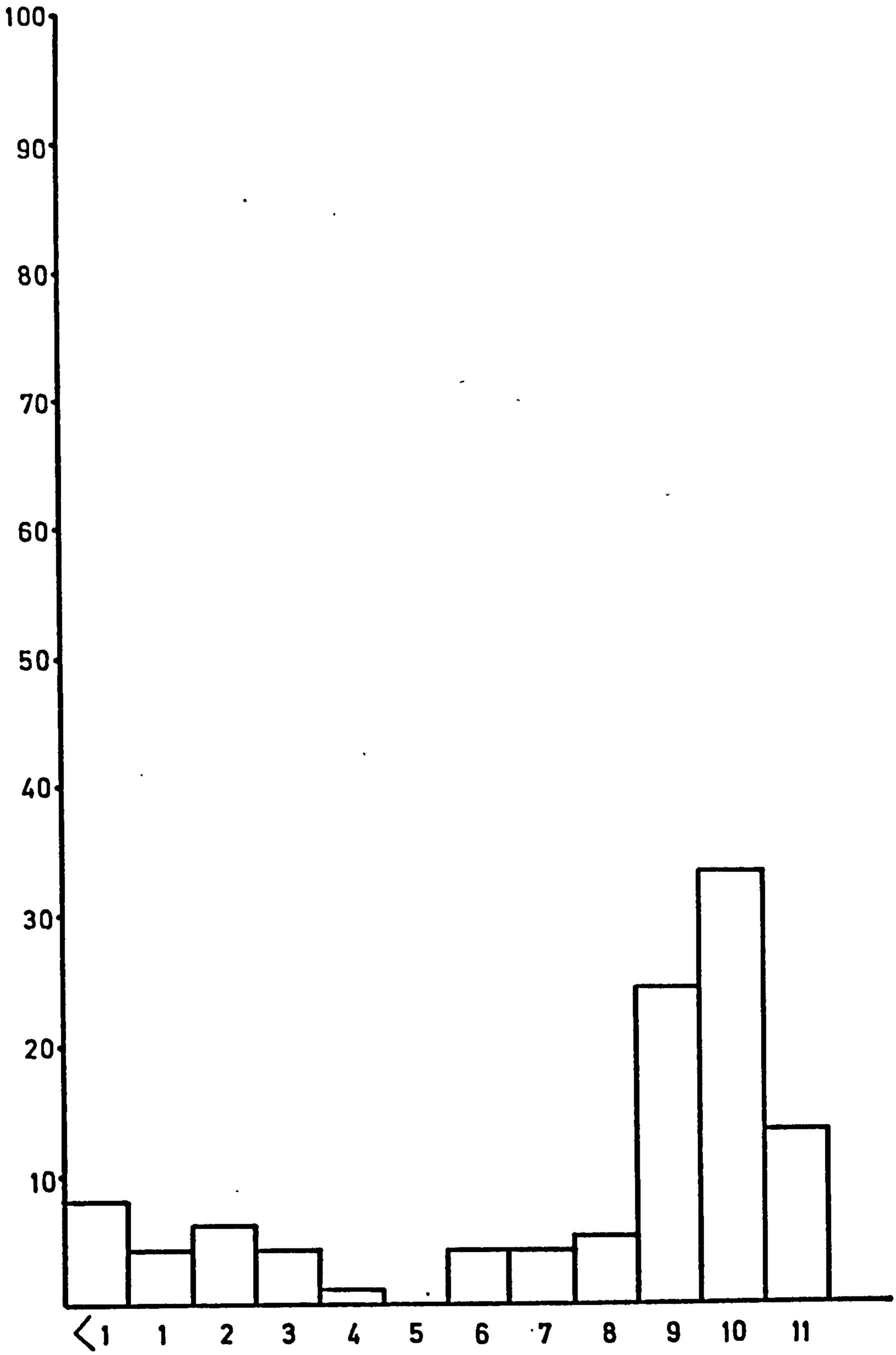



Fig. 6.3.4. The distribution of taste thresholds for P.T.C of the Kurds of Shirvan, Khorasan.

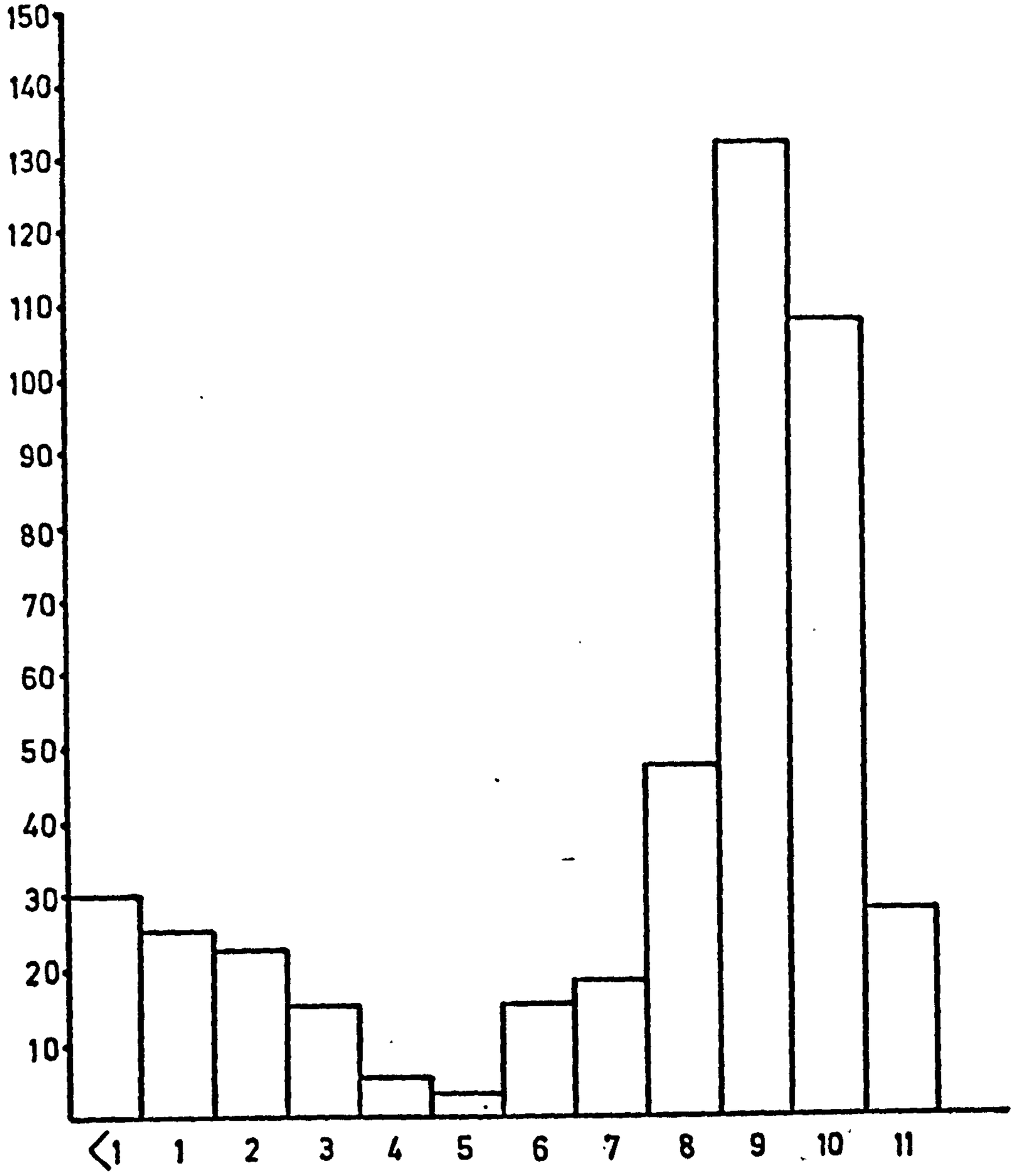



Fig. 6.3.5. The distribution of taste thresholds for P.T.C,of the Tehranis.

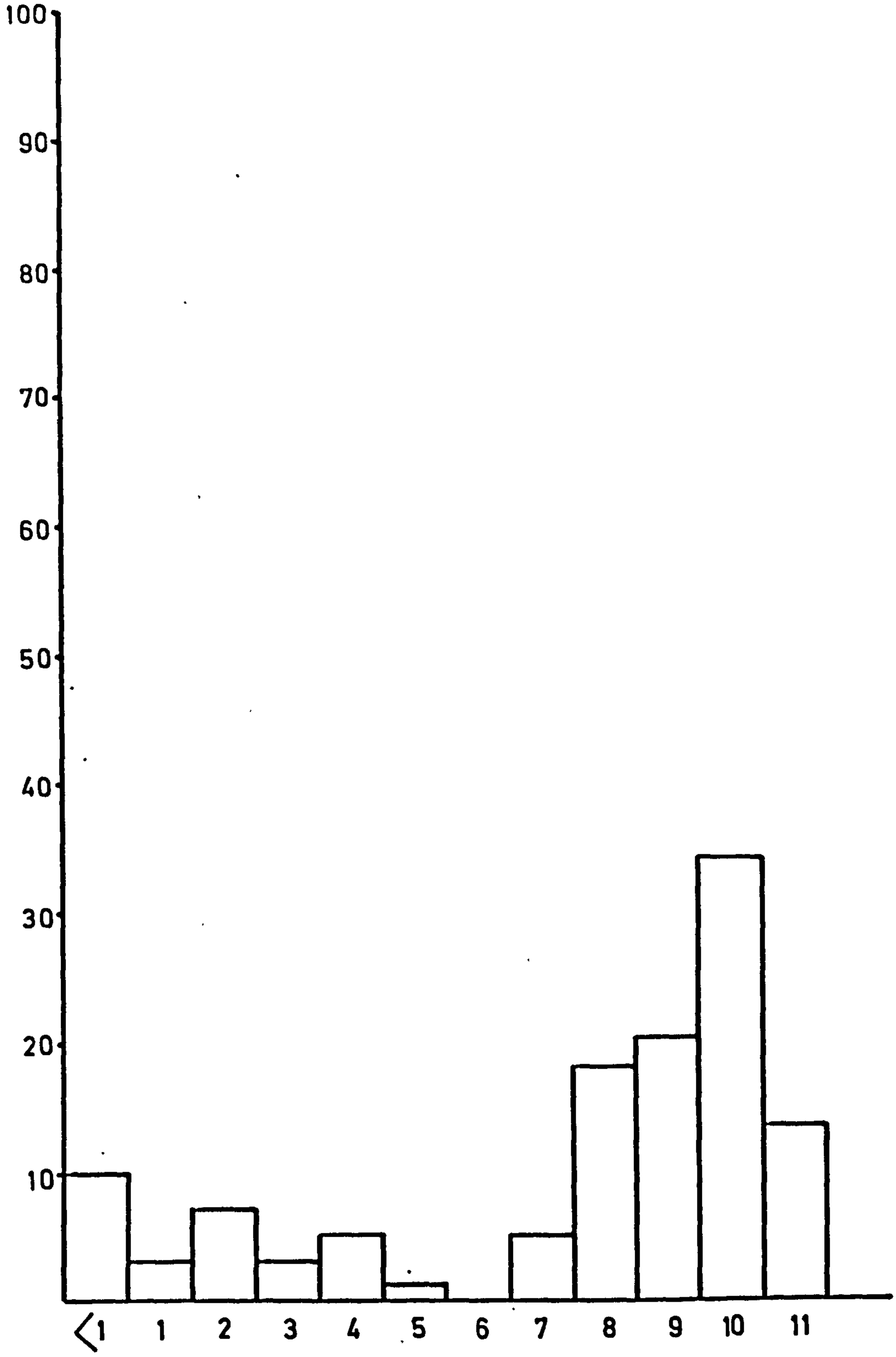



Fig. 6.3.6. The distribution of taste thresholds for P.T.C of the Turks of Shirvan, Khorasan.

Table 6.3. Phenylthiocarbamide (PTC) taster and non-taster phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | Gene frequencies | |
|---|---|---|---|---|---|
| | | Tasters | non-tasters | T | t |
| 1-Zoroastrians | 163 | 154 | 9 | 76.50 | 23.50 |
| 2-Kurds.Rezaieh | 151 | 138.50 | 12.50 | 71.23 | 28.77 |
| 3-Kerman | 128 | 106 | 22 | 58.54 | 41.46 |
| 4-Kurds.Shirvan,Khorasan | 106 | 83 | 23 | 53.42 | 46.58 |
| 5-Tehran | 447 | 348.50 | 98.50 | 53.06 | 46.94 |
| 6-Turks.Shirvan,Khorasan | 119 | 90 | 29 | 50.64 | 49.36 |

Table 6.4.I Phenylthiocarbamide (PTC) taster and non-taster phenotypes and gene frequencies distribution in Iran

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Tasters | Non-tasters | T | t | |
| 1-Zoroastrians | 163 | 154 | 9 | 76.50 | 23.50 | Present study. |
| 2-Kurds.Rezaieh | 151 | 138.50 | 12.50 | 71.23 | 28.77 | Present study. |
| 3-Jews | 336 | 282 | 54 | 59.91 | 40.09 | Sheba,C.,et al. 1962 |
| 4-Kerman | 128 | 106 | 22 | 58.54 | 41.46 | Present study. |
| 5-Kurds.Shirvan,Khorasan | 106 | 83 | 23 | 53.42 | 46.58 | Present study. |
| 6-Tehran | 447 | 348.50 | 98.50 | 53.06 | 46.94 | Present study. |
| 7-Turks.Shirvan,Khorasan | 119 | 90 | 29 | 50.64 | 49.36 | Present study. |
| 8-Kurds.Sanandaj | 346 | 251 | 95 | 47.60 | 52.40 | Lightman,S.L.,et al.1970 |

Table 6.4.II Phenylthiocarbamide (PTC) taster and non-taster phenotypes and gene frequencies distribution in the Caucasus

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | Tasters | Non-tasters | T | t | | |
| 1-Kakhetians.Gurdjaani | 205 | | | 58.68 | 41.32 | Voronov,A.A. | 1973 |
| 2-Azerbaijanians.Nukha | 196 | | | 56.55 | 43.45 | Voronov,A.A. | 1973 |
| 3-Kakhetians.Georgia | 124 | 100 | 24 | 56.01 | 43.99 | Bunak,V.V. | 1960 |
| 4-Azerbaijanians. Shemakha | 180 | | | 52.86 | 47.14 | Voronov,A.A. | 1973 |
| 5-Western Georgia | 455 | 349 | 106 | 51.73 | 48.27 | Boyd,W.C.,& Boyd,L.G. | 1937 |
| 6-Kartalins.Georgia | 121 | 91 | 30 | 50.21 | 49.79 | Bunak,V.V. | 1960 |
| 7-Azerbaijanians.Barda | 158 | | | 46.05 | 53.95 | Voronov,A.A. | 1973 |
| 8-Gurians and Mingrelians Georgia | 32 | 22 | 10 | 44.10 | 55.90 | Bunak,V.V. | 1960 |
| 9-Imeretians.Georgia | 204 | 128 | 76 | 38.97 | 61.03 | Bunak,V.V. | 1960 |

Table 6.4. III Phenylthiocarbamide (PTC) taster and non-taster phenotypes and gene frequencies distribution in Turkey

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Tasters | Non-tasters | T | t | |
| 1-Ankara. Asia Minor | 2000 | 1601 | 399 | 55.33 | 44.67 | Say,B.,et al. 1966 |

Table 6.4. IV Phenylthiocarbamide (PTC) taster and non-taster phenotypes and gene frequencies distribution in Iraq

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors | |
|---|---|---|---|---|---|---|---|
| | | Tasters | Non-tasters | T | t | | |
| 1-Jews.Baghdad | 168 | 139 | 29 | 58.45 | 41.55 | Boyd,W.C.,& Boyd,L.G. | 1941 |
| 2-Baghdadi Jews | 200 | 159 | 41 | 54.72 | 45.28 | Sirsat,Satyava-ti M. | 1956 |
| 3-Kurds.Baghdad | 23 | 17 | 6 | 48.92 | 51.08 | Boyd,W.C.,& Boyd,L.G. | 1941 |
| 4-Cristians.Baghdad | 60 | 44 | 16 | 48.36 | 51.64 | Boyd,W.C.,& Boyd,L.G. | 1941 |
| 5-Moslems.Baghdad | 322 | 227 | 95 | 45.69 | 54.31 | Boyd,W.C.,& Boyd,L.G. | 1941 |

Table 6.4. VIII Phenylthiocarbamide(PTC) taster and non-taster phenotypes and gene frequencies distribution in Pakistan

| Population | Number Tested | Phenotypes | | Gene frequencies | | Authors |
|---|---|---|---|---|---|---|
| | | Tasters | Non-tasters | T | t | |
| 1-Chitrali.Chitral | 43 | 33 | 10 | 51.77 | 48.23 | Graziosi.1964:edited by Bernhard,W. 1967 |
| 2-Calash.Chitral | 60 | 45 | 15 | 50.00 | 50.00 | Graziosi.1964:edited by Bernhard,W. 1967 |
| 3-Kati.Chitral | 33 | 22 | 11 | 42.27 | 57.73 | Graziosi.1964:edited by Bernhard,W. 1967 |
| 4-Pathan.Dir,Malakand, Mohmands,Swat | 111 | 67 | 44 | 37.04 | 62.96 | Bernhard,W. 1967 |
| 5-Pathan.Kohat,Mardan, Peshawar | 148 | 78 | 70 | 31.22 | 68.78 | Bernhard,W. 1967 |
| 6-East of Indus | 49 | 21 | 28 | 24.41 | 75.59 | Bernhard,W. 1967 |

CHAPTER 7.

Colour-blindness

## 7.1.Introduction

One of the recessive sex-linked characters known in human beings is the defect in vision called colour blindness. The gene basic to this defect is carried in the x chromosome. (Pickford,1956). Various types of this abnormality which have been studied genetically are red, green, red-green, blue-yellow, and complete colour-blindness. In the latter the afflicted person has difficulty distinguishing any colour. Total colour-blindness is very rare. The four kinds of partial colour blindness are conditioned by recessive genes carried at distinct loci in the sex chromosomes. The genes for red,green, and blue-yellow are located in the non-homologous portionoof the x chromosome. These genes are x-linked and are transmitted from an afflicted man to his daughter, who is generally normal, to a grandson who again shows the defect. Total colour blindness is conditioned by a recessive gene at a locus in the homologous parts of the x and y chromosome. Total colour-blindness may be inherited through both sexes. Women may transmit it in the x chromosome through the eggs. Men may transmit it in either the x or the y chromosome through the two types of sperms. Total colour-blindness is another of the conditions probably associated with inbreeding, as occurs in cousin marriages.

There are commonly assumed to be three sets of colour-sensitive eye cells, or cones, sensitive to red, green, and blue( or violet). Blue sensitivity is very rarely affected for genetic reasons defects of colour-vision which are inherited are almost without exception defects of the red/gre-

en system. At the outset it should be pointed out that colour blindness, in which a person can perceive only shades of grey, is a rare phenomenon (with a frequency of less than one per 10 million). Colour-blindness affecting the perception of blue is also an extremely uncommon deficiency, often brought about by injury or disease rather than genetic factors. Instead, what is commonly referred to as " colour-blindness" is a difficulty in distinguishing certain colours in the range from red to green as the rest of the population does. This is not actually a blindness, despite its name, and might be better thought of as colour-confusion. It is a fairly common defect of vision in man. The character expresses itself in an inability to distinguish accurately between red and green colours. In females, with two X chromosomes, the recessive genes must be in the homozygous state to manifest themselves. Males have only a single X chromosome, and in them a single recessive gene can determine colour-blindness. Because of the relationship of the hereditary factor to the X chromosomes, more men than women show colour-blindness.

Actually, there are several types or degrees of red and green colour-blindness, each presumably determined by its own gene, perhaps at a different locus in the X chromosome. Possibly multiple alleles are involved in the different degrees of defect in each case.

There are cases of red-blindness and others of green-blindness, the sense for both colours, however, being somewhat impaired in the two types. It is clear that both types of colour-blindness are similar to some extent; they can readily be distinguished, however, because persons with the red defect will show a greatly reduced sensitivity to pure red, but

not to pure green, whereas those with the green defect have reversed characteristics.

In about sixty percent of the cases, however, the reduction in pigment is much less extreme so that discrimination between red and green is more easily made, but sometimes with difficulty.A common question is: how do the moderately red/green colour-deficient see red and green? They will, in fact, see red and green at the proper points in the spectrum,but the limited response of one of the two types of cones tends to obscure the sharp differences between the two colours of which normal people are quite aware.

This does not, however, impair their ability to distinquish red from green, as in traffic lights, provided that the lights are bright and not too distant.

As mentioned above colour vision deficiency of congenital origin is the commonest form of colour vision disturbances. Most cases of congenital colour vision deficiency are characterized by a red-green deficiency which may be of two types; first, a protan type which may be absolute (protanopia) or partial(protanomalia), and secondly, a deutan type which may be absolute(deuteranopia) or partial(deuteranomalia).

In protanopia, the visible range of the spectrum is shorter at the red end compared with that of the normal, and that part of the spectrum which appears to the normal as blue-green, appears to those with protanopia as grey.

The whole visible range of the spectrum in protanopia consists of two areas which are separated from each other by this grey part. Each area appears to those with protanopia as one system of colour with different brightness and saturation

within each area, the colour in one area being different from that of the other. The red with a slight tinge of purple which is the complementary colour of blue-green appears also as grey.

In deuteranopia, that part of the spectrum which appears to the normal as green, appears as grey, and the visible range of the spectrum is divided by this zone into two areas,each of which appears to be one system of colour. The visible range of the spectrum is not contracted, in contrast to protanopia. Purple-red which is the complementary colour of green appears also as grey.

In protanomalia and deuteranomalia, there is no part of the spectrum which appears as grey, but the part of spectrum which appears to those with protanopia as grey, appears to those with protanomalia as a greyish indistinct colour,and likewise, the grey part of the spectrum seen by the person with deuteranopia appears to those with deuteranomalia as indistinct colour close to grey. Consequently, one of the peculiarities of red-green deficiencies is that blue and yellow colours appear to be remarkably clear compared with red and green colours.

In the congenital colour vision deficiencies, although very rare, there is total colour-blindness which may be typical or atypical. The subject who suffers from typical total colour-blindness shows a complete failure to discriminate any colour variations, usually with an associated impairment of central vision with photophobia and nystagmus. In the atypical total colour-blindness, the colour sensitivity to red and green, as well as to yellow and blue is so low that only very clear colours can be perceived, but, except for the

clour sensitivity, there is no abnormality in the visual functions.

Furthermore, a failure in the appreciation of blue and yellow may be termed tritanomalia if partial,and tritanopia if absolute, but, even if such cases do exist,they ar extremely rare.

In most populations there are about three times as many deutan males as protan males. There are very significant differences in the total frequency of colour-blindness between populations.In general,the more "Primitive"the population,the lower the frequency.Thus,in hunting and gathering communities the frequency of colour-blindness is about 1-2 percent whereas in an industrial society it is around 7 percent.It has been suggested by Neel and Post(1963)and by Post (1962)that colour-blindness may be disadvantage to primitive people because it impairs their ability to distinguish the flora and fauna that are essential for their livelihood. The negative selection against colour-blindness has most likely been relaxed in more modern societies,which would explain its higher frequency in such societies. Whatever the mechanism, colour-blindness presumably had a selective advantage at one time in man's evolution.

According to Post(1962), it is expected that there will be high prevalence of colour-blindness incidence among the populations belonging to pastoral-agricultural economies or settled habitat and low incidence rate in the hunter and food gatherer groups due to the effect of relaxed selection. Virtually all the tribals practise agriculture and secondarily, hunting. Nevertheless, in order to study the selective mechanisms, populations belonging to "culturally advanced "

societies and " Primitive tribals" can be taken into account (Dutta, 1966).

The " primitive tribal" category people having their primitive cultural background have the lowest mean prevalence rates of defective colour-vision. It may by thought that life would be handicapped with such a condition, and the low prevalence may, for one reason, be due to the result of the high degree of selection pressure, whereas the culturally advanced people, the " advanced non-tribal", have exhibited the higher overall prevalence rates of defective colour-vision, which may, at least in part, be due to the low degree of selection pressure. If these two divisions are compared, it appears that the higher prevalence rates of the advanced non-tribals might have been caused by the effect of relaxed selection against colour-blindness.

If the present polymorphism is transient rather than stable, this evolutionary trend may, perhaps, have been reversed by the relaxation of selection against colour-blindness in modern society. If colour-blindness were a stable polymorphism in primitive societies and also now in modern society, it would seem most likely that it is maintained by female heterozygote advantage, since it is hard to believe that selection with respect to colour-blindness in the two sexes could be in opposite directions. It should be noted, however,that for mutation alone to cause the increase from 1 to 6 percent would require a time that is much greater than the length of time separating primitive and modern societies.

*The* best known of tests for colour-blindness are those of Professor Shinobu Ishihara, who constructed a series of

plates with numbers made up of coloured dots. The normal person associates all reds together and all greens together, regardless of shade or mixture, and sees one number. The colour-blind, being less sensitive to the distinctions between red and green, will associate the lighter shades with each other, regardless of whether they are yellow, pink, or light green, and so see an entirely different number.Different plates can be designed to determine whether it is the red or green cone that is deficient, and whether the red/green colour-blindness is partial or complete.

Such tests are now routinely included in the series of eye tests given to driver's license applicants and it is not unusual for a male in his late teens or early twenties to discover for the first time that he has colour vision deficiency, but has quite successfully and unconsciously compensated for it in his early years. Generally speaking, such colour-deficient persons will be able to discriminate among red, green, and amber sufficiently to qualify for a driver's license.

## 7.2. Material and method

Tests for colour-blindness were made on six out of the ten Iranian samples.A total of 1370 individuals including 795 males and 575 females was tested.The tests were performed by means of the Ishihara book of 24 colour plates, edition 1974. The plates were read in well illuminated rooms during daylight, shaded from strong sun.The answers to the plates were recorded and colour defect assessed according to the method prescribed by Ishihara(1974). In accordance with Ishihara's statement on the analysis of the results, if, of the first 17 cards, 13, or more

were read correctly, the vision was regarded as normal. If nine or fewer were read correctly, then the preception of colour was regarded as defective. Individuals reading 10, 11 or 12 cards correctly were not encountered.

However, the use of Ishihara plates is not without strong limitations and pitfalls. Krill et al (1966) suggested that the Ishihara tests strength " lies in its ability to differentiate the normal from the abnormal and no more". Salzano (1972) supports this view, commenting that the Ishihara plates detect only a fraction of the colour-blinds present in a group, and ideally therefore,surveys should be conducted with portable anomaloscopes. The problem is that their use involve complex time consuming procedures and this was the case with the present study. So, in the present study no attempt was made to separate protanopes from deuteranopes or to classify the subject according to the degree of the defect.

## 7.3. Results

The frequencies of red-green colour-blindness for the six Iranian samples(795 males and 575 females) are shown in Table 7.3. Of all the six male series,the Turks of Shirvan, Khorasan record the maximum frequency of colour-blindness as 7.81 percent and the Tehranis the minimum of 2.53 percent. No colour-blind individual was found in any female series in the present investigation.

## 7.4. Discussion

The frequencies of red-green colour-blindness in Iranian and neighbouring populations are presented in Tables 7.4. As can be seen from the tables, there is little information about the frequency of colour-blindness in Iran and neigh-

bouring countries and what there is has been obtained with the Ishihara plates which are known to be somewhat unreliable in discriminating anomalous trichromats ( Kalmus , 1965).

The frequency of defective red-green vision ranges from 2.53 to 8.1 percent in Iranian males, being lowest in the Tehrani sample ( present study) and highest in the Kurds of Sandaj (Lightman et al, 1970). With an overall male frequency of 4.88 percent, the population of Iran appears to exhibit a lower frequency of the trait than that of around 8 percent found in Europeans ( Klein and Franceshetti, 1964).

Regarding neighbouring populations, the only reported frequency of 8.19 percent of red-green defect is that of Al-Amood et al (1981) for the Arabs of Iraq which is higher than the average for Iran and very similar to the European frequency.

There is no information about the frequency of colour vision defects in other neighbouring populations.

Table 7.3. Frequency of colour-blindness in Iran

| Population | Number Tested | | Sex | Normal | Colour-blind | |
|---|---|---|---|---|---|---|
| | | | | | No. | % |
| 1-Tehran | 646 | 316 | M | 308 | 8 | 2.53 |
| | | 330 | F | 330 | - | 0.00 |
| 2-Kerman | 140 | 97 | M | 94 | 3 | 3.09 |
| | | 43 | F | 43 | - | 0.00 |
| 3-Kurds.Rezaieh | 154 | 61 | M | 59 | 2 | 3.28 |
| | | 93 | F | 93 | - | 0.00 |
| 4-Kurds.Shirvan,Khorasan | 141 | 108 | M | 103 | 5 | 4.63 |
| | | 33 | F | 33 | - | 0.00 |
| 5-Zoroastrians | 133 | 85 | M | 81 | 4 | 4.70 |
| | | 48 | F | 48 | - | 0.00 |
| 6-Turks.Shirvan,Khorasan | 156 | 128 | M | 118 | 10 | 7.81 |
| | | 28 | F | 28 | - | 0.00 |

Tale 7.4. I Frequency of colour-blindness in Iranian males

| Population | Number Tested | Normal | Colour-blind | | Authors |
|---|---|---|---|---|---|
| | | | No. | % | |
| 1-Tehran | 316 | 308 | 8 | 2.53 | Present study. |
| 2-Kerman | 97 | 94 | 3 | 3.09 | Present study. |
| 3-Kurds.Rezaieh | 61 | 59 | 2 | 3.28 | Present study. |
| 4-Kurds.Shirvan,Khorasan | 108 | 103 | 5 | 4.63 | Present study. |
| 5-Zoroastrians | 85 | 81 | 4 | 4.70 | Present study. |
| 6-Turks.Shirvan,Khorasan | 128 | 118 | 10 | 7.81 | Present study. |
| 7-Kurds.Sanandaj | 504 | 463 | 41 | 8.1 | Lightman,S.L.,et al. 1970 |

Table 7.4. IV Frequency of colour-blindness in Iraq

| Population | Number Tested | Sex | Normal | Colour - blind | | Authors |
|---|---|---|---|---|---|---|
| | | | | No. | % | |
| 1-Arabs.Basrah | 2066 1221 | M | 1121 | 100 | 8.19 | Al-Amood,Wathib.S., |
| | 845 | F | 818 | 27 | 3.02 | et al. 1981 |

REFERENCES

1- Abramson,N.,Alper, C.A., Lachmann,P.J., Rosen, F.S., and Jandl, J.H. 1971
Deficiency of C3 inactivator in man. J. Immun. 107:19

2- Afkari, A.H. 1967
Statistical study of ABO groups in Khorasan. J.Mashad. Med. School. 10, 3: 286-293

3- Agarwal, D.P., Benkmann, H.G., & Goedde, H.W. 1972
Genetic polymorphism of the third component of complement (C3) and levels of $\beta 1^C$/ $\beta 1^A$/globulin in sera of German and Spanish populations. Hum. Hered. 22: 356-361

4- Ager, J.A.M., and Lehmann, H. 1961
Laboratory detection of abnormal haemoglobins. Ass. Clin. Path., broadsheet No. 33

5- Aksoy, M., Dincol, G., and Erdem, S. 1980
Survey on haemoglobin variants, $\beta$ thalassaemia Glucose-6-Phosphate dehydrogenase Deficiency and Haptoglobin types in Turkish people living in manavgat, Serik and Boztepe (Antalya). Hum. Hered. 30, 1: 3-6

6- Aksoy, M., and Erdem, S. 1968
Determination of G-6-PD and other enzymes in Turkish people.(in Turkish).J. Istanbul.Med.Fac. 31: 39-49

7- Aksoy, M., Ikin, Elizabeth W., Mourant,A.E., 1958
and Lehmann, H.
Blood groups, heaemoglobins, and thalassaemia
in Turks in southern Turkey and Eti-Turks. Brit.
Med. J. ii : 937-939

8- Al-Amood, W.S., Mohammed, S.G., Al-Sanawi,D.A. 1981
H., Radhi, A.R.H., and Rehman, B.A.
Incidence of colour blindness in Iraqi-Arabs.
Hum. Hered. 31: 122-123

9- Alciati, G. 1968
Caratteri empotipologici di una popolazione del-
lo Swat(Pakistan occidentale). Riv.Antrop.55:
203-210

10- Al-Khafaji,S.D., and Al-Rubeai, M.A.F. 1976
The frequencies of the ABO and Rh(D) blood
groups in the Kurdish population of Iraq. Ann.
Hum. Biol. 3: 189-191

11- Allen, F.H., Corcoran, Patricia A., Kenton,H. . 1958
B., and Breare, Nancy.
$M^g$, a new blood group antigen in the MNS system.
Vox Sang. 3: 81-91

12- Allen, F.H., Diamond, L.K.,and Niedziela, B. 1951
A new blood group antigen. Nature, Lond. 167:482

13- Allen, F.H., and Lewis, S.J. 1957
$KP^a$( Penny) a new antigen in the Kell blood
group system. Vox Sang. 2: 81-87

14- Allen, F.H., Lewis, S.J., and Fudenberg, H. 1958
Studies of anti-$KP^b$, a new antibody in the Kell blood group system. Vox Sang. 3: 1-13

15- Allison, A.C. 1960
Glucose-6-Phosphate dehydrogenase deficiency in red blood cells of East Africans. Nature. 186: 531-532

16- Allison, A.C. 1963
Malaria and glucose-6-Phosphate dehydrogenase deficiency. Nature. 197: 609

17- Allison, A.C. 1975
Abnormal hemoglobin and erythrocyte enzyme deficiency traits. In: Human variation and natural selection. Vol. 13. Roberts, D.F.(ed.),PP. 101-122. London: Taylor and Francis.

18- Allison, A.C., and Clyde, D.F. 1961
Malaria in African children with deficient erythrocyte glucose-6-phosphate dehydrogenase. Brit. Med. J. i: 1346-1349

19- Alper, C.A. 1969
Genetic polymorphism of human C3; in H.Peeters, ed., Protides of the biological fluids.Proceedings of the seventeenth colloquium. PP. 295-300. Elsevier, Amsterdam.

20- Alper, C.A. 1970
Genetic polymorphism of human C3. In: Protides of the biological fluids. 17.Coll.Bruges, hrsg.Von.H.Peeters.Pergamon Press, Oxford.(S.295).

21- Alper, C.A., Colten, H.R., Gear, J.S.S., Rabson, A.R., and Rosen, F.S. Homozygous C3 deficiency. J. Clin.Invest. 57: 222-229 1976

22- Alper, C.A., Colten, H.R., Rosen, F.S., Rabson, A.R., and Macnab, W. Homozygous deficiency of C3 in a patient with repeated infections. Lancet. ii: 1179 1972

23- Alper,C.A. and Propp, R.P. Genetic polymorphism of the third component of human complement(C3). J. Clin. Invest. 47: 2181-2191 1968

24- Alper, C.A., Propp, R.P., Klemperer, M.R., Rosen, F.S., and Watson, L. Inherited deficiency of the third component of human complement (C3).J.Clin.Invest. 48:553-557 1969

25- Alper, C.A., and Rosen, F.S. Genetics of the complement system. Adv.Hum. Genet. 7: 141-188 1976

26- Altay,C., Say, B., and Tuncbilek, E. Frequency of red cell adenosine deaminsae and 6-phosphogluconate dehydrogenase in a sample of the Turkish population. Hum. Hered. 24, 3: 306-308 1974

27- Altay, C ., and Yetgin, S. Haemoglobin S and some other haemoglobinopathies in Eti-Turks. Hum. Hered. 28, 1: 56-61 1978

28- Alter, A.A., Gelb, A.G., and Lee, S.L. 1962
Hemolytic disease of the newborn caused by a new antibody(anti-$Go^a$). 9th.Congr.Int.Soc. Blood Transfus.,Mexico. 341-343

29- Altland, K.,Hackler , R., and Knoche, W. 1980
Double one-dimensional electrophoresis of human serum transferrin; a new high resolution screening method for genetically determined variation. Hum. Genet. 54: 221-231

30- Ananthakrishnan, R. 1975
6-Phosphogluconatdehydrogenase. In:Humangenetik, Band 1/3. Becker, P.E.(ed). Stuttgart:Thieme.

31- Ananthakrishnan, R., and Beck, W. 1973
A mother-child combination analysis for ABO-HP interaction. Humangenetik. 18: 202-206

32- Ananthakrishnan, R., and Walter, H. 1972
Some notes on the geographical distribution of the human red cell Acid phosphatase phenotypes. Humangenetik. 15: 177-181

33- Arasteh, A.R. 1964
Man and Society in Iran. E.J. Brill, Leiden.

34- Arnaud, P., Burdash, N.M., Wilson,G.B., and 1976
Fudenberg H.H.
$Alpha_1$-antitrypsin (Pi) types in Down's syndrome. Clin. Genet. 10: 239-243

35- Arvilommi, H. 1973
Genetic Polymorphism in serum C3 in Finns. Vox. Sang. 25: 13

36- Aseyeva, S.M. 1936
Gruppy Krovi u nasele niya Abkhazii.Trudy.Trop. Inst. Abkhazii.2: 110-117

37- Ashley, M.J., Chan-Yeung, M.,and Corey, P.N. 1980
Pi Phenotypes in North American workers: racial differences and comparison with reported frequencies. Hum. Hered. 30: 107-111

38- Ashton, G.C. 1965
Cattle serum transferrin: a balanced polymorphism. Genetics. 52: 983-997

39- Attabudin 1954
Cited in: Boyd, W.C., and Boyd, Lyle G.
The blood groups in Pakistan. Amer. J. Phys.Anthrop. 12: 393-405

40- Axelsson, U., and Laurell, C.B. 1965
Hereditary variants of serum $\alpha_1$-antitrypsin. Amer. J. Hum. Genet. 17: 466

41- Azen,E.A., and Smithies, O. 1968
Genetic polymorphism of C3 ( $\beta_1$C-globulin) in human serum. Science. 162: 905-907

42- Azen, E.A., Smithies, O., and Hiller, O 1969
High-voltage starch-gel electrophoresis in the study of post-albumin proteins and C3 ( $\beta_1$C-globulin) polymorphism. Biochem. Genet. 3: 215-228

43- Azhir, A. 1951
Personal communication. Cited in Mourant et al. 1976

44- Badakere, S.S., Vasantha, K., Bhatia,H.M., 1980
Ala, F., Clarke, V.A., Rustom Moesri., Sringarm Sommai., and Amin, A.B.
High frequency of $In^a$ antigen among Iranians and Arabs. Hum. Hered. 30: 262-263

45- Baecher. 1935
Cited in: Mizan et al. 1965

46- Bajatzadeh, M., and Walter, H. 1968
Serum Protein Polymorphism in Iran. Humangenetik. 6: 40-54

47- Bajatzadeh, M., and Walter, H. 1969 a
Investigations on the distribution of blood and serum groups in Iran. Hum. Biol. 41, 3: 401-415

48- Bajatzadeh, M., and Walter, H. 1969 b
Studies on the population genetics of the cerulo-plasmin polymorphism. Humangenetik. 8: 134-136

49- Balakrishnan , V., Sanghvi, L.D., and Kirk, R.L.1975
Genetic diversity among Australian aborigines.Australian institute of aboriginal studies, Canberra.

50- Bargagna, M., Domenici, R., and Morali, A. 1975
Red cell esterase-D polymorphism in the population of Tuscany. Humangenetik. 29: 251-253

51- Baxi, A.J., and Camoens, H. 1969
Studies on haptoglobin types in various Indian Populations. Hum. Hered. 19: 65

52- Beaconsfield, P., Mahboubi, E., and Rainsbury,R.1967
Epidemiologie des glukose-6-phosphate dehydroge-

nase. Mangels, Munch, Med. Wschr. 109: 1950 - 1952

53- Beadle, G.E., and Tatum, E.L. 1941
Genetic control of biochemical reactions in neurospora. Proc. Nat. Acad. Sci., Wash. 27: 499-506

54- Bearn, A. G. 1966
Wilson's disease, in stanbury J.B., Wyngaarden, J.B., and Fredrickson, D.S.(eds.) The metabolic basis of inherited disease, 2nd edn.P. 761. McGraw-Hill, New York.

55- Bearn, A.G., Kitchin, F.D., and Bowman, B.H. 1964
Heterogeneity of the inherited group-specific component of human serum. J. Exp. Med. 120: 83

56- Becker, B., Kolker, A.E., and Ballin,N. 1966
Thyroid function and glaucoma. Amer. J. Ophthal. 61: 997-999

57- Becker, B., and Morton, W.R. 1964
Phenylthiourea taste testing and glaucoma.Arch. Ophthal., Chicago. 72: 323-327

58- Beckett, P.H. 1950
Unpublished report of Oxford University expedition to Persia: Cited in Mourant. 1954

59- Behzad, D., Lee, C.L., Gavin, J., and Marsh,W.L.1973
A new anti-erythrocyte antibody in the Duffy system. Vox Sanguins. 24: 337-342

60- Benbanaste, M. 1963
Distribuzione del sistema ABO e del fattore Rh in Turchia. Haemat. lat.(Milano). 6: 95-96

61- Bender, K. 1975
Genetik der Menschlichen Komplement Proteine. Edited by Becker, P.E. Stuttgart, Georg Thieme-Verlag.

62- Bender, K., und Frank, R. 1974
Esterase-D polymorphismus: Darstellung in der Hochspannungselektrophorese und Mitteilung der allgemeinhaufigkeiten. Humangenetik. 23:315-318

63- Benercetti, A., Silvano,S., Cattanei,A., and Khan, P.M. 1972
A new variant allele $AK^5$ of the red cell adenylate Kinase polymorphism in a non-tribal Indian population. Hum. Hered. 22: 171

64- Benkmann, H.G., and Goedde, H.W. 1974
Esterase D polymorphism: Gene frequencies and family data. Humangenetik. 24: 325-327

65- Berelian-Jahanshahi, F. 1973
Blood groups in Iran. Acta Medica Iranica. 16: 52-57

66- Berg, K., Noer, G., and Heiberg, A. 1970
Studies on the genetic polymorphism of the C3 of human serum. Clin. Genet. 1: 177-180

67- Berg, K., Schwarzfischer, F., and Wischerath,H. 1976
Esterase D polymorphism: Description of the"New" allele $EsD^4$. Hum. Genet. 32: 81-83

68- Bernhard, W. 1967 a

Die Haufigkeit der ABO Blutgruppen und des Rhesus faktors(D) in der Gebirgsregion Nord West Pakistan. Anthrop. Anz. 30: 128-134

69- Bernhard, W. 1967 b

Die Haufigkeit der PTC-schmeckfahigkeit in Nord West Pakistan. Anthrop. Anz. 30: 135-141

70- Bernstein, F. 1924

Ergebinss einer biostatischen Zus ammen fassenden Betrachtung uber die erblichen Blutstruckturen des Menschen. Klin. Wschr. 3: 1495. Cited in Race and Sanger. 1970

71- Beutler, E., and Yoshida, A. 1973

Human glucose-6-phosphate dehydrogenase variants: a supplementary tabulation. Ann.Hum.Genet. 37: 151-155

72- Bidar, A. 1956

Personal communication. Cited in Mourant et al. 1976

73- Bienzle. U., Ayeni, O., Lucas, A.O., and Luzatto, L. 1972

Glucose-6-phosphate dehydrogenase and malaria. Greater resistance of females heterozygous for enzyme deficiency and of males with non-deficient variant. Lancet. 1: 107-110

74- Bissbort, S., Kompf, J., Bethge,R., and Gussmann, S. 1975

Population genetics of human red cell phosphoglu-

comutase isozyme $PGM_3$(E.C.: 2.7.5.1.).Humangenetik. 27: 57-58

75- Blake, N.M. 1976
Glutamic pyruvic transaminase and esterase D types in the Asian-Pacific area. Hum.Genet. 35: 91-102

76- Blake, N.M. 1978
Malate dehydrogenase types in the Asian-Pacific area, and a description of new phenotypes. Hum. Genet. 43: 69-80.

77- Black, J.A., and Dixon, G.H. 1968
Amino-acid sequence of alpha-chains of human haptoglobins. Nature, Lond. 218: 736-741

78- Blake, N.M., and Kirk, R.L. 1969
New genetic variant of 6-phosphogluconate dehydrogenase in Australian aborigines. Nature, Lond. 221: 278

79- Blake, N.M., Kirk, R.L., McDermid, E.M., Omoto, K., and Ahuja, Y.R. 1971
The distribution of serum protein and enzyme group systems among North Indians. Hum.Hered. 21:440-457

80- Blake, N.M., Kirk, R.L., Simons, M.J., and Alpers, M.P. 1970
Genetic variants of soluble malate dehydrogenase in New Guinea populations. Humangenetik.11:72-74

81- Blake, N.M., Saha, N., McDermid,E.M., Kirk, R.L., and Crane, G.G. 1974

Additional electrophoretic variants of 6-phosphogluconate dehydrogenase. Humangenetik. 21: 347-354

82- Blakeslee, A.F., and Salmon, M.R. 1931
Odour and taste blindness. Eugen.News. 16: 105-108

83- Blumberg, B.S. 1963
Polymorphisms of the human serum proteins and other biological systems, In: the genetics of migrant and isolate populations, ed. by Elizabeth Goldschmidt. New York, Williams and Wilkins. 20-26

84- Bodmer,W.F., and Cavalli-Sforza L.L. 1976
Genetics, evolution, and man. W.H. Freeman, San-Francisco. PP. 718-723

85- Bottini, E., Lucarelli.P., Bastianon.V., and Gloria, F. 1972
Erythrocyte acid phosphatase polymorphism and hemolysis. J.Med. Genet. 9: 434-435

86- Boue', A., and Boue', Joelle. 1955
Etude sur la repartition des groups sanguins en Iran. Sang. 26: 705-713

87- Boue', A., and Boue', Joelle. 1956
Etude sur la repartition des groups sanguins en Iran. ii, Ann. Inst.Pasteur. 91: 898-911

88- Bowman, J.E. 1959
Personal communication. Cited in Mourant et al. 1976

89- Bowman, J.E. 1964
Haptoglobin and transferrin differences in some Iranian populations. Nature, Lond. 201: 88

90- Bowman, J.E., Frischer, H., Ajmar, F., Carson, P.E., and Gower, M.K. 1967
Population, family and biochemical investigation of human adenylate Kinase polymorphism. Nature, Lond. 214: 1156-1158

91- Bowman, J.E., and Ronaghy, H. 1967
Haemoglobin, glucose-6-phosphate dehydrogenase, phosphogluconate dehydrogenase, and adenylate Kinase polymorphism in Moslem in Iran. Amer. J.Phys. Anthrop. 27, 2: 119-123

92- Bowman, J.E., and Walker, D.G. 1961 a
The origine of Glucose-6-phosphate dehydrogenase deficiency in Iran: theoretical considerations. 2nd. Int. Congr. Hum. Genet. Rome. 1: 583-586

93- Bowman, J.E., and Walker, D.G. 1961 b
Virtual absence of glutathione instability of theerythrocytes among Armenians in Iran. Nature, Lond. 191: 221-222

94- Boyd, W.C. 1939
Blood groups. Tabul. biol.,Hague. 17, ii: 113-240

95- Boyd, W.C., and Boyd, Lyle G. 1937
Sexal and racial variations in ability to taste phenylthio-carbamide. With some data on the inheritance. Ann. Eugen., Lond. 8: 46-51

96- Boyd, W.C., and Boyd, Lyle G. 1941
Blood groups and types in Baghdad and Vicinity.
Hum. Biol. 13: 398-404

97- Boyd,W.C., and Boyd, Lyle G. 1954
The blood groups in Pakistan. Amer. J. Phys. Anthrop. 12: 393-405

98- Boyer, J.T., Gall, E.P., Norman, M.E., Nilsson, 1975
U.R., and Zimmermann, T.S.
Hereditary deficiency of the seventh component of complement. J. Clin. Invest. 56: 905

99- Braidwood, R.J., How, B., and Reed,C.A. 1961
The Iranian Prehistoric Project. Science. 133: 2008-2010

100- Brain, P. 1966
Subgroups of A in the South African Bantu.Vox. Sang. 11: 686-698

101- Brinkmann, B. 1974
Occurrence of silent allele $P^0$ in 10 German families. Z. Rechtsmed. 75: 25-30

102- Brinkmann, B., Krukenberg, P., Brinkmann,M., 1972
and Hoppe, H.H.
Gene frequencies of soluble glutamic-pyruvic-transaminase in a Northern German population (Hamburg). Humangenetik. 16: 355-356

103- Brinkmann, B., Reiter, J., and Kruger, O. 1973
Genhaufigkeiten einiger Enzympolymorphismen in Mittelmeerlandern. Humangenetik. 20: 141-146

104- Bronnestam,R. 1971

Genetic polymorphism of the complement component C3 in a northern Swedish population.Hum.Hered. 21:263

105- Bronnestam, R. 1973

Studies of the C3 polymorphism. Relationship between C3 phenotypes and rheumatoid arthritis. Hum. Hered. 23: 206-213

106- Bronnestam, R., and Cedergren, B. 1973

Studies of the C3 polymorphism.Hum.Hered. 23: 214-219

107- Bulletin of the World Health Organization. 1968

39: 935

108- Bunak, V.V. 1960

Anthropological composition of the population of the Caucasus, In: contributions to the physical anthropology of the Soviet union.ed.by V.V. Bunak et al. Cambridge, Mass., Peabody Mus.,192 PP. (Peabody Museum Russian Translations series, 1, no. 2).

109- Callender, S.T., and Race, R.R. 1964

A serological and genetical study of multiple antibodies formed in response to blood transfusion by a patient with lupus erythematosus diffusus. Ann. Eugen. 13: 102-107

110- Callender, S.T., Race, R.R., and Paykoc, Z.V. 1945

Hypersensitivity to transfused blood. Brit. Med. J. ii: 83

111- Cameron, G.L., and Staveley, I.M. 1957
Blood group P substance in hydatidcyst fluid.
Nature, Lond. 179 : 147

112- Cannings, C., and Cavalli-Sforza, L.L. 1974
In: Advances in human genetics, P. 105, Editors:
H. Harris and K. Hirschorn. Plenum Press, New York
and London.

113- Carter N.D., Fildes,R.A., Fitch,L.I., and Parr, 1968
C.W.
Genetically determined electrophoretic variants
of human phosphogluconate dehydrogenase.Acta Genet. Basel. 18: 109-122

114- Cartwright, R.A., Bethel, I.L., Hargreaves,H., 1976
Izatt,M., Jolly, J., Mitchell, R.J., Sawhney,K.S.,
Smith, M., Sunderland, E., and Teasdale, D.
The red blood cell Esterase D polymorphism in Europe and Asia. Hum. Genet. 33: 161-166

115- Cartwright, R.A., and Sunderland, E. 1967
Phenylthiocarbaide(PTC) tasting ability in population in the North of England: with a endemic goitre. Acta Genet. Basel. 17: 211-221

116- Cavalli-Sforza, L.L. 1973
Analytic review. Some current problems of human population genetics. Amer. J. Hum. Genet. 25: 82-104

117- Chakraborty, R. 1980
Gene diversity analysis in nested subdivided populations. Genetics. 96: 721-726

118- Chakraborty, R., Haag,M., Ryman,N., and Stahl,G. 1982
Hierarchical gene diversity analysis and its application to brown trout population data. Heredi-
tas. 97: 17-21

119- Chakraborty, R., and Tateno, Y. 1976
Correlations between some measures of genetic
distance. Evolution. 30: 851-853

120- Chalmers, J.N.M., Ikin, E.W., and Mourant,A.E. 1953
The ABO, MNS and Rh blood groups of Nigerians.
Ann. Eugen, Lond. 17: 168-176

121- Chandanayingyong, D., Sasaki, T.T., and Greenwalt 1967
T.J.
Blood groups of the Thais.Transfusion, Philad.
7: 269-276

122- Chaudhri, I.M., Ikin, Elizabeth W., Mourant,A.E., 1952
and Walby, Jean A.E.
The blood groups of the people of North-West Pa-
kistan. Man. 52: 168-169

123- Chen, S.H., and Giblett, E.R. 1971
Polymorphism of soluble qlutamic-pyruvic trans-
aminase: a new genetic marker in man.
Science. N.Y. 173: 148-149

124- Chen,S.H., Giblett, E.R., Anderson, J.E., and 1972
Fossum, B.L.G.
Genetics of glutamic-pyruvic transaminase; its
inheritance, common and rare variants,population
distribution, and differences in catalytic activi-
ty. Ann. Hum. Genet. 35: 401-409

125- Chen, S.H., Scott, C.R., and Giblett, E.R. 1974
Adenosine deaminase: demonstration of a 'silent' gene associated with combined immunodeficiency disease. Amer. J. Hum. Genet. 26: 103-107

126- Chesney, A.M., Clawson, T.A., and Webster, B. 1928
Endemic goitre in rabbits. i. Incidence and characteristics. Bull. Johns Hopkins Hosp. 43: 261-277

127- Chown, B., Lewis, M., and Kaita, H. 1962
Atypical Duffy inheritance in three Caucasian families: a possible relationship to mongolism, Amer. J.Hum. Genet. 14: 301.308

128- Chown, B., Lewis, M., and Kaita, H. 1965 a
The Duffy blood system in Caucasians: evidence for a new allele. Amer. J. Hum. Genet. 17: 348

129- Chown, B., Lewis, M., and Kaita, H. 1965 b
The Kidd blood group system in Caucasians. Transfusion, Philad. 5: 506-507

130- Christie, G.S., and Judah, J.D. 1953
Intracellular distribution of enzymes. Proc.R. Soc. Biol. 141: 420

131- Chung, C.S., Witkop, C.J., and Henry,J.L. 1964
A genetic study of dental caries with special reference to PTC taste sensitivity. Amer. J.Hum.Genet. 16: 231-245

132- Clegg, E.J., Ikin, Elizabeth W., and Mourant, A.E. 1961

The blood groups of the Baltis. Vox. Sang. 6: 604-614

133- Cleve, H. 1973
The variants of the group specific component. A review of their distribution in human populations. Israel. J. Med. Sci. 9: 1133-1146

134- Cleve, H., and Bearn, A.G. 1962
The group specific component of serum: genetic and chemical consideration, in Steinberg, A.G., and Bearn, A.G.(eds) progress in Medical genetics, Vol.2, P. 64. Grune and Stratton, New York

135- Constandse Westermann, T.S. 1972
Coefficients of biological distance. Anthropological Publications. Oosterhout,N.B.The Netherlands.

136- Constans, J., Cleve, H., Bennet, A., Bouillon, R., Cox, DW., Daiger, SP., Ehnholm, Chr., Fujiki, N., Johnson, AM., Kirk, R.L., Kuhnl, P., Martin, W., Matsumoto, H., Mayr, W.R., Miyake,K., Miyazaki, T., Omoto, K., Porck, H.J.Seger, J.,Thymann,M., Tills, D., Toyomasu, M., Van Baelen,H., Vavrusa, B., and Viau, M. 1979
Group-specific component: Report on the First International Workshop. Hum. Genet. 48: 143-149

137- Constans, J., Lefevre-Witier, Ph., Richard,P., and Jaeger, G. 1980
Gc(Vitamin D Binding Protein) subtype polymorphism and variants distribution among Saharan,

Middle East, and African populations. Amer.J.Phys. Anthrop. 52: 435-441

138- Constans, J., and Viau, M. 1977
Group specific component: Evidence for two subtypes of the $Gc^1$ gene. Science. 198 : 1070-1071

139- Constans, J., Viau, M., Cleve,H., Jaeger, G., Quilici, J.C., and Palisson, M.J. 1978
Analysis of the Gc polymorphism in human populations by isoelectrofocusing on polyacrylamide gels.Demonstration of subtypes of the $Gc^1$ allele and of additional Gc variants. Hum. Genet. 41: 53-60

140- Constans, J., Viau,M., Jaeger,G., and Palisson, M.J. 1981
Gc, Tf, HP subtype and $alpha_1$-antitrypsin polymorphism in a pygmy Bi-Aka sample. Hum.Hered. 31: 129-137

141- Constans, J., Viau, M., Pison, G., and Langaney,A. 1978
Gc subtypes demonstrated by isoelectric focusing: Further data and description of new variants among an African sample( Fula) from Senegal.Jpn.J.Hum. Genet 23: 111-117

142- Cook, P.J.L. 1975
The genetics of $\alpha_1$-antitrypsin. A family study in England and Scotland. Ann.Hum. Genet. 38: 275-289

143- Coombs,R.R.A., Mourant,A.E., and Race,R.R. 1946
In-Vivo isosensitization of red cells in babies with haemolytic disease. Lancet, i: 264-266

144- Corinne, M., Yoshihara and Harvey W, Mohrenweiser. 1980
Characterization of $ACP_1$ TIC-1, an electrophoretic variant of erythrocyte acid phosphatase restricted to the Ticuna Indians of Central Amazonas. Amer. J. Hum. Genet. 32: 898-907

145- Crawford, M.N., Greenwalt, T.J., Saski, T.,Tippett, P., Sanger, R., and Race, R.R. 1961
The phenotype Lu(a-b-) together with unconventional Kidd groups in one family. Transfusion,Philad. I: 228.

146- Curtain, C.C., Gajdusek, D.C., Kidson,C ., Gorman, J.G., Champness, L.,and Rodrigue, R. 1965
Haptoglobins and transferrins in Melanesia: Relation to hemoglobin, serum haptoglobin and serum iron levels in population groups in Papua-New Guinea. Amer. J. Phys.Anthrop. 23: 363-380

147- Cutbush, M., and Chanarin, I. 1956
The expected blood group antibody, anti-$Lu^b$.
Nature, Lond. 178: 855

148- Cutbush, M., and Mollison, P.L. 1950
The Duffy blood group system. Heredity. 4:
383-389

149- Cutbush, M., Mollison, P.L., and Parkin,D.M. 1950
A new human blood group. Nature, Lond. 165: 188

150- Daiger,S.P. 1979
Biologic significance of genetic variation in human group specific component (Gc).The plasma vitamin D binding protein.Abstracts.IVth Workshop on Vitamin D,Berlin,February 18-22

151- Daiger,S.P., and Cavalli-Sforza L.L. 1977
Detection of genetic variation with radioactive ligands. II. Genetic variants of Vitamin D.Labelled group specific component(Gc)proteins.Amer.J. Hum.Genet. 29: 593-604

152- Daiger,S.P., Schanfield,M.S., and Cavalli- 1975
Sforza, L.L.
Group specific component(Gc) proteins bind vitamin D and 25 hydroxy vitamin D.Proc. Nat.Acad. Sci.(USA). 72: 2076-2080

153- Darnborough,J., Firth,R.,Giles,Carolyn M., 1963
Goldsmith, K.L.G., and Crawford, Mary N.
A 'new' antibody anti-$Lu^{a}Lu^{b}$ and two further examples of the genotype Lu(a-b-). Nature,Lond. 198:796, and personal communication to Race and Sanger. 1968

154- Daneshmand,P. 1980
Study of $alpha_1$-antitrypsin in patients with toxoplasmosis.M.Sc.thesis.Faculty of Medicin. University of Tehran.Tehran.Iran.

155- Davidson,R.G. 1967
Electrophoretic variants of human 6-phosphogluconate dehydrogenase:population and family studies and description of a new variant.Ann.Hum.

Genet. 30:355-361

156- Davidson,R.G., and Cortner,Jean A. 1967
Genetic variant of human erythrocyte malate dehydrogenase.Nature, Lond. 215:761-762

157- Decastello,A.V., and Sturli,A. 1902
Ueber die Isoagglutinine in serum gesunder und Kranker Menschen.Munch.Med.Wschr.,1090, in Race and Sanger. 1970

158- Defai,H. 1971
Personal report from evaluation of blood groups distribution in Alborz School.Tehran.Cited in: Sadatzadeh and Hamidi. 1972

159- Detter,J.C., Stamatoyannopoulos,G.,Giblett,E.R., 1970
and Motulsky, A.G.
Adenosine deaminase: racial distribution and report of a new Phenotype.J.Med.Genet. 7: 356-357

160- Detter,J.C., Ways,P.O., Giblett,E.R.,Baughan, 1968
M.A., Hopkinson,D.A., Povey,S., and Harris,H.
Inherited variations in human phosphohexose Isomerase.Ann.Hum.Genet. 31:329-338

161- Dickson,H.R.P. 1956
Kuwait and her neighbours. C.Witting, ed.London: George Allen and Unwin,Ltd., P. 26

162- Dincol, G., Erdem, S., and Aksoy, M. 1976
Transferrin types in Turkish people.Hum.Hered. 26, 5: 349-350

163- Dissing, J., and Knudsen, J.B. 1970
Human erythrocyte adenosine deaminase polymorphism in Denmark. Hum.Hered. 20: 178

164- Dissing, J., Lund,J., and Sorensen, H. 1972
C3 polymorphism in a group of old arteriscle-rotic patients. Hum. Hered. 22: 466

165- Dissing,J., and Svensmark,O. 1976
Human red cell acid phosphatase: Quantitative evidence of a silent gene $P^0$, and a Danish population study. Hum. Hered. 26: 43-58

166- Donaldson, V.H., and Evans, R.R. 1963
A biochemical abnormality in heredity angioneurotic edema. Absence of serum inhibitor of Cl-esterase.Amer. J.Med. 35: 37

167- Douglas, G.R., McAlpine, P.J., and Hamerton,J.L.1973
Regional localisation of loci for human $PGM_1$ and 6PGD on human chromosome one by use of hybrids of chinese hamster-human somatic cells. Proc.Natl. Acad.Sci.USA 70: 2737

168- Dungern, E.V., and Hirszfeld, L. 1911
uber gruppen spezifische strukturen des Blutes. Z. Immun. Forsch. 8: 526 cited in Race and Sanger. 1970

169- Dunsford, I., Ikin, E.W., and Mourant,A.E. 1953
A human blood group gene intermediate between M and N. Nature, Lond. 172: 688

170- Dutta, P.C. 1966
A review of the inherited defective colour-vision variability and selection relaxation among In-

dians. Acta Genetica et Statistica Medica,Basel. 16: 327-339

171- Edwards, A.W.F. 1971
Distances between populations on the basis of gene frequencies. Biometrics. 27: 873-881

172- Edwards, A.W.F., and Cavalli-Sforza, L.L. 1972
Affinity as revealed by differences in gene frequencies. In Weiner, J.S., and Huizinga,J.( (edt.). The assessment of population affinities. Oxford.Clarendon Press.

173- Engel, W., und Schmidtke, W. 1975
Die Bedeutung von Genduplikationen fur die Evolution der wirbeltiere.In: Humangenetik. Ein Kurzes Handbuch in funf Banden, Bd. 1/3,P.E. Becker, ed., PP. 618-645. Stuttgart: Thieme.

174- Erdem, S., and Aksoy, M. 1975
A note on the distribution of haptoglobin types in Turkey. Hum. Hered. 25, 1: 18-19

175- Erdem, S., Aksoy, M., and Cetingil, A.I. 1966
Distribution of haptoglobin types in Turkish people. Nature, Lond. 210: 315-316

176- Eriksson, S. 1964
Pulmonary emphysema and alpha$_1$-antitrypsin deficiency. Acta. Med. Scand. 175: 197

177- Eriksson, S. 1965
Studies on $\alpha_1$-antitrypsin deficiency.Acta Med. Scand. 177.(Supp. 432),1

178- Eriksson, S., and Laurell, C.B. 1963
A new abnormal serum globulin $\alpha_1$-antitrypsin.
Acta.Chem. Scand. 17: 5150

179- Eze, L.C., Tweedie, M.C.K., Bullen,M.F.,Wren, 1974
P.J.J.,and Evans D.A.P.
Quantitative genetics of human red cell acid phosphatase. Ann. Hum. Genet. 37: 333-340

180- Fagerhol, M.K. 1972
Acid starch gel electrophoresis for detection of Alpha$_1$-Antitrypsin(Pi) types: outline of techniques employed currently. In: Pulmonary Emphysema and Proteolysis. Hrsg. Von C. Mittman Academic Press, New York. (S. 145).

181- Fagerhol, M.K., and Braend, M. 1965
Serum prealbumin: polymorphism in man. Science. 149: 986-987

182- Fagerhol, M.K., and Braend, M. 1966
Classification of human serum prealbumin after Starch gel electrophoresis. Acta. Path.Microbiol. Scand. 68: 434

183- Fagerhol, M.K., and Laurell, C.B. 1967
The polymorphism of ' prealbumins' and $\alpha$1-antitrypsin in human sera. Clin.Chem.Acta.16: 199

184- Fargerhol, M.K., and Tenfjord, O.W. 1968
Serum Pi types in some European, American,Asian and African populations. Acta. Path.Microbiol. Scand. 72: 601-608

185- Fakhrai, H., Karimi Boushehri, F., Rasouli-Nia,A., 1978

Marzban., M., Tayebi, N., Yosefi, S., and Kazem-Pour, K.
Comparison of ABO blood system in Persian and Arabic speaking Khuzestanies. The seventh National Genetics Congress. Genetics Research Center.Jundishapur University. Ahwaz. Iran.

186- Fakhrai, H., and Marzban M. 1978
A Preliminary report on a possible selection pressure on MN blood system. The seventh National Genetics Congress. Jundishapur University. Genetics Research Center, Ahwaz. Iran.

187- Fakhrie, S.D. 1966
Rates of blood groups in Iraq. 11th Congr.Int. Soc. Blood Transfus., Sydney, Abstracts.P. 137

188- Farhud, D.D. 1972
Genetik und population genetik Von C3( $\beta_1$C globulin). Ph.D. Thesis. University of Mainz FRG.

189- Farhud, D.D. 1980
Haptoglobin polymorphism in the Middle east
Jpn. J. Hum. Genet. 25, 3: 203-206.

190- Farhud, D.D., and Amirshahi, P. 1974
Haptoglobin types in the Ghashghai tribe of Iran. Unpublished observations.

191- Farhud, D.D., Amirshahi, P., Amirnazmi,A., and Tavakoli, Sh. 1981
Association between PTC taster and goiter in Iran. 2nd. Internat. Anthropol. Poster.Conf. Zagreb. Yugoslavia.

192- Farhud, D.D., Amirshahi, P., Hedayat, Sh., and Sawhney, K.S. 1978
Haptoglobin polymorphism in south of Iran (Dezfool). 7th National Genetics Congress. Jundishapur University, Ahwaz. Iran.

193- Farhud, D.D., Ananthakrishnan, R., and Walter,H. 1972
Association between C3 phenotypes and various diseases. Humangenetik. 17: 57-60

194- Farhud, D.D., Ananthakrishnan,R., Walter,H., and Loser, J. 1973
Electrophoretic investigations of some red cell enzymes in Iran. Hum. Hered. 23: 263-266

195- Farhud, D.D., and Daneshmand, P. 1977
Transferrin types in some Iranian populations. unpublished observations.

196- Farhud, D.D., Hedayat, Sh., Amirshahi, P., Tavakoli Sh., Daneshmand,P., Sawhney, K.S., and Montazami, K. 1978
Tf, Gc, and CP phenotypes in favism and G-6-PD deficiency. Indian. J.Med. Res. 68: 990-994

197- Farhud, D.D., Kamali, M.S., Marzban, M., and Kamali, P. 1983
Consanguinity in Iran ( In preparation).

198- Farhud, D.D., and Walter, H. 1972
Haptoglobin subtypes in Iranians. Hum.Hered. 22: 184-189

199- Farhud, D.D., and Walter, H. 1973
Polymorphism of C3 in German, Bulgarian,Iran-

ian and Angola population. Humangenetik. 17: 161-164

200- Fiedler, H., and Pettenkofer, H. 1969
Ein 'neuer' Phanotyp im Isoenzymsystem der Phosphoglucomutasen des Menschen($PGM_1$0).2.Mitteillung. Blut. 18: 358

201- Field, H. 1939
Contributions to the anthropology of Iran. FMNH, Anthropological Series. 29: 1-706

202- Field, H. 1957
Personal communication. Cited in Mourant et al. 1958

203- Field, H. 1959
Ancient and modern man in South-Western Asia.University of Miami Press, Coral Gables, Florida.

204- Fildes, R.A. and Harris, H. 1966
Genetically determined variation of adenylate Kinase in man. Nature, Lond. 209: 261-266

205- Fildes, R.A., and Parr, C.W. 1963
Human red cell phosphogluconate dehydrogenase. Nature, Lond. 200: 890-891

206- Fildes, R.A., and Parr, C.W. 1964
Various forms of human erythrocytic-6-phosphogluconate dehydrogenase. Proc. 6th Congr.Int.Biochem. 229. Cited in Giblett. 1969

207- Fineman, R.M., Kidd, K.K., Johnson,A.M., and Breg, W.R. 1976

Increased frequency of heterozygotes for alpha$_1$-antitrypsin variants in individuals with either sex chromosome mosaicism or trisomy 21. Nature. 260: 320-321

208- Fischer, R. 1967

Genetics and gustatory chemoreception in man and other primates. In: The chemical senses and nutrition, ed. by M.R. Kare and O. Maller. Baltimore, Johns Hopkins Press. 61-81

209- Fishel, W.J. 1948

The Universal Jewish Encyclopedia, 8. New York.

210- Fisher, R.A. 1953

The variation in strength of human blood group P. Heredity. 7: 81-89

211- Fisher, R.A., quoted by Race, R.R. 1944

An incomplete antibody in human serum. Nature,Lond. 153: 771-772

212- Fitch, L.I., and Parr, C.W. 1967

( Unpublished observations); in Carter et al. 1968

213- Flatz, G., and Xirotiris, N. 1975

Glukose-6-Phosphat Dehydrogenase. In: Humangenetik,Band 1/3. Becker, P.E.(ed.). Stuttgart: Thieme

214- Forbat, A., Lehmann, H., and Silk,E. 1953

Prolonged apnoea following injection of succinyldicholine. Lancet. ii: 1067

215- Fox, A.L. 1932

The relationship between chemical constitution and taste. Proc. Natn.Acad. Sci., Wash. 18: 115

216- Frazer Roberts, J.A., and Pembrey, M.E. 1978
An introduction to medical genetics. Seventh edition. Oxford University Press. Oxford. New York.

217- Fried, K., Bloch, N., Sutton, E., Neel, J.V., 1963
Bayanisioson, P., Ramot, Brachai, and DuvDevani, P.
Haptoglobins and transferrins. In: The genetics of migrant and Isolate populations. ed. by Elizabeth Goldschmidt. 266-267

218- FuruhJelm, U., Nevanlinna,H.R., Nurkka,Riitta 1969
Gavin, June, and Sanger, Ruth.
Evidence that the antigen $Ul^a$ is controlled from the Kell complex locus. Vox. Sang. 16: 496-499

219- FuruhJelm, U., Nevanlinna,H.R., Nurkka,Riitta 1968
Gavin, June, Tippett, Patricia, Gooch, Ann, and Sanger, Ruth.
The blood group antigen $Ul^a$( Karhula). Vox. Sang. 15: 118-124

220- Gadzhiev, A.G. 1964
Blood group data of the Dagestan populations. ( In Rassian). Vop. Antrop. 17: 120-124

221- Galatius-Jensen, F. 1958
On the genetics of haptoglobins. Acta Genet. Basel. 8: 232

222- Ganrot, P.O. 1967
Studies on serum protease inhibitors with spe-

cial reference to $\alpha_2$-macroglobulin.Acta.Univ. Lund. II, 2, P.5

223-Ganrot, P.O., Laurell, C.B., and Eriksson,S. 1967
Obstructive lung disease and trypsin inhibitors in $\alpha_1$-antitrypsin deficiency. Scand.J.Clin. Lab. Invest. 19: 205

224-Garrod, A.E. 1909
Inborn errors of metabolism. Reprinted with supplements by H. Harris. London, etc., 1963(Oxford Monographs on Medical Genetics)

225-Gelpi, A.P. 1965
Glucose-6-phosphate dehydrogenase deficiency in Saudi Arabia: a survey. Blood. 25: 486-493

226-Gershowitz, H., and Fried,K. 1966
Anti-$M^V$, a new antibody of the MNS blood group system. i. $M^V$, a new inherited variant of the M gene. Amer. J. Hum. Genet. 18: 264-281

227-Giblett, E.R. 1958
Js, a 'new' blood group antigen found in Negroes. Nature, Lond. 181: 1221-1222

228-Giblett, E.R. 1967
Variant phenotypes: haptoglobin,transferrin and red cell enzymes, in Greenwalt, T.J.(ed).Advances in Immunogenetics. P. 99, J.B. Lippincott, Philadelphia.

229-Giblett, E.R. 1969
Genetic markers in human blood. Blackwell Scientific Publ., Oxford.

230- Giblett, E.R., Anderson, J.E., Cohen,F., Pol- 1972
lara, B., and Meuwissen, H.J.
Adenosine deaminase deficiency in two patients
with severely impaired cellular immunity.Lancet.
ii: 1067-1069

231- Giblett, E.R., and Scott, N.M. 1965
Red cell acid phosphatase: racial distribu-
tion and report of a new phenotype.Amer.J.Hum.
Genet. 17: 425

232- Godber, Marilyn J., Kopec, Ada C., Mourant,A. 1973
E., Tills, D., and Lehmann, E.E.
Biological studies of yemenite and Kurdish Jews
in Israel and other groups in south-west Asia.
Ix. The hereditary blood factors of the yemeni-
te and Kurdish Jews. Phil.Trans., S.B. 266, 169-
184

233- Goedde, H.W., Benkmann, H.G., and Flatz.G. 1980
$Alpha_1$-antitrypsin subtypes in the populations
of Germany, Ecuador, Afghanistan,Cameroon,and
Saudi Arabia. Z. Morph.Anthrop. 70, 3: 341-346

234- Goedde, H.W., Benkmann, H.G., Flatz, G., Rahi-1977
mi, A.G., Kaifie, S., and Delbruck, H.
Red cell enzyme polymorphisms in different po-
pulations of Afghanistan. Ann.Hum.Biol. 4,3:
225-232

235- Goedde, H.W., Benkmann, H.G.,Agarwal,D.P., 1972
and Hirth, L.
Further investigations on the inherited poly-

morphism of the third component of complement
( $\beta_1$C/ $\beta_1$A-globulin). Hum.Hered. 22:488-495

236- Goedde, H.W., Benkmann, H.G., Agarwal,D.P., 1979
Hirth,L., Bienzle, u., Dietrich, M., Hoppe,
H.H., Orlowski, J., Kohne, E and Kleihauer, E.
Genetic studies in Saudi Arabia: Red cell enzyme, Haemoglobin and serum protein polymorphisms. Amer. J.Phys. Anthrop. 50, 2: 271-278

237- Goedde, H.W., Benkmann, H.G., and Hirth, L. 1970
Genetic polymorphism of C3( $\beta_1$c-globulin)
component of complement in a German and a Spanish population. Hum. Genet. 10: 231-234

238- Goldschmidt, Elizabeth. 1963
The genetics of migrant and isolate populations. Proceedings of a conference on human population genetics in Israel, Jerusalem, 1961, Baltimore, Williams and Wilkins, XXi, 369 PP.

239- Goldschmidt, Elizabeth. 1967
Summary and conclusions. 9th Int.Congr.Life Ass. Med. Tel-Aviv. 200-206

240- Goldschmidt, Elizabeth., Fried, K., Stein- 1976
berg, A.G., and Cohen, T.
The Karaite community of Iraq in Israel: A genetic study. Amer.J.Hum.Genet. 28, 3: 243-252

241- Goodman, M., and Laseer, G.W. 1974
Measurement of distance and propinquity in

anthropological studies. Genetic distance, J.F. Crow and C. Denniston eds., Plenum press, New-York. 5-22

242- Gordon, H., Keraan, M., Woodburne,V., and Sophangisa, E. 1969
Quantitative variation of 6-phosphogluconate dehydrogenase in an African population. Nature, 221: 96-97

243- Gotze, O., and Muller-Eberhard, H.J. 1976
Adv. Immunol. 24: 1-35

244- Graziosi. 1964
Die Haufigkeit der PTC-schmeckfahigkeit in Nord west Pakistan. Anthrop. Anz. 30:135-141

245- Grikurov, V.S. 1963
Izoagglyutinatsionnye svoystvakrovi zdorovogoi bol nogo zobom naseleniya Karachaya.Trud.Kubansk. Med.Inst.16, 3: 165-170

246- Gurevitch,J.,Brzezinski,A.,and Polishuk,Z. 1947
Rh factor and haemolytic disease of the newborn in Jerusalem Jews. Lancet. ii: 943

247- Gurevitch,J.,Hasson,E.,and Margolis, E. 1956
Blood groups in Persian Jews.A comparative study with other oriental Jewish communities. Ann.Hum.Genet. 21:135-138

248- Gurevitch,J.,Hermoni,D.,and Margolis,E. 1953
Blood groups in Kurdistani Jews.Ann.Eugen., Lond. 18: 94-95

249- Gurevitch,J., and Margolis,E. 1954-5
Blood groups in Jews from Iraq.Ann.Hum.Genet.

19: 257-259

250- Gussmann, S., and Schwarzfischer,F. 1972
Rare GPT phenotypes in a random sample of southern Germany: Evidence for a third allele.
Z. Rechtsmed. 70: 251-252

251- Haddad, J.G., and Walgate, J. 1976
25 hydroxyvitamin D transport in human plasma. Isolation and partial characterization of calcifidiol binding protein.J.Bioch.Chem. 251, 16: 4803-4809

252- Hamamy, H.A., and Saeed, T.Kh. 1981
Glucose-6-phosphate dehydrogenase deficiency in Iraq. Hum. Genet. 58: 434-435

253- Harris, H. 1959a
Human biochemical genetics. Cambridge University press.

254- Harris, H. 1959b
Genetics of the plasma protein variants, In: Ciba Foundation Symposium on biochemistry of human genetics. ed. by G.E.W. Wolstenholme and Cecillia M.O' connor. London, J. and A. Churchill. 151-173

255- Harris, H. 1961
Inherited variations of human plasma proteins.
Brit. Med. Bull. 17: 217

256- Harris, H. 1970
The principles of human biochemical genetics.

North Holland Public., Amsterdam-London.

257- Harris, H. 1975
The principles of human biochemical genetics.
Amsterdam-Oxford. North-Holland.

258- Harris,H., and Gilles,H.M. 1961
Glucose-6-phosphate dehydrogenase deficiency
in the peoples of the Niger Delta. Ann. Hum.
Genet. 25: 199-206

259- Harris, H., and Hopkinson, D.A. 1972
Average heterozygosity per locus in man: an
estimate based on the incidence of enzyme poly-
morphism. Ann.Hum. Genet 36: 9-20

260- Harris, H., and Hopkinson, D.A. 1976
Handbook of enzyme electrophoresis in human ge-
netics. Amsterdam-Oxford-New York : North Hol-
land, Elsevier.

261- Harris, H., Hopkinson,D.A., and Robson,E.B. 1973
The incidence of rare alleles determining ele-
ctrophoretic variants: data on 43 enzyme loci
in man. Ann.Hum. Genet. 37: 237

262- Harris, H., Hopkinson,D.A., Robson,Elizabeth 1963
B., and Wittaker, Mary.
Genetical studies on a new variant of serum
cholinesterase detected by electrophoresis. Ann.
Hum. Genet. 26: 359-382.

263- Harris, H., and Kalmus, H. 1949a
The measurement of taste sensitivity to phenyl-
thiocarbamide. Ann. Eugen. 15: 24

264- Harris, H., and Kalmus, H. 1949b
Genetical differences in taste sensitivity to phenylthiourea and to antithyroid substances. Nature, Lond. 163: 878

265- Harris, H., and Kalmus, H. 1950
The measurement of taste sensitivity to phenylthiourea (P.T.C.).Ann. Eugen, Lond. 15: 24-31

266- Harris, H., Kalums, H., and Trotter, W.R. 1949
Taste sensitivity to phenylthiourea in goitre and diabetes. Lancet, ii: 1038-1039

267- Harris, H., Robson, E.B., and Siniscalco,M. 1959
Genetics of the plasma proteins variants, in Biochemistry of human genetics, eds. Wolstenholme, C.E. W. and O' connor, C.M. 151-173.London. Chirchill.

268- Harris, H., and Wittaker, Mary. 1962
The serum cholinesterase variants. A study of twenty-two families selected via the 'intermediate' phenotype. Ann. Hum. Genet. 26: 59-72

269- Harrison,G.A., Boyce,A.J., Hornabrook, R.W., and Craig. W.J. 1976
Association between polymorphic variety and disease susceptibility in two New Guinea populations. Ann. Hum. Biol. 3: 253-268

270- Hedayat, S., Amirshahi,P., and Khademi,B. 1969
Frequency of G-6-PD deficiency among some Iranian ethnic groups. Trop. Geogr.Med. 21: 163-168

271- Henningsen, K. 1954
The relationship between blood factors P and Q.
5th Int. Congr.Blood Transfus., Paris. 215-218

272- Henningsen,K., and Jacobsen, Tyge. 1955
Relationship between blood factors P and Q.
Nature, Lond. 176: 1179-1180

273- Herbich, J., Fischer, R.A., and Hopkinson,D.A. 1970
Atypical segregation of human red cell acid phosphatase phenotypes: evidence for a rare'silent' allele $P^0$. Ann.Hum.Genet.(Lond.) 34: 145-151

274- Herbich, J., and Meinhart, K. 1972
The rare 'silent' allele $P^0$ or $P^V$($P^{Vienna}$)of human red cell acid phosphatase,typed in a second family. Humangenetik. 15: 345-348

275- Hiernaux, J. 1977
Long term biological effects of human migration from the African savanna to the equatorial forest: A case study of human adaptation to a hot and wet climate. In: Population Structure and Human Variation. G.A. Harrison,ed. Cambridge University Press, Cambridge. PP. 187-217

276- Hirschfeld, J. 1959
Immunoelectrophoretic demonstration of qualitative differences in normal human sera and their relation to the haptoglobins. Acta Path.Microbiol. Scand. 47: 160-168

277- Hirschfeld, J., Jonsson, B., and Rasmuson, M. 1960
Inheritance of a new group-specific system demonstrated in normal human sera by means of an immu-

noelectrophoretic technique. Nature. 185: 931-932

278- Hirszfeld, L., and Hirszfeld,H. 1919
Serological differences between the blood of different races. The results of researches on the macedonian front. Lancet, ii: 675

279- Holmberg, C.G., and Laurell, C.B. 1948
Investigations in serum copper. II. Isolation of the Copper containing protein, and a description of some of its properties. Acta.Chem.Scand. 2: 550

280- Holmberg, C.G., and Laurell, C.B. 1951
Oxidase reactions in human plasma caused by ceruloplasmin. Scand. J. Clin.Lab.Invest. 31: 103

281- Hopkinson, D.A., Cook, P.J.L., and Harris.H. 1969
Further data on the adenosine deaminase(ADA) polymorphism and a report of a new phenotype.Ann. Hum. Genet., Lond. 32: 361-367

282- Hopkinson, D.A., Edwards,Y.H., and Harris,H. 1976
The distribution of subunit numbers and subunit sizes of enzymes: A study of the product of 100 human gene loci. Ann. Hum. Genet(London).39:383-411

283- Hopkinson,D.A., and Harris, H. 1965
Evidence for a second ' structural locus' determining human phosphoglucomutase. Nature, Lond. 208: 410

284- Hopkinson, D.A., and Harris H. 1966
Rare phosphoglucomutase phenotypes. Ann.Hum. Genet. 30: 167-181

285- Hopkinson, D.A., and Harris, H. 1968
A third phosphoglucomutase locus in man.Ann. Hum. Genet. 31: 359-367

286- Hopkinson,D.A., and Harris, H. 1969
Red cell acid phosphatase, phosphoglucomutase andadenylate Kinase. In: Biochemical Methods in Red cell Genetics.YUNIS, J.J.(ed). Academic Press, New York. P. 337

287- Hopkinson, D.A., Mestriner, A.A., Cortner,J., 1973
and Harris, H.
Esterase D: a new human polymorphism.Ann.Hum.Genet. 37: 119-137

288- Hopkinson, D.A., Spencer,N., and Harris,H. 1963
Red cell acid phosphatase variants: a new human polymorphism. Nature, Lond. 199:969

289- Hopkinson, D.A., Spencer,N., and Harris,H. 1964
Genetic studies on human red cell acid phosphtase. Hum. Genet. 16: 141-154

290- Horsfall, W.R., and Smithies, O. 1958
Genetic control of some human serum β-Globulins. Science. 128: 35

291- Huestis, D.W., Catien,Mary Lu., and Busch, 1964
Shirley.
A 'new' Rh antibody (anti-Rh 26) which detects a factor usually accompanying hr'.Transfusion,Phi-

lad. 4: 118-119

292- Hummel, K., Pulverer,G., Schaal, K.P., and Weidtman, V. 1970
Haufigkeit der sichttypen in den Erbsystemen Haptoglobin,Gc, Saure Erythrocyten phosphatase, Phosphoglucomutase und Adenylate Kinase Sowie den Erbeigenschaften Gm(1),Gm(2), und Inv(1)bei Deutschen (aus dem Raum Freiburgi.Br.und Koln) und bei Turken. Humangenetik. 8: 330-333

293- Ikin, E.W., and Mourant,A.E. 1951
A rare blood group antigen occurring in Negroes. Brit.Med.J. i: 465-467

294- Ikin, Elizabeth, W., and Warburton, K. 1959
Personal communication. Cited in Mourant et al. 1976

295- Ikin, Elizabeth, W., Mourant,A.E., and Lehmann, H. 1965
The blood groups and haemoglobin of Assyrians of Iraq. Man. 65: 110-111

296- Ikin,E.W., Mourant, A.E., Pettenkofer,H.J., and Blumenthal, G. 1951
Discovery of the expected haemagglutinin,anti-$Fy^b$. Nature, Lond. 168: 1077

297- Ishihara, S. 1974
The series of plates designed as test for col-our-blindness. 24 plates edition.Kanehara Shuppan Co., LTD. Tokyo. Japan.

298- Ishimoto, G., and Kuwata, M. 1974
Red cell glutamic-pyruvic transaminase polymorphism in Japanese populations. Jpn.J.Hum.Genet. 18: 373-377

299- Jack, J.A., Tippett, Patricia, Noades,Jean., 1960
Sanger,R., and Race, R.R.
$M_1$, a subdivision of the human blood-group antigen M. Nature, Lond. 186: 642

300- Jenkins, T., and Corfield, V. 1972
The red cell phosphatase polymorphism in south Africa. Population data and studies on the R,RA and RB phenotypes. Ann.Hum.Genet. 35: 379

301- Johnston, F.E., Hertzog,K.P., and Malina,R.M. 1966
Phenylthiocarbamide taste sensitivity and its relationship to growth variation. Amer.J.Phys. Anthrop. 24: 253-255

302- Jongsma, A., Vansomeren,H.,Westerfeld,A., 1973
Hagemeijer, A., and Pearson, P.
Localisation of genes on human chromosomes by studies of human-chinese hamster somatic cell hybrids. Assignment of $PGM_3$ to chromosomes C6 and regional mapping of the PGD, $PGM_1$ and PeP-C genes on chromosome A1. Humangenetik. 20: 195

303- Jorgensen,G., and Hopfer, A. 1967
Die verteilung der Gc-phanotypen und Gc-Allele bei einigen Krankheiten(Diabetes mellitus, Leberparenchymschaden, Psoriasis Vulgaris).Hum. Genet. 3: 273

304- Kalmus, H. 1965
Diagnosis and genetics of defective colour vision. Pergamon press.

305- Kalow, W. 1959
Cholinesterase types. In: Ciba foundation symposium on biochemistry of human genetics, ed. by G.E.W. Wolstenholme and Cecilia M. O'Connor. London, J. and A. Churchill. 39-56, discussion 57-59

306- Kamel, K., Chandy,R., Mousa,H., and Yunis,D. 1980
Blood groups and types,Hemoglobin variants,and G-6-PD Deficiency among Abu Dhabians in the United Arab Emirates. Amer.J.Phys.Anthrop. 52, 4: 481-484

307- Karp, G.W., and Sutton, H.E. 1967
Some new phenotypes of human red cell acid phosphatase. Amer.J.Hum.Genet. 19: 54

308- Kattamis, C.A., Chaidas,A., and Chaidas,S. 1969
G6PD deficiency and favism in the Island of Rhodes( Greece). J.Med.Genet. 6: 286-291

309- Kayssi,A.I. 1940
The Rh blood groups of the population of Baghdad. Amer.J.Phys.Anthrop. 7: 549-551

310- Kayssi, A.I., Boyd,W.C., and Boyd,L.G. 1938
Blood groups of the Bedouin near Baghdad.Amer. J. Phys. Anthrop. 23: 295-298

311- Kellermann, G., and Walter, H. 1970
Investigations on the population genetics of the

Alpha$_1$-antitrypsin polymorphism. Humangenetik.10: 145-150

312- Kennedy,W.P., and Macfarlane, J. 1936
Blood groups in Iraq.Amer.J.Phys.Anthrop. 21:87

313- Khaled,E., Al-Nassar,P., Michael Conneally, 1981
Catherine G, Palmer., and Pao-10 yu.
The genetic structure of the Kuwaiti population. Hum. Genet. 57: 192-198

314- Kherumian,R. 1948
Contribution a L'anthropologie du Caucase.
(Notes Preliminaires sur les Daghestaniens).
Anthropologie, Paris. 52: 43-80

315- Kherumian,R., Moullec, J., and Desabie,J. 1954
Les groupes sanguins A1A2Bo, MN, Rh et P chez les Armeniens et les Georgiens. 5th Int.Congr. Blood Transfus., Paris.264-270

316- Kirk, R.L. 1968
The world distribution of transferrin variants and some unsolved problems. Acta Genet.Med.(Roma). 17: 613-640

317- Kirk, R.L. 1971
A haptoglobin mating frequency, segregation and ABO blood-group interaction analysis for additional series of families. Ann.Hum.Genet. 34:329-337

318- Kirk, R.L. 1976
Genetic distance. A pragmatic approach.Reprinted from Excerpta Medica International congress series No. 411. Human genetics. Proceedings of

the fifth international congress of human genetics. Mexico City. Excerpta Medica, Amsterdam.

319- Kirk, R.L., Cleve,H., and Bearn,A.G. 1963
The distribution of the group specific component (Gc) in selected populations in South and South East Asia and Oceania. Acta Genet. Basel. 13: 140-149

320- Kirk, R.L., Bronya, Keats., Black,N.M., 1977
Mcdermid,E.M., Ala,F., Karimi,M., Nickbin, B., Shabazi, H., and Kmet,J.
Genes and people in the Caspian Littoral: A population genetic study in Northern Iran. Amer. J. Phys. Anthrop. 46, 3: 377-390

321- Kirk, R.L., Kinns,H., and Morton,N.E. 1970
Interaction between the ABO blood group and haptoglobin systems. Amer.J.Hum.Genet. 22:384-389

322- Kirk, R.L., and Lai, L.Y.C. 1961
The distribution of haptoglobin and transferrin groups in south and south east Asia. Acta.Genet. 11, 2: 97-105

323- Kitchin,F.D., and Bearn,A.G. 1964
Distribution of the serum group-specific components( Gc) in Afghanistan, Korean,Nigerian and Isreael populations.Nature,Lond. 202: 827-828

324- Kitchin, F.D., and Bearn,A.G. 1966
The electrophoretic patterns of normal and variant phenotypes of the group specific (Gc) components in human serum. Amer.J.Hum.Genet.18:201-214

325- Kitchin,F.D., Bearn,A.G., Alpers,M., and Gajdusek, D.C. 1972
Genetic studies in relation to Kuru.III.Distribution of the inherited serum grou-specific protein (Gc) phenotypes in New Guineans: An association of Kuru and the Gc Ab phenotype.Amer. J.Hum.Genet. 24: 72

326- Kitchin,F.D., Howel-Evans,W., Clarke,C.A., McConnell, R.B., and Sheppard, P.M. 1959
PTC taste response and thyroid disease.Brit. Med.J. i: 1069-1074

327- Klein,D., and FrancesChetti,A. 1964
Missbildungen und Krankheiten des Auges.Humangenetik.B.IV. edited by P.E. Becker.Georg thieme verlag. Stuttgart.

328- Klemperer,M.R., Woodsworth, H.C., Rosen,F.S., and Austen, K.F. 1966
Hereditary deficiency of the second component of human complement (C2) in man.J.Clin.Invest.45:880

329- Kompf,J., and Bissbort,S. 1974
The polymorphimsm of alanine aminotransferase (E C: 2.6.1.2): Spectrophotometrical assay of GPT. Humangenetik. 22: 251-253

330- Kompf, J., and Ritter, H. 1979
Polymorphism of Alanine Aminotransferase (E.C. 2.6.1.2): Common and Rare Alleles.Hum.Genet. 51: 287-292

331- Koster,B., Leupold,H., and Mauff,G. 1975
Esterase D polymorphism: high voltage agarose-gel electrophoresis and distribution of phenotypes in different European populations.Humangenetik. 28: 75-78

332- Kraus,A.P., and Neely, C.L. 1964
Human erythrocyte lactate dehydrogenase:four genetically determined variants.Science. 145: 595-597

333- Krill, A.E., Bowman, J.E., and Schneiderman,A. 1966
An investigation of so-called x-linked errors on the Ishihara plates. Ann.Hum.Genet. 29: 253

334- Kueppers, F., and Bearn,A.G. 1966
Inherited variations of human serum of $\alpha_1$-antitrypsin. Science. 154: 407

335- Kueppers, F., Briscoe,W.A., and Bearn,A.G. 1964
Hereditary deficiency of serum $\alpha_1$-antitrypsin. Science. 146: 1678

336- Kueppers,F., and Christopherson,M.J. 1978
Alpha-1-antitrypsin: Further genetic heterogeneity revealed by isoelectric focusing.Amer. J. Hum. Genet. 30: 359-365

337- Kuhnl,P., and Spielmann, W. 1978
Transferrin: Evidence for two common subtypes of the $Tf^C$ allele. Hum.Genet. 43: 91-95

338- Kuhnl, P., and Spielmann, W. 1979
A third common allele in the transferrin system, $Tf^{C3}$, detected by isoelectric focusing.Hum.Genet.

50: 193-198

339-Kuhnl, P., Spielmann,W., and Weber, W. 1979
Isoelectric focusing of rare transferrin(Tf) variants and common $Tf^C$ subtypes. Hum.Genet.46: 83-87.

340- Lachmann, P.J. 1973
In: Defense and Recognition, edited by R.R. Porter. London: Butterworths. P. 361

341- Lachmann, P.J., and Coombs,R.R.A. 1965
Complement, conglutinin and immunoconglutinins. Ciba found. Symp. Complement. 242

342- Lahav, M., and Szeinberg, A. 1972
Red-cell glutamic-pyruvic transaminase polymorphism in several population groups in Israel.Hum. Hered. 22: 533-538

343- Lai, L.Y.C., Nevo, S., and Steinberg,A.G. 1964
Acid phosphatases of human red cell: Predicted phenotype conforms to a genetic hypothesis. Science. 145: 1187

344- Lamm, L.U. 1970
Family studies of red cell acid phosphatase types. Report of a family with the D variant. Hum. Hered. 20: 329

345- Landsteiner, K. 1900
Zur Kenntnis der antifermentativen Iytischen und agglutinierenden Wirkungen des Blutserums und der lympha. Zbl. Bakt. 27,357.Cited in Race and Sanger. 1970

346- Landsteiner, K. 1901
Uber agglutination ser scheinungen normalen menschlichen Blutes. Wien Klin. Wschr. 14, 1132. Cited in Race and Sanger. 1970

347- Landsteiner, K., and Levine, P. 1927a
A new agglutinable factor differentiating individual human bloods. Proc.Soc.Exp.Biol.N.Y. 24: 600-602

348- Landsteiner, K., and Levine, P. 1927b
Further observations on individual differences of human blood. Proc. Soc. Exp.Biol.N.Y. 24: 941

349- Landsteiner, K., and Levine, P. 1928a
On the inheritance of agglutinogens of human blood demonstrable by immune agglutinins.J.Exp. Med. 48: 731-749

350- Landsteiner, K., and Levine, P. 1928b
On individual differences in human blood.J. Exp. Med. 47: 757

351- Landsteiner, K., and Levine, P. 1929a
On isoagglutinin reactions of human blood other than those defining the blood groups.J. Immunol. 17: 1-28

352- Landsteiner, K., and Levine, P. 1929b
On the racial distribution of some agglutinable structures of human blood. J.Immunol. 16: 123

353- Landsteiner, K., and Levine, P. 1931
The differentiation of a type of human blood by means of normal animal serum. J.Immunol. 20:179

354- Landsteiner,K., Strutton,W.R., and Chase,M.W. 1934
An agglutination reaction observed with some human bloods, chiefly among Negroes.J.Immunol. 27: 469-472

355- Landsteiner, K., and Wiener, A.S. 1940
An agglutinable factor in human blood recognised by immune sera for Rhesus blood.Proc.Soc. Exper.Biol. and Med. 43: 223

356- Lauer, V.V. 1929
Studium der isoagglutination seigenschaften des Blutes bei der gesunden und an Kropfleidenden BevolKerung des Karatschaj Autonomgebietes.(In.Russian; German Summary). Proc. N. Cauc.Ass.Sci.Res. Inst. 3: 121-133

357- Laurel, C.B., and Eriksson,S. 1963
The electrophoretic alpha$_1$-globulin patterns of serum in Alpha$_1$-antitrypsin deficiency. Scand. J.Clin. Lab. Invest. 15: 132-140

358- Laurell, C.B., Laurell,S., and Skoog, N. 1956
Buffer composition in paper electrophoresis. Considerations on its influence, with special reference to the interaction between small ions and proteins. Clin.Chem. 2: 99-111

359- Laurell, C.B., and Lundh, B. 1967
Electrophoretic studies of the conversion pro-

ducts of serum $\gamma_1$c-globulin. Immunology.
12:313

360-Layrisse, M., Arends, T., and Dominguez Sisco, 1955
R.
Nuevo grupo sanguineo encontrado en descendientes de Indios. Acta Medica Venez. 3: 132-138.
Cited in Race, R.R., and Sanger, R.(1970).

361- Layrisse, M., and Wilbert, J. 1960
El Antigen del Sistema Sanguineo Diego,pp.160,
La Fundacion Creole y la Fundacion Eugenio Mendoza, Caracas. Cited in Race and Sanger. 1970

362- Leakey, T.E.B., Coward,A.R., Warlow,A., and 1972
Mourant, A.E.
The distribution in human populations of electrophoretic variants of cytoplasmic malate dehydrogenase Hum.Hered. 22: 542-551

363- Leddy,J.P., Frank, M.M., Gaither,T., Baum,J., 1974
and Klemperer, M.R.
Heredity deficiency of the sixth component of complement in man. 1. Immunochemical, biological and family studies. J. Clin.Invest. 53: 544

364- Lehmann,H.,Ala,F., Hedayat,S.,Montazemi,K., 1973
Karimi-Nejad,H., Lightman,S., Kopec,A.C., Mourant, A.E., Teesdale,P., and Tills,D.
The hereditary blood factors of the Kurds of Iran. Phil.Trans.R.Soc. Lond. 266:195-205

365- Lehmann,H., and Ryan, E. 1956
The familial incidence of low pseudocholinesterase level. Lanct. ii: 124

366- Lehrs,H. 1930

Uber gruppenspezifische Eigenshaften des menschlichen speichels. Z. Immun.Forsch. 66: 175-192

367- Levence, C. 1968

Personal communication. Cited in Mourant et al. 1976

368- Levine,P., Backer, M., Wigod,M., and Ponder, R., 1949

A new human hereditary blood property (Cellano) Present in 99.8% of all bloods.Science. 109: 464-466

369- Levine, P., Bobitt, O.B., Waller, R.K., and Kuhmichel, A. 1951

Isoimmunization by a new blood factor in tumor cells. Proc. Soc.Exp. Biol., N.Y. 77: 403

*

370- Levine, P., and Stetson, R.E. 1939

An unusual case of intragroup agglutination. J. Amer. Med. Ass. 113: 126

371- Levy, B. 1948

The Universal Jewish Encyclopedia, 8. New York.

372- Lewis, Marion., Chown,B., Kaita, Kiroko,Hahn, D., Kangelos, M., Shepeard,W.L., and Shackelton K. 1967

Blood group antigen $Go^a$ and the Rh system.Transfusion,Philad. 7: 440-441

* For Levine et al (1973) see page 880

373- Lightman, S.L., Carr-Locke, D.L., and Pickles, 1970
Hilary G.
The frequency of PTC tasters and males defective in colour vision in a Kurdish population in Iran.
Hum.Biol. 42: 665-669

374- Livingstone. F.B. 1971
Malaria and Human polymorphisms. Annu.Rev. Genet. 5: 33-64

375- Lodhi, M.A.K. 1960
A study of blood groups in Multan(West Pakistan). Medicus, Karachi. 20, 2: 53-61

376- Loomis, W.F. 1967
Skin-pigment regulation of vitamin D biosynthesis in man. Science. 157: 501-506

377- Lopez, V., Oetliker, O., Colombo, J.P., and 1964
Butler, R.
Ein fall von familiarem $\alpha_1$-antitrypsin-Mangel.
Helv. Paediat. Acta. 19: 296

378- Luzatto, L., Usanga, E.A., and Reddy,S. 1969
Glucose-6-phosphate dehydrogenase deficient red cells: Resistance to infection by malarial parasites. Science. 164: 839-842

379- Mahallatee, E.A., and Haghshenas, M. 1969
Frequency and distribution of ABO and $Rh_o$(D) blood groups in Shiraz. Israel. J.Med. Sci. 5: 1081-1082

380- Maranjian,G., Ikin, Elizabeth W., Mourant,A. 1966
E., and Lehmann, H.
The blood groups and haemoglobins of the Saudi
Arabians. Hum. Biol. 38: 394-420

381- Marceau, N., and Sass-Kortsak,A. 1970
The in vivo transfer of Ceruloplasmin copper
to cytochrom oxidase .Canad.Fed.Biol.Soc.Abstr.
13: 127

382- Markert, C.L., and Moller,F. 1959
The multiple forms of enzymes:tissue,ontogene-
tic and species specific patterns. Proc.Natn.
Acad. Sci. Wash. 45: 753

383- Marks, M.P., Jenkins,T., and Nurse,G.T. 1977
The red cell glutamic-pyruvate transaminase,
carbonic anhydrase I and II and esterase D poly-
morphisms in the Ambo populations of South West
Africa with evidence for the existence of an $EsD^{o}$
allele. Hum.Genet. 37: 49-54

384- Martin, J.P., Sesboue, R., Charlionet,R., and 1975
Ropartz, C.
Does alpha-1-antitrypsin Pi null phenotype
exist. Hum. Genet. 30: 121-125

385- Martin,G., McAlister,R., Pelter,W., and Bendi- 1961
tt,E.P.
Heterogeneity of Ceruloplasmin.Proc.2nd Intern.
Congr. Hum. Genet. 2: 752

386- Martin,W., and Niebuhr,R. 1973
Polymorphismus der menschlichen erythrocyten-

glutamat-pyruvat-Transaminase. Humangenetik.19: 203-206

387- Marzban, M. 1978
Study of some blood group systems in Dasht-Mishan, Khuzistan. M.Sc. Thesis. University of Jundishapur. Ahwaz. Iran.

388-McAlpine,Phillis J., Chen,S.H., Cox,Diane W., 1974
Dossetor,J.B., Giblett, Eloise.,Steinberg,A.G., and Simpson, Nancy,E.
Genetic markers in blood in a Canadian Eskimo population with a comparison of allele frequencies in circumpolar populations.Hum.Hered.24:114-142

389-McAlpine,P.J., Hopkinson,D.A., and Harris H. 1970
The relative activities atrributable to the three phosphoglucomutase loci($PGM_1$,$PGM_2$,$PGM_3$) in human tissues. Ann.Hum.Genet. 34: 169-173

390-McAlpine,P.J.,Mohandas,T., and Hamerton,J.L. 1975
Isozyme analysis of somatic cell hybrids:Assignment of the phosphoglucomutase$_2$($PGM_2$) gene locus to chromosome 4 in man with data on the molecular structure and human chromosome assignments of six additional markers. In:Isozymes,IV,P.149,Genetics and evolution. New York: Academic Press.

391- McAlister,R., Martin,G.M., and Benditt,E.P. 1961
Evidence for multiple ceruloplasmin components in human serum. Nature, Lond. 190: 927

392- McCurdy, P.R., and Mahmood,L. 1970
Red cell glucose-6-phosphate dehydrogenase deficiency in Pakistan.J.Lab.Clin.Med. 76:943-948

393- McLean, R.H., and Hoefnagel,D. 1980
Partial Lipodystrophy and familial C3 deficiency. Hum.Hered. 30: 149-154

394- Metaxas-Buhler,M., Metaxas,M.N., and Giles,Car-1972
Olyn M.
A Swiss family showing inheritance of the Swann antigen with $Lu^a$. Vox Sang. 23: 429-432

395- Metaxas,M.N., and Metaxas-Buhler,M. 1964
$M^k$: an apparently silent allele at the MN locus . Nature, Lond. 202: 1123

396- Meuwissen,H.J., Pollara,B., and Pickering,R,J.1975
Combined immunodeficiency disease associated with adenosine deaminase deficiency.J.Pediat. 86: 169-181

397- Miller,L.H., Mason,S.J., Dvorak,J.A.,McGinniss,1975
M.H., and Rothman, I.K.
Erythrocyte receptors for (Plasmodium Knowlesi) malaria. Duffy blood group determinants.Science. 189: 561-563

398- Minorsky, V. 1945
The tribes of Western Iran. JIR.anthrop.Inst. 75: 73-80

399- Mitchell,R.J. 1972
A review of the literature concerning postulated associations between Phenylthiocarbamide (P.T.C.) tasting and clinical conditions with special reference to thyroid disease and glaucoma. Dyn. 2: 70

400- Mithal, Y., Lane, A.B., and Trefor Jenkins. 1980
Absence of red cell glutamic-pyruvate transaminase: Discovery of a "silent" allele Homozygote. Amer. J. Hum.Genet. 32: 42-46

401- Miyashita, T., Ohkura,K., Matsumoto,H.,and 1975
Matsumoto, K.
Distribution of polymorphic traits in Iran.
Jpn. J.Hum. Genet. 20:55

402- Mizan,N., Turan,-., and Alptekin,Ayten. 1963
The frequencies of ABO blood groups and their distribution according to the geographical regions in Turkey. Ankara,The Turkish Red Crescent Soc. 16 PP.

403- Mizan,N., Ozemek, C., and Alptekin, Ayten. 1965
MN blood group system in Turkey.(In Turkish).
Tip. Fak. Mecm. 18: 761-766

404- Mohr, J. 1951
Estimation of linkage between the Lutheran and Lewis blood groups. Acta.Path.Microbiol.Scand. 29: 339-344

405- Mollison, P.L. 1965
The role of complement in hemolytic processes in Vivo. Ciba found.Symp. Complement. 323

406- Montazami,K. 1978
Distribution of ABO groups in Iran.Iranian J. Publ. Health. 7, 1: 39-56

407- Morin,T., Martin, J.P., Feldmann,G.,Rueff,B., 1975
Benkamou, J.P., and Ropartz C.

Heterozygous alpha-1-antitrypsin deficiency and cirrhosis in adults, a fortuitous association. Lancet. i: 250-251

408- Morton, N.E. 1974
Kinship bioassay. Genetic distance, J.F.Crow and C. Denniston eds., Plenum Press,New York. 97-104

409- Motamed, M. 1949
Personal communication. Cited in Mourant. 1954

410- Moten, A.N., and Stewart, G.T. 1956
Blood groups of Muslims and Parsees in Pakistan. Brit. J. Haemat. 2: 61-64

411- Motulsky, A.G. 1960
Metabolic polymorphisms and the role of infectious disease in human evolution.Hum.Biol. 32:28-63

412- Motulsky,A.G. 1961
Glucose-6-phosphate dehydrogenase deficiency, hemolytic disease of the newborn and malaria. Lancet. I: 1168-1169

413- Motulsky,A.G. 1964
Hereditary red cell traits and malaria.Amer. J.Trop. Med. Hyg. 13: 147-158

414- Motulsky, A.G. 1965
Theoretical and clinical problems of glucose -6-phosphate dehydrogenase deficiency.In:Abnormal hemoglobins in Africa. Jonxis, J.H.P.(ed.).

Oxford: Blackwell Scientific.

415- Motulsky,A.G., and Campbell-Kraut,J.M. 1961
Population genetics of glucose-6-phosphate dehydrogenase deficiency of the red cell. In:Proceedings of the Conference on Genetic Polymorphisms and Geographic Variations in Disease.Blumberg, B.S.(ed.).New York: Grune and Stratton.

416- Motulsky,A.G., Vandepitte,J., and Fraser,G.R. 1966
Population genetic studies in the Congo:I.Glucose-6-phosphate dehydrogenase deficiency.Hemoglobin S, and malaria. Amer.J.Hum.Genet.18:514-537

417- Mourant, A.E. 1954
The distribution of the human blood groups.Blackwell Scientific Publ. Oxford.

418- Mourant,A.E., Kopec,A.C., and Domaniewska 1976
Sobczac, K.
The distribution of the human blood groups and other polymorphisms.(2nd edition).Oxford University Press. Oxford,New York and Toronto.

419- Mourant,A.E., Kopec,A.C., and Domaniewska 1978
Sobczac,K.
Blood groups and diseases. Oxford University Press. Oxford London - New York.

420- Mourant,A.E., Tills,D., and Domaniewska 1976
Sobczac, K.
Sun shine and the geographical distribution of the alleles of the Gc system of plasma proteins. Hum. Genet. 33: 307-314

421- Muller-Eberhard,H.J. 1968
Chemistry and reaction mechanisms of complement. Immunology. 8:1

422- Muller-Eberhard, H.J. 1975
Annu.Rev.Biochem. 44: 697-724

423- Muller-Eberhard, H.J., and Nilsson,U. 1960
Relation of a $\beta_1$-glycoprotein of human serum to the complement system. J.Exp.Med. 111: 217-234

424- Muller-Eberhard,H.J., Nilsson,U., and 1960
Aronsson,T.
Isolation and characterization of two $\beta_1$-glycoproteins of human serum.J.Exp.Med.111: 201-215

425- Muller-Eberhard, H.J., Nilsson,U.R.,Dalmasson, 1966
A.P., Polley,M.Jr., and Calcott,M.A.
A molecular concept of immune cytolisis.Arch. Pathol. 82: 205

426- Neel,J.V., and Post, R.H. 1963
"Transitory' positive' selection for colour-blindness?" Eugen. Quart. 10: 33-35

427- Nei,M. 1975
Molecular population genetics and evolution. North-Holland Publishing Co., Amsterdam.

428- Nei,M. 1978
The theory of genetic distance and evolution of human races.Jpn .J.Hum.Genet.23:341-369

429- Nelson,R.A. 1962

Immune-adherence, in Mechanisms of cell and Tissue Damage produced by immune reaction.(P. Grabar and P. Miescher, editors),Basel/Stuttgart, Schwabe. 245 1962

430-Neumann, S., and Walter, H. 1968
Frequencies of Pseudocholinesterase variants in Icelanders, Greeks and Pakistanis.Nature, Lond. 219: 950

431-Nezbeda, P. 1979
Occurrence of the $ACP_1^0$ allele in Czechoslovakia. Hum.Genet. 46: 227-229

432- Nguyen,V.C., Billardon,C.,Picard,J.Y., Feingold, J., and Frezal,J. 1971
Liaison probable(linkage) entre les locus $PGM_1$ et peptidase C chez l'homme.C.R. Acad.Sci.(D) Paris. 272: 485

433-Nijenhuis, L.E. 1964
Blood group frequencies in Iran. Vox Sang. 9: 723-740

434-Nikitin,B. 1943
Les Kurdes: etude sociologique et historique (V). Paris: Imprimerie Nationale,Klincksieck.

435-Nishioka, K., and Linscott,W.D. 1963
Components of guinea pig complement.1.separation of a serum fraction essential for immune hemolysis and immune adherence.J.Exp.Med. 118:767

436-Olaisen, B. 1973a
Atypical segregation of erythrocyte glutamic-

pyruvic transaminase in a Norwegian family.
Hum.Hered. 23: 595-602

437- Olaisen,B. 1973b
Two rare GPT phenotypes in a Norwegian family, evidence of a seventh allele.Humangenetik.19: 289-291

438- Olaisen,B.,Teisberg,P.,and Jonassen,R. 1976
EsD polymorphism in Norway.Hum.Genet.34:63-64

439- Olmstead,A.T. 1948
History of the Persian Empire.The University of Chicago Press, Chicago, Illinois.

440- Onsi,A.A., Eid,H., and Hovagiumian,H. 1969
The Rh blood groups in Kuwait.J.Kuwait Med. Ass. 3: 83-86

441- Onsi,A.A., and El-Alfi,D.C. 1968
The ABO blood groups in Kuwait.J.Kuwait Med. Ass. 2: 3-10

442- Paidoussis, M. 1967
Personal communication. Cited in Mourant et al. 1976

443- Palmarino,R., Agostino,R., Gloria,F.,Lucarel- 1975
li, P., Businco,L., Antonioni,G.,Maggioni.G., Workman,L., and Bottini,E.
Red cell acid phosphatase:another polymorphism correlated with malaria?Amer.J.Phys.Anthrop.43: 177-185

444- Papiha,S.S. 1979

Complement variation and Anthropology.Man.14: 549

445- Papiha,S.S., and Al-Agidi,S.K. 1976
Esterase D and superoxide dismutase polymorphisms in Iraq.Hum.Hered.26, 5: 394-400

446- Papiha,S.S., Bernal,J.E., and Malhotra,M. 1979
Qualitative and quantitative serum protein variation in Chattisghari,Brahmins of Madhya Pradesh, India. Hum.Genet.Submited 1979

447- Papiha,S.S., Bernal,J.E., Roberts,D.F., 1979
Habeebullah, C.M., and Mishra,S.C.
C3 polymorphism in some Indian populations. Hum.Hered. 29: 193-196

448- Papiha,S.S., and Nahar,A. 1977
The world distribution of the electrophoretic variants of the red cell enzyme esterase D.Hum. Hered. 27: 424-432

449- Papiha,S.S., Roberts,D.F., and Rahimi,A.G. 1977
Genetic polymorphisms in Afghanistan.Ann.Hum. Biol. 4, 3: 233-241

450- Papiha,S.S., Seyedna,Y., and Sunderland,E. 1982
Phosphoglucomutase(PGM) and group specific component(Gc), isoelectricfocusing subtypes among Zoroastrians of Iran.Ann.Hum.Biol.9,6:571-574

451- Parr.C.W. 1966
Erythrocyte phosphogluconate dehydrogenase polymorphism. Nature. 210: 487-489

452- Parr,C.W., and Fitch,L.I. 1964

Hereditary partial deficiency of human-erythrocyte phosphogluconate dehydrogenase. Biochem.J. 93: 28-30

453- Parr,C.W., and Fitch,L.I. 1967
Inherited quantitative variations of human phosphogluconate dehydrogenase.Ann.Hum.Genet. 30: 339-353

454- Parr,C.W., and Parr,I.B. 1965
Stability differences of inherited variants of human red cell phosphogluconate dehydrogenase. Biochem. J. 95: 160

455- Pauling,L., Itano,H., Singer, S.J., and Wells,I.C. 1949
Sickle cell anaemia, a molecular disease.Science. 110: 543-548

456- Peterson,B.H., Graham,J.A., and Brooks,G.F. 1975
Human deficiency of the 8th component of complement: Lack of bactericidal activity.Clin.Res. 23: 295 A.

457- Pickford,R.W. 1956
Colour blindness and its inheritance.Biology and Human Affairs. 21: 19

458- Pinkerton,F.J., Mermod,L.E., Liles,Bobbie Ann, 1959
Jack, J.A., and Noades, Jean.
The phenotype JK(a-b-) in the Kidd blood group system. Vox Snag. 4: 155

459- Plaut, G., Ikin,E.W., Mourant,A.E.,Sanger,R., 1953

and Race, R.R.

A new blood group antibody,anti-$JK^b$.Nature, Lond. 171: 431

460- Polonovski, M., and Jayle, M.F. 1938

Existence dans le plasma Sanguin d'une substance activant l'action peroxydasique de l'hemoglobine. C.r.Soc.Biol. 129: 457

461- Polonovski,M., and Jayle, M.F. 1940

Sur la preparation d'une nouvelle fraction des proteines plasmatiques, l'haptoglobine.C.R.Acad. Sci. Paris. 211: 517-519

462-Post, R.H. 1962

Population differences in red and green colour vision deficiency: A review, and a query on selection relaxation.Eugen.Quart. 9: 131-146

463- Poulik,M.D. 1957

Starch-gel electrophoresis in a discontinuous system of buffers. Nature, Lond. 180: 1477

464- Putkonen,T. 1930

Uber die gruppenspezifischen Eigenschaften verschiedener Korperflussigkeiten Acta Soc. Med. 'Duodecim'. Ser.A. 14, 12: 1-113

465- Quick,C.B., Fisher,R.A., and Harris,H. 1972

Differentiation of the $PGM_2$ locus isozymes from those of $PGM_1$ and $PGM_3$ in terms of phosphopentomutase activity. Ann.Hum.Genet. 35: 445-454

466- Quick,C.B., Fisher,R.A., and Harris,H. 1974
A Kinetic study of the isozymes determined by the three human phosphoglucomutase loci $PGM_1$, $PGM_2$, and $PGM_3$. Eur.J. Biochem. 42:511-517

467- Race, R.R., and Sanger, R. 1950
Blood groups in Man.1st edn.Blackwell Scientific Public. Oxford.

468- Race, R.R., and Sanger, R. 1954
Blood groups in man. 2nd edn.Blackwell Scientific Public. Oxford.

469- Race, R.R., and Sanger, R. 1962
Blodd groups in Man. 4th edn.Blackwell Scientific Public. Oxford.

470- Race, R.R., and Sanger, R. 1968
Blood groups in man.5th edn.Blackwell Scientific Public. Oxfor. XXViii, 599.pp.

471- Race, R.R., and Sanger, R. 1970
Blood groups in man.5th edn.Blackwell Scientific Public. Oxford.

472- Race,R.R., Sanger,R., Allen,R.H.,Diamond, 1951
L.K., and Niedziela, B.
Inheritance of the human blood group antigen $JK^a$. Nature, Lond. 168:207

473- Race, R.R., Sanger, R., Lawber,Sylvia D., and 1949
Bertinshaw, Doreen.
The inheritance of the MNS blood groups: a second series of families. Heredity. 3: 205-213

474- Radam,R., Strauch,H., und Prokop, O. 1974
Ein seltener phanotype im Adenosindesaminase polymorphismus: Hinweis auf die existenz eines neuen allels. Humangenetik. 25: 247-250

475- Rahimi,A.G., Goedde,H.W., Flatz,G.,Kaifie,S., 1977
Benkmann,H.G., and Delbruck, H.
Serum protein polymorphisms in four populations of Afghanistan.Amer.J.Hum.Genet. 29: 356-360

476- Ramot, Bracha., Duvdevani-Zikert,Poriya.,and 1961
Tauman, Gisela.
Distribution of Haptoglobin types in Israel.
Nature, Lond. 192: 765-788

477- Ramot, Bracha., Duvdevani-Zikert,Poriya.,and 1962
Kende,G.
Haptoglobin and transferrin types in Israel.Ann. Hum.Genet. 25: 267-271

478- Rapley,S., Robson,E.B., Harris,H., and Smith, 1967
S.M.
Data on the incidence, segregation and linkage relations of the adenylate Kinase(AK)polymorphi-sm.Ann.Hum.Genet. 31: 237-242

479- Renninger, W. 1969
Izozymmuster der phosphoglucomutase der mens-chlichen Thrombocyten(Thr.$PGM_1$).Humangenetik. 8: 255-257

480- Richard,P. 1976
Hemotypologie des Kurdes. Doctorate thesis, Toulouse. France. 85 PP.

481- Rimon,A., Shamash, Y., and Shapiro,B. 1966
The plasmin inhibitor of human plasma.J.Biol. Chem. 241: 5102

482- Ritter, H., and Hinkelmann, K. 1966
Zur balance des polymorphismus der Haptoglobine. Humangenetik. 2: 21-24

483- Rittner, C., and Muller, G. 1975
Esterase D. Some population and formal genetical data. Hum.Hered. 25: 152-155

484- Rittner, C., and Rittner, B. 1974
Report 1973/74 of the reference Laboratory for the polymorphism of the third component(C3) of the human complement system.Vox Sng.27: 464-472

485- Roberts,D.F., and Al-Agidi,S.K. 1979
The $C_3$ component of complement: gene frequency variation in Iraq.Amer.J.Phys.Anthrop. 50, 4 : 511-514

486- Robson,Elizabeth B., and Harris, H. 1966
Further data on the incidence and genetics of the serum cholinesterase phenotype $C_5$+. Ann. Hum. Genet. 29: 403-408

487- Ronald, A.R., Underwood,B.A., and 1968
Woodward, T.E.
Glucose-6-phosphate dehydrogenase deficiency in Pakistani males. Trans.R.Soc.Trop.Med.Hyg. 62: 531-533

488- Ropartz, C., Rivat, L., Rousseau,P.Y.,Debeaus, 1965
P.,et Steinbuch, M.

un nouveau polymorphisme serique humain defini par l'utilisation de globules rouges tannes et revetus de beta-IC; in Hollander proc.10th.Congr. Int. Soc. Blood Transf.,Stockholm.P. 383-384 (Karger, Basel).

489- Rosenfeld, S.I., Kelly,M.E., and Leddy,J.P. 1976
Hereditary deficiency of the 5th component of complement in man. 1. Clinical,immunochemical and family studies. J. Clin.Invest. 57: 1226

490- Roshan,G., Shams-Ara., and Esteghamat. 1969
Study of blood groups and Rh factor.(Firouz-gar Medical Centre). J.Mashad.Med.School.12, 4: 488-498

491- Saatcioglu, A. 1979
An analysis of the ABO gene frequencies in Turkey.J.Hum.Evolut.8, 3: 367-373

492- Sadatzadeh,H., and Hamidi,A.H. 1972
Structure of blood group antigens and their distribution in Iran. J. Iranian Medical Council. 2, 6: 444-450

493- Saha, N., Bayoumi, R.A., Elsheikh,F.S.,Samuel, 1980
A.P.W., Elfadil, I., Elhouri,I.S., Sebai,Z.A., and Sabaa, H.M.A.
Some blood genetic markers of selected tribes in Western Saudi Arabia. Amer.J.Phys.Anthrop.52, 4: 595-600

494- Salzano, F.M. 1972
Visual acuity and colour blindness among Bra-

zilian Cayapo. Indians. Hum. Hered. 22: 72

495- Samin, P. 1965

Personal communication. Cited in Mourant et al. 1976.

496- Sanger, R. 1955

An association between the P and Jay Systems of blood groups. Nature, Lond. 176: 1163

497- Sanger, R., and Race, R.R. 1947

Sub-division of MN blood groups in man.Nature, Lond. 160: 505-506

498- Sanger,R., Race, R.R., and Jack,J. 1955

The Duffy blood groups of the New York Negroes: the phenotypes FY(a-b-).Brit.J.Haemat. 1: 370-374

499- Sanghvi, L.D., and Balakrishnan, V. 1972

Comparison of different measures of genetic distance between human populations.In the Wiener, J.S., and Huizinga, J.(edt.).The assessment of population affinities in man.Clarendon Press, Oxford.

500- Santachiara Benercetti, A.S., Beretta,M., and Pampiglione, S. 1975

Red cell glutamic-pyruvic transaminase polymorphism in a sample of the Italian population.A new variant allele: $GPT^3$. Hum.Hered. 25:276-278

501- Sawhney,K.S. 1975

Genetic polymorphisms in selected populations

in south and south east Asia. Ph.D.Thesis.
University of Durham, Durham. U.K.

502- Sawhney,K.S., and Farhud, D.D. 1979
First report of Lutheran blood group system in Iranian Moslems. Iranian J.Publ.Hlth. 8,2: 67-70

503- Sawhney,K.S., Sunderland,E., and Farhud,D.D. 1981
Study of Red cell enzyme systems in Tehran and Isfahan Iranians.Jpn.J.Hum .Genet. 26:289-294

504- Say,B., Kiran,O., Altay,C., and Berkel,I. 1966
The incidence of PTC taste sensitivity in the population of Ankara.Turk.J.Pediat. 8: 171-175

505- Say,B.,Ozand,P., Berkel,I., and Cevik,N. 1965
Erythrocytose glucose-6-phosphate dehydrogenase deficiency in Turkey. Acta Paediat.Scand.54: 319-324

506- Sayek,I., Karahasanoglu, A.M., and Ozand,P. 1967
Pseudocholinesterases iii. The presence of Pseudocholinesterase variants in a Turkish population. Turk.J. Pediat. 9: 8-12

507- Schanfield,M.S., Giles, E., and Gerschowitz, 1975
H.
Genetic studies in the Markham valley, north-eastern Papua. New Guinea: gamma globulin(Gm and Inv), group specific component(Gc) and ceruloplasmin(CP) typing.Amer.J.Phys.Anthrop. 42:1-7

508- Schiff, F., and Sasaki, H. 1932
Der Ausscheidungstypus, ein auf serologischem wege nachweisbares mendelndes merkmal.Klin.Ws-

chr. 11: 1426-1429

509- Schiotz, P.O., Hoiby,N., Morling,N., and Soren- 1978
sen, H.
C3 polymorphism in a Danish cystic Fibrosis population and its possible association with antibody response. Hum.Hered. 28: 292-300

510- Schultze, H.E., Heide,K., and Haupt,H. 1962
$\alpha_1$-antitrypsin aus Humanserum.Klin.Wschr. 40: 427

511- Schultze, H.E., and Heremans, J.F. 1966
Molecular biology of human proteins.Vol.1. Amsterdam-London-New York: Elsevier Publ.

512- Scott, E.M., Duncan,I.W., Ekstrand,V., and 1966
wright, R.C.
Frequency of polymorphic types of red cell enzymes and serum factors in Alaskan Eskimos and Indians. Amer.J.Hum.Genet. 18:408

513- Seth, P.K., and Seth, S. 1976
Human complementary component C3.Hum.Genet. 33: 201-211

514- Shaker, Yehia., Onsi,A., and Aziz,R. 1966
The frequency of glucose-6-phosphate dehydrogenase deficiency in the new-borns and adults in Kuwait.Amer.J.Hum.Genet. 18,6:609-613

515- Shapiro, M. 1956
Inheritance of the Henshaw(He) blood factor. J. forensic Med. 3: 152-160

516- Shargo, E. 1965
Cytoplasmic characteristics of human erythrocytic malic dehydrogenase. Archs.Biochem.Biophys. 109: 57

517- Sharp. H. 1970
Alpha$_1$-antitrypsin deficiency.Hosp.Pract. 6: 83-96

518- Sheba,C., Ashkenazi,I., and Szeinberg,A. 1962
Taste sensitivity to phenylthiourea among the Jewish population groups in Israel.Amer. J.Hum.Genet. 14: 44-51

519- Sheba,C., Szeinberg,A., Adam,A., and Ramot, 1961
Bracha.
Distribution of glucose-6-phosphate dehydrogenase deficiency (G6PD) among various communities in Israel. 2nd Int.Congr.Hum.Genet.,Rome. 633-634

520- Shih,L.Y., and Hsia,D.Y.Y. 1969
The distribution of genetic polymorphisms among Chinese in Taiwan.Hum.Hered. 19:227-233

521- Shim, B.S., and Bearn,A.G. 1964
The distribution of haptoglobin subtypes in various populations, including subtype patterns in some nonhuman primates.Amer.J.Hum.Genet.16: 477-483

522- Shokeir, M.H.K., Rucknagel,D.L., Shreffler,D.C.,1968
Na-Nakorn,S., and Wasi,P.
Genetic polymorphism of human serum ceruloplas-

min in Thailand.Amer.Soc.Hum.Genet.Abstr.
21: 68

523- Shokeir,M.H.K., and Shreffler,D.C. 1969
Cytochrom oxidase deficiency in Wilson's
disease:
a suggested ceruloplasmin function.Proc.Nat.
Acad.Sci.(Wash.) 62: 867-872

524- Shokeir, M.H.K., and Shreffler D.C. 1973
Two new ceruloplasmin variants in Negroes-
Data on three populations.Human biochemical
genetics - MSS Information Corporation.New York.

525- Shokeir,M.H., Shreffler,D.C., and Gall.J.C. 1967
Further electrophoretic variation in human ce-
ruloplasmin. Meet.Amer.Soc.Hum.Genet.Toronto.
Quoted by Giblett, 1969

526- Shreffler, D.C., Brewer,G.J., Gall,J.C., and 1967
Honeyman, M.S.
Electrophoretic variation in human serum ceru-
loplasmin: a new genetic polymorphism.Biochem.
Genet. 1: 101-115

527- Silberstein,W., and Goldstein,N. 1958
Blood groups in women of various oriental Jew-
ish communities(In Hebrew) Harefuah. 54:295-296

528- Simhai, B. 1974
Enzymatic Genetic polymorphisms among Persian
Jews in Israel.M.Sc.Thesis. The Hebrew Univer-
sity of Jerusalem. Israel.

529- Simhai, B. 1976

Haptoglobin polymorphism among the Iranian Jews.

7th Pahlavi Medical Conference.Shiraz. Iran.

530- Simhai, B. 1978

Enzyme polymorphisms in Iranian Armenians.

7th National Genetics Congress. Jundishapur University,Ahwaz.Iran.

531- Singh,S., Amma,M.K.P., Sareen,K.N., and Goedde, H.W. 1971

A study of the Pseudocholinesterase polymorphism among a Punjabi population.Hum.Hered. 21: 388

532- Sirsat, Satyavati M. 1956

Effect of migration on some genetical characters in six endogamous groups in India.Ann. Hum.Genet. 21: 145-154

533- Smith, C.A.B. 1977

A note on genetic distance. Ann.Hum.Genet. 40: 463

534- Smithies, O. 1955

Zone Electrophoresis in Starch gel: group variations in the serum proteins of normal human adults. Biochem.J. 61: 629

535- Smithies, O. 1957

Variations in human serum $\beta$ -globulins. Nature, Lond. 180: 1482

536- Smithies,O., Connell,G.E., and Dixon,G.H. 1962a

Inheritance of haptoglobin subtypes.Amer.J.Hum. Genet. 14: 14

537- Smithies,O., Connell, G.E., and Dixon,G.H. 1962b
Chromosomal rearrangements and the evolution of haptoglobin genes.Nature,Lond. 196:232-236

538- Smithies.O., and Hiller, O. 1959
The genetic control of transferrin in humans. Biochem.J. 72: 121

539- Smithies, O., and Walker, N.F. 1955a
Genetic control of some serum proteins in normal humans. Nature, Lond. 176: 1265

540- Smithies, O., and Walker, N.F. 1955b
Genetic control of some serum protein groups and the gene controlling their inheritance. Nature, Lond. 178: 694

541- Smithies,O., and Walker N.F. 1956
Notation for serum protein groups and the genes controlling their iheritance. Nature,Lond. 178: 694

542- Snyder, L.H. 1932
Studies in human inheritance.ix.The inheritance of taste deficiency in man. Ohio.J.Sci. 32: 436-440

543- Solovyeva, T.G. 1967
Distribution of blood groups, MN and Rh-factors among the Georgians of the Gori Region.(In.Russian). Vop.Antrop. 25: 70-71

544- Spedini, G. 1969
Frequenza dei tipi 'Hp' e ' Gc' in un Campione de popolazione dello Swat(Pakistan occidentale): Riv. Antrop. 56: 117-134

545- Spedini,G., Rickards, O., Bonomi,P., D'Agosti-1978 no,G., and Fuciarelli, M.
I marcatori genetici nei programmi biologici per 10 studio della popolazione. Risultati di un' indagine sui Tofinu, pescatori lacustri dell'Africa.

546- Spencer, N., Hopkinson,D.A., and Harris,H. 1964a
Quantitative differences and gene dosage in the human red cell acid phosphatase polymorphism. Nature. 201: 299-300

547- Spencer,N., Hopkinson,D.A., and Harris,H. 1964b
Phosphoglucomutase polymorphism in man.Nature., Lond. 204: 742

548- Spencer,N., Hopkinson,D.A., and Harris,H. 1968
Adenosine deaminase polymorphism in man.Ann. Hum.Genet. Lond. 32: 9-14

549- Spielmann, W.,Kuhnl,P., Rexrodt,Ch.,and 1973 Hansel, G.
Untersuchungen zum GPT-system unter besonderer berucksichtigung des stummen allele $GPT^{o}$.Human-genetik. 18: 341-348

550- Spuhler, J.N. 1954
Some problems in the physical anthropology of the American South West.Amer.Anthrop.56:604

551- Stern,M.A., Kynoch, Pamela A.M., and Lehmann, 1968
H.
β -thalassaemia, glucose-6-phosphate dehydrogenase deficiency, and haemoglobin D-Punjab in Pathans. Lancet. i: 1284-1285

552- Stratton, F. 1946
A new Rh allelomorph. Nature, Lond.158:25

553- Stroup,M., Macllory,M., Walker,R., and Ayde 1965
lotte, J.V.
Evidence that Sutter belongs to the Kell blood group system. Transfusion, Philad. 5: 309

554- Stoffersen,Erik., and Jorgensen Kaj Anker. 1980
C3 polymorphism in patients with chronic uremia. Hum. Hered. 30: 46-49

555- Sunderland, E. 1966
The tasting of phenylthiocarbamide(PTC) in selected populations in the United Kingdom. Eugen. Rev. 58: 143

556- Sunderland, E. 1968
Early man in Iran. In: The Cambridge History of Iran. Vol. 1. The Land of Iran.W.B. Fisher, ed. Cambridge University Press, Cmabridge,PP. 395-408

557- Sunderland,E. 1973
Elements of human and social geography.Pergamon Press, Oxford.

558- Sunderland,E., and Smith, H.M. 1966
The blood groups of the shi'a in Yazd,Central

Iran.Hum.Biol. 38: 50-59

559- Swick, R.B., Barnstein,P.L., and Stange,J.L. 1965
Distribution and characteristics of the isozymes of alanine aminotransferase in rate liver.J. Biol. Chem. 240:3334-3340

560- Szeinberg, A. 1963
G6PD deficiency among Jews-genetic and anthropological considerations, In: The genetics of migrant and isolate populations.ed.by Elizabeth Goldschmidt. 69-72

561- Szeinberg, A., Pipano,Serena., and Ostfelde,E. 1966
Frequency of atypical Pseudocholinesterase in different population groups in Israel. 2nd.Congr. Europ. Anaesth., Kopenhagen.Acta anaesth,Suppl. 24: 199-205

562- Szeinberg,A., and Tomashevsky-Tamir,S. 1971
Red cell adenylate Kinase and phosphoglucomutase polymorphism in several population groups in Israel. Hum.Hered. 21: 289

563- Tabatabai, H. 1977
A comparative genetical investigation of the Iranian Armenians and Jews. M.Sc.Thesis.University of Tehran. Tehran.Iran.

564- Tahmasbian, A. 1972
Personal report from blood transfusion centre of Shiraz. Cited in: Sadatzadeh and Hamidi.1972

565- Talamo,R.C., Blennerhassett,J.B., and Austen, 1966
F.

Familial emphysema and alpha 1-antitrypsin deficiency. New England. J.Med. 275: 1301

566- Tashian, R.E. 1961
Multiple forms of esterases from human erythrocytes. Proc.Soc.Exp.Biol.(N.Y.)108:364

567- Tashian,R.E. 1969
The esterases and carbonic anhydrases in human erythrocytes. In:Biochemical methods in red cell genetics(ed.J.J.Yunis).PP.307-336. New York-London: Academic Press.

568- Tashian,R.E., Brewer,G.J., Lehmann,H.,Davies, 1967
D.A., and Rucknagel, D.L.
Further studies on the xavante Indians.V.Genetic variability in some serum and erythrocyte enzymes, hemoglobin, and the urinary excretion of β -amino-isobutyric acid.Amer. J. Hum. Genet. 19: 524

569- Tehranchian,-. 1963
Personal report from blood transfusion centre and immunological section of Medical School of Tehran University.Cited in Sadatzadeh and Hamidi. 1972

570- Teisberg, P. 1970
High voltage agarose gel electrophoresis in the study of C3 polymorphism. Vox Sang. 19:47-56

571- Teisberg,P. 1971
The distribution of C3 types in Norway.Hum. Hered. 21: 154-161

572- Thompson,P.R., Childers,D., and Hatcher,D.E. 1967
Anti-$Di^b$: first and second examples .Vox Sang. 13: 314-318

573- Thomsen,O., Friendenreich,V., and Worsaae,E. 1930
Uber die moglichkeit der existenz zweier neuer Blutgruppen; auch eim Beitrag zur Belenchtung sogennanter untergruppen.Acta Path.microbiol. Scand. 7:157. Cited in Race and Sanger. 1970

574- Thymann,M. 1978
Indentification of a new serum protein polymorphism as transferrin. Hum.Genet. 43:225-229

575- Tills,D., and Lehmann,H. 1975
Unpublished observations.Cited in Mourant et al. 1978

576- Tills,D., Van den Branden,J.L., Clements,V.R.,1970
and Mourant, A.E.
The distribution in man of genetic variants of 6-phosphogluconate dehydrogenase.Hum.Hered. 20: 523-529

577- Tills,D., Van den Branden,J.L., Clements,V.R.,1971
and Mourant,A.E.
The distribution in man of genetic variants of 6-phosphogluconate dehydrogenase.Hum.Hered. 21: 305-308

578- Tills,D., Branden, Janet L.Van den, 1971
Clements, Vivien R., and Mourant,A.E.
The world distribution of electrophoretic variants of the red cell enzyme adenylate Kinase. (ATP: AMP Phosphotransferase)EC 2.7.4.3. Hum. Hered. 21: 302-304

579- Tills,D., Warlow,A., Mourant,A.E., Kopec,A.C.,1977
Edholm,O.G., and Garradr,G.
The blood groups and other hereditary blood factors of Yemenite and Kurdish Jews.Ann.Hum. Biol. 4, 3: 259-274

580- Tippett, Patricia., and Sanger, Ruth. 1962
Observations on subdivisions of the Rh antigen D. Vox Sang. 7: 9-13

581- Undevia, J.V. 1969
Population genetics of the Parsis.Comparison of genetical characteristics of the present Parsi population with its ancestral and affiliated groups. Ph.D. Thesis Bombay,India.269 PP

582- Vaisblit,R.I., and Lobodenko,R.L. 1965
Distribution of blood groups according to the ABO system, rhesus factor, and M and N factors among the residents of the Moldavian SSR.(In Rassian). Probl.Gematol.Pereliv.Krovi. 10, 11: 49-50

583- Valentine,W.N., Paglia,D.E., Tartaglia,A.P., 1977
and Gilsanz, F.
Hereditary hemolytic anemia with increased red cell adenosine deaminase(45-to-70 fold)and decreased adenosine triphosphate. Science.N.Y. 195: 783-785

584- Vana L.R., and Steinberg,A.G. 1975
Haptoglobin-ABO interaction. A possible explanation of the excess of HP among offspring of ABO incompatible matings. Amer.J.Hum.Genet. 27: 224-232

585- Van Baelen,H., R. Bouillon, and P. de Moore, 1978
The heterogeneity of human Gc globulin.J.Biol. Chem. 253:6344-6345

586- Van den Branden,J.L., Clements,V.R.,Mourant, 1971
A.E.,and Tills,D.
The distribution in human populations of genetic variants of adenosine deaminase. Hum.Hered. 21: 60-62

587- Vazin, H. 1969
Rh and blood transfusion.Factors of blood groups distribution. J. Tehran.Med.School.26,9: 797-817

588- Verbitsky,M.Sh., Annenkov,G.A., Bashlay,A.G., 1972
Morgulis, N.B., and Mirvis, A.B.
Distribution of genetic isoantigens of blood-group systems among the population of Abkhazian Svanetia. ii. Isoantigens of systems Rh-Hr,K-k,

FY, and Jk.(In.Rassian.). Vop. Antrop.40:
123-133

589- Verbitsky, M.Sh., Gogokhiya,Sh.D., Bashlay, A.G., Morgulis, N.B., Annenkov,G.A., Miris, AB., and Shnittsler, Sh. 1971
Distribution of genetic isoantigens of blood group systems among the population of Abkhazian Svanetia.i. Izoantigens of systems ABO, MNSs, P, Lewis and Lutheran.(In Russian). Vop. Antrop. 39: 17-30

590- Vesell, E.S. 1965
Genetic control of isozyme patterns in human tissues, In: progress in medical Genetics.4: 128-175(eds) Steinberg, A.G. and Bearn,A.G. New York, Grune and Stratton.

591- Voronov,A.A. 1968
Ethnogeographie des types generaux du haptoglobine de proteine serique.(In Rassian).Sovet. Etnogr. 2: 68-86

592- Voronov, A.A. 1973
Genogeography of blood factors in Trans Caucasus. IXth International Congress of anthropological and ethnological Sciences. Chicago, U.S.A.

593- Voronov, A.A. 1981
Ethnic factors and geography. Coll.Antropol. 5, 1: 109-118

FY, and Jk.(In Rassian.). Vop. Antrop.40: 123-133

589- Verbitsky, M.Sh., Gogokhiya,Sh.D., Bashlay, A.G., Morgulis, N.B., Annenkov,G.A., Miris, AB., and Shnittsler, Sh. 1971
Distribution of genetic isoantigens of blood group systems among the population of Abkhazian Svanetia.i. Izoantigens of systems ABO, MNSs, P, Lewis and Lutheran.(In Russian). Vop. Antrop. 39: 17-30

590- Vesell, E.S. 1965
Genetic control of isozyme patterns in human tissues, In: progress in medical Genetics.4: 128-175(eds) Steinberg, A.G. and Bearn,A.G. New York, Grune and Stratton.

591- Voronov,A.A. 1968
Ethnogeographie des types generaux du haptoglobine de proteine serique.(In Rassian).Sovet. Etnogr. 2: 68-86

592- Voronov, A.A. 1973
Genogeography of blood factors in Trans Caucasus. IXth International Congress of anthropological and ethnological Sciences. Chicago, U.S.A.

593- Voronov, A.A. 1981
Ethnic factors and geography. Coll.Antropol. 5, 1: 109-118

594- Voronov,A.A., and Potapov,M.I. 1967
The anthropology and application of data on the Gy-factor in blood groups(In Rassian).Vop. Antrop. 26: 130-136

595- Vos, G.H., and Comley,P. 1967
Red cell and Saliva studies for the evaluation of ABH and Lewis factors among the Caucasian and aboriginal populations of Western Australia. Acta. Genet. Stat. Med. 17:495-510

596- Vos, G.H., and Kirk, R.L. 1961
$Di^a$, $Js^a$ and V blood groups in south and south east Asia. Nature. 189: 321-322

597- Walaas, E., Lovstad, R.A., and Walaas,O. 1967
Interaction of dimethyl-p-phenylenediamine with ceruloplasmin. Arch.Biochem.Biophys. 121: 480

598- Walker, R.H., Argall, C.I., Steane,E.A.,Sasa- 1963
ki, T.T., and Greenwalt, T.J.
Anti-$Js^b$, the expected antithetical antibody of the sutter blood group system. Nature,Lond. 197: 295-296

599- Walsh, R.J., and Montgomery, C. 1947
A new human isoagglutinin sub-dividing the MN blood groups.Nature, Lond. 160:504-505

600- Walter, H., and Ananthakrishnan, R. 1975
Adenosindesaminase-system. Humangenetik. Band I/3. edited by P.E. Becker. Georg Thieme Verlag. Stuttgart. 549-556

601- Walter, H., and Bajatzadeh, M. 1968
Studies on the distribution of the human red cell acid phosphatase in Iranians and other populations. Acta.Genet., Basel. 18: 421-428

602- Wassenich, W.K., and Walter,H. 1968
Genetical and population-genetical investigations on β -lipoprotein polymorphism.Acta. Genet. Stat. Med. 18: 349-360

603- Walter, H., and Bajatzadeh,M. 1971
Investigations on the geographical variability of the human transferrins.Humangenetik. 12: 267-274

604- Walter,H., Bernhard,W., Tahir Hassan,S., and Bajatzadeh,M. 1966
Untersuchungen uber die verteilung der HP-,Gc- und Gm-Gruppen in Pakistan.Humangenetik. 2: 262-270

605- Walter, H., and Djahanschahi,D. 1963
Zur Haufigkeit der serum gruppen in Persien. Homo. 14: 70-76

606- Walter,H., and Steegmuller,H. 1969
Studies on the geographical and racial distribution of the HP and Gc polymorphisms.Hum.Hered. 19: 209-221

607- Walter, H., Strodtmann, H., Hilling,Maria., 1981
Singh,Indera P., Bhasin, M.K., and Veerraju,P.
Transferrin subtypes in six Indian population samples. Hum. Hered. 31: 152-155

608- Weiner, A.S., and Wexler, I.B. 1963
An Rh-Hr syllabus. Grunne and Stratton,New-York.

609- Weitkamp, L.R., Arends,T., Gallango,M.L., 1972
Neel,J.V., Schultz, Jane., and Shreffler, D.C.
The genetic structure of a tribal population: the yonomama Indians. iii. Seven serum protein systems. Ann.Hum.Genet. 35: 271-279

610- Welch, S.G. 1972
Quantitative differences between the human red cell glutamate-pyruvate transaminase phenotypes. Hum. Hered. 22: 190-197

611- Welch, S., and Lee, J. 1974
The population distribution of genetic variants of human esterase D. Humangenetik. 24: 329-331

612- Wendt, G.G., Kruger, J., and Kindermann,-. 1968
Serumgruppen und Krankheiten. Hum. Genet. 6: 281

613- West, C.D., Davis,N.C., Forristal,J., Herbst, 1965
J., and Spitzer, R.
Antigenic determinations of human $\gamma_1$C and $\gamma_1$G-globulins.J.Immunol. 96: 650-658

614- Whittaker, Mary. 1968
Frequency of atypical pseudocholinesterase in groups of individuals of different ethnographical origin. Acta.Genet. Stat. Med. 18: 567-572

615- Widstrom, G., and Henschen, Agnes. 1963

The relation between P.T.C. taste response and protein bound iodine in serum. Scand.J. Clin. Lab. Invest. 15, 69: 257-261

616- Wiebecke,D., and Brackebusch, H.D. 1974
Studies on the polymorphism of the soluble glutamic-pyruvic transaminse in the population of northern Bavaria (Germany).Humangenetik. 23: 227-229

617- Wieme, R.J. 1965
Agar gel electrophoresis. Elsevier, Amsterdam.

618- Wieme, R.J., and Demeulenaere, L. 1967
Genetically determined electrophoretic variant of the human complement component C3. Nature, Lond. 214: 1042-1043

619- Wieme, R.J., and Segers, J. 1968
Genetic polymorphism of the complement component C3 in a Bantu population. Nature,Lond. 220: 176

620- Wiener, A.S., Sonn, Eve B., and Belkin,R.B. 1945
Distribution and heredity of the human blood properties, A,B,M, N, P and Rh. J. Immunol. 50: 341-348

621- Wiener, A.S., Unger,L.J., and Katz, L. 1959
Studies on blood factors $Rh^A$, $Rh^B$ and $Rh^C$. J. Exp. Med. 110: 495-510

622- Wiener,A.S., and Unger, L.J. 1962
Further observations on the blood factors $Rh^A$, $Rh^B$, $Rh^C$ and $Rh^D$. Transfusion,Philad.2:230-233

623- Woodd-Walker,R.B., Smith, H.M., and Clarke, V.A. 1967
The blood groups of the Timuri and related tribes in Afghanistan. Amer. J.Phys.Anthrop. 27: 195-204

624- Wysiouchowa, B. 1970
Red cell acide phosphatase types in Poland. Population and genetic studies. Hum.Hered. 20: 199

625- Yamakami, K. 1926
The individuality of semen, with reference to its property of inhibiting specifically iso-hemagglutination. J.Immunol. 12: 185

626- Yasuda, N., and Morton,N.E. 1967
Studies on human population structure.In Proceedings of the third international congress of human genetics-edited by Crow, F., and Neel,J.V. John Hopkins Press, Balitmore.

627- Yoshida, A., and Beutler, E. 1978
Human glucose-6-phosphate dehydrogenase variants: a supplementary tabulation. Ann.Hum.Genet. 41: 347-355

628- Yoshida, A., Beutler, E., and Motulsky, A.G. 1971
Table of human glucose-6-phosphate dehydrogenase variants. Bull. WHO. 45: 243-253

## ADDITIONAL REFERENCE

Levine, P., Celano, M.J., and Falkowski, F. 1963
The specificity of the antibody in paroxymal cold hemoglobinuria (P.C.H.). Transfusion, Philad. 3: 278-280