Nottingham: the genetics of an urban population

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NOTTINGHAM : THE GENETICS OF AN URBAN POPULATION

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This thesis, which is entirely the result of my own work, has not been accepted for any degree, and is not being submitted concurrently in candidature for any other degree.

CONTENTS

Abstract

Nearly one thousand blood donor specimens from the county of Nottinghamshire were typed for seven different blood group systems. Demographic information was also gathered for each subject. The historical background and population development of the area was investigated and it was found useful to divide the county into three distinct geographical areas :- city, conurbation and the remaining area, and into three distinct types of residence pattern :- pit, rural and urban. These distinctions were also applied to the places of birth of the entire sample and to the birthplaces of the donors' parents and grandparents. Comparisons were made both within and between the different Nottinghamshire subsamples. Gene frequency results indicated that the whole of Nottinghamshire formed a homogeneous unit genetically, although there was some heterogeneity amongst the vertical structuring of occupational groupings. Further comparisons were made at a national level comparing the Nottinghamshire data with those from other areas in the United Kingdom.

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1. NOTTINGHAM - HISTORICAL BACKGROUND AND POPULATION DEVELOPMENT

1.1 Introduction

Modern Nottingham is a prosperous centre for the manufacture of cigarettes and tobacco, pharmaceuticals, leather, textiles, bicycles and electronic equipment, as well as its traditional Nottingham lace. Indeed, from its early beginnings, Nottingham has always prospered and appears to have preferred to play a more peaceful role than some of its neighbours, trading rather than fighting with the invaders of earlier centuries.

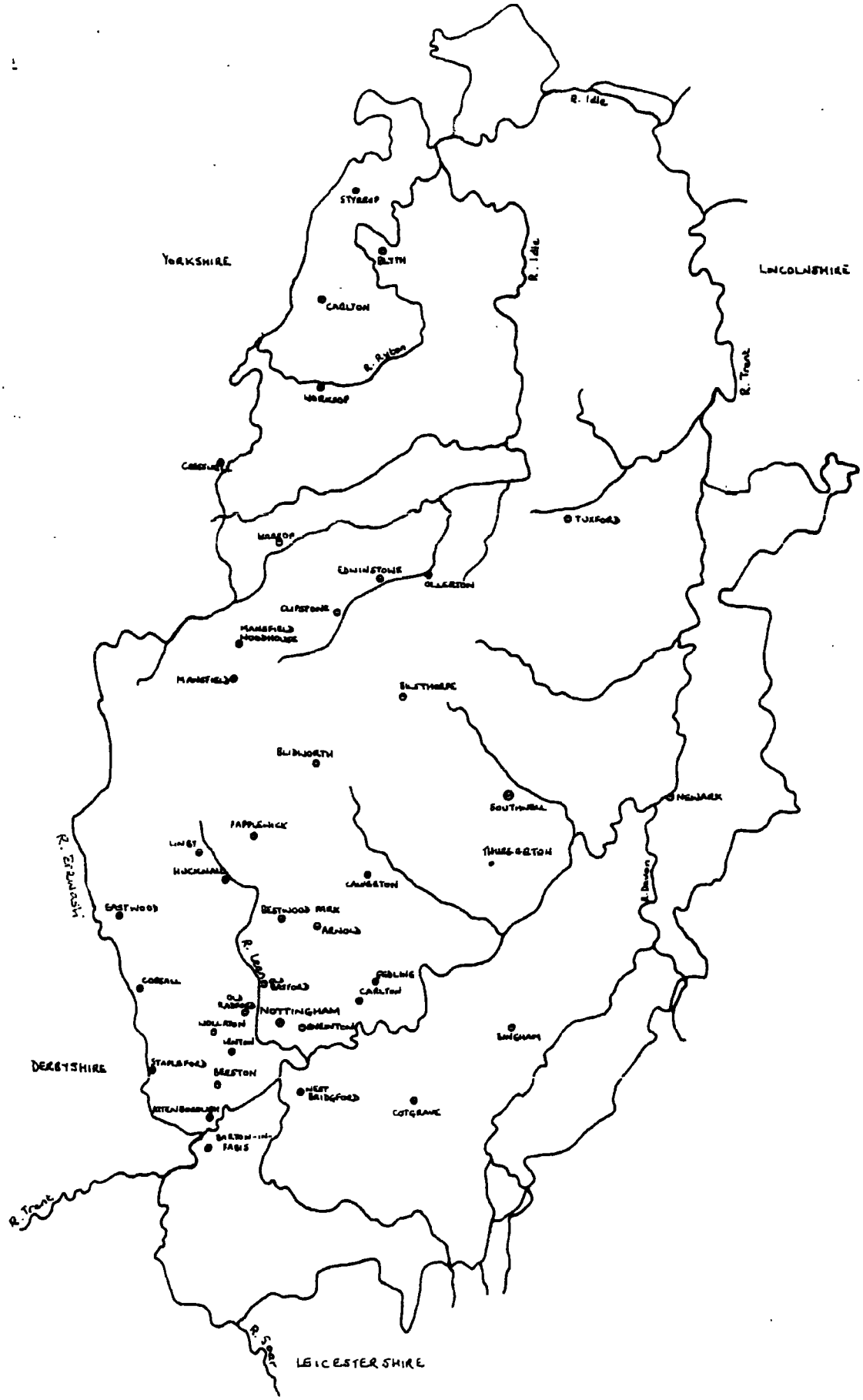
The change from an agrarian to an industrial centre came with the Industrial Revolution of the early nineteenth century and from this period onwards, continuously rapid expansion was possible, not only because principal industries were prosperous for long periods, but also because the successful growth of new kinds of manufacture offset stagnation and contraction of others.

For greater comprehension all places referred to in the text are located on the map of Nottingham (Fig. 1). A further two maps are included illustrating the location and size of the present day county boroughs and county districts of Nottingham (Fig. 2) and the regional divisions of the area (Fig. 3). Also included is a table listing the population figures for the years 1801-1971 and the intercensal variations (Table 1).

1.2 Prehistory

There is proof that Nottingham lay on the northern fringe of Palaeolithic settlement as indicated by the series of implements found in the gravel terraces of the middle Trent basin, particularly at Beeston and Attenborough, and from the excavations on either side of the gorge at Cresswell.

Figure 1. Map of Nottinghamshire



However, finds are scanty and Nottingham is poorly represented even in the later Neolithic and Bronze Ages.

One important site for the Middle and late Bronze Age period is Clifton, where a series of implements from both periods was found, together with three dug-out canoes and piles driven into the river bank, suggestive either of a landing stage or else of the foundations for the huts of a riverside settlement.

Again there is sparse evidence of settlement for the Iron Age, apart from a group of six to ten small fortresses distributed along the high ground of Keuper Marl hills and which overlook the left bank of the Trent and extend in a north-westerly direction into the Mansfield area.

From Ptolemy comes the information that the tribe which dwelt in the area now roughly covered by Leicestershire, Nottinghamshire and Lincolnshire was that of the Coritani. Their main political centre appears to have been Leicester, with the regions only known pre-conquest mint centre at Old Sleaford. It is not known whether Coritanian territory included the whole of modern Nottinghamshire, for it is possible that the Brigantes may have controlled the northern part of the county.

1.3 The Roman Period

The Roman legions first landed in 43 A.D. and by 47 A.D. the Coritani had been conquered, apparently without a struggle. Nottinghamshire became a frontier zone against the hostile Brigantes and four forts, or military posts, were built along the Fosse : Vernemetum near the modern Willoughby, Margidunum near East Bridgford, Ad Pontem at East Stoke and Crococalana at South Collingham. These forts were served by the Fosse Way and Ermine Street.

By the 2nd century the legions had progressed as far as York and the Scottish border, and consequently the Fosse frontier was no longer necessary.

The region made a significant contribution to the wealth of Romano-Britain, with deposits of lead, coal and iron, gravel and limestone. Remains of Romano-British villages have been discovered at Broxtowe and Tuxford and four villas at Styrrup near Blyth, Mansfield Woodhouse, Barton-in-Fabis and Potters Hill, Norton Disney. In the third and fourth centuries there was also an elaborate villa at Southwell and a villa complex at Thurgarton Beck.

1.4 The Anglo-Saxon and Danish Period

By the end of the fourth century German mercenaries were employed in the Roman army to aid the authorities in their struggle against raiders and it was these who in part paved the way for the beginnings of a considerable Anglo-Saxon settlement in the middle of the fifth century A.D. Evidence for the settlement pattern of this period may be gleaned from place-names as well as from archaeological finds.

The positioning of these place-names suggests that the first invaders entered the country by way of the Humber estuary and Trent valley, and did not extend further than Nottingham. They settled on both sides of the River Trent. Later settlement was by way of the valleys of the Trent's tributaries:- the Idle, Devon, Leen, Soar, Erewash, Derwent and Dove. A number of settlements are also found a short way from the Roman roads:- Ermine Street, Fosse Way and Ryknield Street. These settlements formed part of the kingdom of Mercia, and Nottingham itself became the frontier zone in the long struggle between the kingdoms of Northumbria and Mercia.

During the last quarter of the ninth century, there was an immigration of Danes into the North East Midlands and many made their permanent homes there. Military training and administrative headquarters were established at five important sites : Derby, Nottingham, Leicester, Lincoln and Stamford, the five Boroughs of the Danelaw. Although some

English villages were taken over by the Danes, in general it appears that the large number of places with names ending in -by represent new settlements, mostly in the tributary valleys of the Trent. The existence of five villages with the name Normanton - the Norseman's or Norwegian's village - shows that the army was not entirely made up of Danes.

In 1016 there is the first reference in the Anglo-Saxon Chronicle to Nottingham-shire and the borders of Nottingham have varied very little since this date. At this time Nottingham itself occupied a rectangular enclosure of approximately thirty nine acres which was surrounded on three sides by an artificial ditch, being flanked on the other, south, side by cliffs which formed a natural line of defence.

1.5 The Eleventh to Eighteenth Centuries

After 1066 William I ordered castles to be built at strategic points throughout the country and William Peverell was entrusted to erect one in Nottingham. Thus a new Norman borough came into being. It was located west of the existing Anglo-Danish settlement, for the Normans built their houses close to the castle and the English remained in the old town centred on St Mary's church. The two boroughs differed in their legal customs and until the reform of the Corporation in 1835, two sheriffs and two coroners continued to be elected each year. The last relics of the two boroughs are the two silver maces that are still carried before the Sheriff of Nottingham.

Trade flourished under Norman protection, for there is evidence to suggest that Nottingham people, never warlike, having offered opposition to none, were quite content to continue trading and to leave the protection of the town and any fighting to the Norman army. By the twelfth century Nottingham had become the trading centre of an area which stretched into Derbyshire and the Vale of Belvoir, and the enlargement of the town formed a pattern which was to continue for centuries.

Steady growth was maintained and by the seventeenth century the town had increased in area to 876 acres, supporting a population of about 3,000.

1.6 The Eighteenth Century

The stocking frame created the mechanized industry on which the growth and prosperity of Nottingham was to be built. At first London was the main centre of the industry, but it specialized only in fine silk work and therefore it was not surprising that worsted manufacturers should look for a centre nearer to their market and also to their supply of raw material. Such a centre proved to be Nottingham and by 1739 Nottingham had as many as 1200 frames at work. It was mainly a domestic industry and small houses were adapted each to hold a stocking frame and so towards the end of the century when the town became overcrowded, the industry was able to spread to outlying areas, for example, Arnold and Calverton.

Until the middle of the eighteenth century, there was no overcrowding and the population had not even reached five figures. However, from 1750 onwards the population grew at an incredible rate. This was a direct result of the many new inventions which mechanized the industry and thus brought about its concentration in urban centres, as well as providing employment for machine builders and machine maintenance workers. Also, it was a consequence of a change in agricultural policy to enclose a great deal of agricultural land for cattle rearing, which thus released many agricultural workers who migrated to the towns for work. This, together with the change in the population's age structure and a shift in emphasis from the death rate to the birth rate, brought about a population explosion. From a population of 10,000 in 1750, there was an increase to 17,500 in 1779. Between 1779-1801 the population grew by 11,000 of which nearly 60 per cent were immigrants.

Furthermore, as the fields and meadows surrounding Nottingham were owned separately and a decision was made not to enclose, the town was left no choice but to expand within its ancient manorial boundaries and this gave rise to a period of unequalled overcrowding and squalor. From 1801 to 1811 the population increased by 20 per cent to 34,030 and when enclosure came in 1845 a population of 53,000 was attempting to live on a site which had earlier been occupied by little more than one fifth of that number.

1.7 The Nineteenth Century

From 1801-1811 there was a 20% increase in population to 34,030. The manufacture of Nottingham lace contributed significantly to the doubling and redoubling of the town's population during the nineteenth century. It also accounted for the large number of female workers in the town.

Workers unable to find accommodation in the town spilled over into the neighbouring townships of Basford, Radford and Sneinton and framework knitter houses sprang up on the town-ward side of these townships, becoming known as New Basford, New Radford and New Sneinton. Thus semi-agricultural settlements were transformed into thriving industrial village suburbs which were all later to become part of Nottingham. The population of these townships trebled between 1830 and 1851. Conditions were appalling and epidemic diseases rampant.

1839 witnessed the coming of the railway; and the entry of steam power into industry, particularly for the lace machines, followed shortly. This led to a changeover from domestic to factory work for many people, although a great deal of stocking knitting and lace finishing continued to be a domestic industry. Insufficient sites in the town resulted in the land between the town and the outlying townships becoming dotted with factories in Basford, Hyson Green, Radford, Lenton and Sneinton. These new factories

Table 1. Population 1801-1971 and Intercensal Variations

Year of Census		Population	Intercensal increase	
			Amount	% per year
1801		140,350		
1811		162,964	22,614	1.48
1821		186,873	23,909	1.38
1831		225,394	38,521	1.89
1841		249,910	24,516	1.04
1851		270,427	20,517	0.81
1861		293,867	23,440	0.83
1871		319,758	25,891	0.85
1881		391,784	72,026	2.05
1891	(Ancient)	445,792	54,008	1.30
	(Admin.)	445,622		
1901		514,459	68,837	1.45
1911		604,098	89,639	1.62
1921		641,149	37,051	0.58
1931		712,731	71,582	1.08
1939	mid year estimate	757,000	44,269	0.74
1951	(1951 boundaries)	841,211	84,211	0.90
	(1971 boundaries)	841,188		
1961		902,976	61,788	0.71
1971		976,413	73,437	0.78

From 1801-1891 the figures relate to the Ancient County as altered by the operation of the Counties (Detached Parts) Act, 1844.

From 1891-1951 the figures relate to the Administrative County (with associated County Borough) as constituted in 1951.

From 1951-1971 to the same as constituted in 1971.

The relations between the various areas are indicated by the difference between the two populations shown for 1891 and 1951 respectively.

attracted workers from Leicestershire, Derbyshire, Lincolnshire, Huntingdonshire and Cambridgeshire, and also some foreigners from France, Germany, Poland, Hungary and elsewhere.

Between 1851 and 1861, 6,406 migrants born outside the Nottingham county boundary migrated to the town and, in addition, 7,230 people born in Nottinghamshire also moved to the regional capital. During the next decade there was an influx of people from outside Nottinghamshire who came to live and work in the suburban villages. By comparison, between 1851 and 1861 one half of the migrants came from within the county boundary, whereas between 1861 and 1871 only one fifth were Nottinghamshire people. These demographic patterns which continued until the end of the century were a result of a response to the changing location of economic activity and shift in the relative importance of different occupations.

Beyond the boundary of greater Nottingham, new pits opened to meet the increasing demand for coal required by the factories and the locomotives. There was a drift of population into these areas and small mining communities began to appear, for example at Cinder Hill, which was only 3 miles from Nottingham.

From 1831 onwards, there was an increased incidence of population decline in agricultural parishes. The 1834 Reform of the Poor Law and the coming of the railway network greatly facilitated the physical mobility of the population. From the beginning of the nineteenth century some of the lead mining villages in Derbyshire were losing people to Nottingham, for when the lead mines failed, or became uneconomical to work, people moved on to the coalfields. Other villages (for example, Linby, Papplewick) were affected by the closure of small country cotton-mills. Small canal ports declined when traffic was taken from them by the railways. Also, the population of certain parishes on the exposed coalfield declined in numbers when the pit workings were abandoned after centuries of work (for example, Bilborough, Cossall, Shelley, Wollaton).

Towards the end of the century, hosiery and lace, as employers, were already challenged by engineering and mining. The percentage of men working in the coal mines was rising and mining provided a welcome alternative employment in this traditional hand framework knitting area, and this eased the transition from hand labour to machinery. Employment was also helped by the establishment of what were to become three important industries.

John Boot, a labourer, opened a tiny herbal shop in Goose Gate in the City. When he died, in 1860, his wife and ten year old son Jesse carried on the business, which is now the vast Boot enterprise. In 1862 the small retailing and tobacco concern of William Wright passed into the hands of John Player. In 1880, the Raleigh Cycle Company was founded. The rapid rise of the cycle industry made great demands upon the supply of skilled mechanics and the existence in the Coventry and Nottingham districts of this kind of labour helps to explain why the cycle industry developed in these parts.

In 1877 the Borough Extension Act was passed. This brought about a consolidation of the industrial parishes for administrative purposes. The area encompassed by the enlarged boundaries increased from 1,996 to 10,935 acres, whilst its population rose from roughly 86,000 to 157,000.

1.8 The Twentieth Century

By the twentieth century on both a regional and national level there was a reduction in rural population decline. For a time, agriculture became more prosperous and suburbanization became easier and more popular with the help of a good local train service and the advent of the bicycle. However, there was still a reduction in the rates of natural increase with the loss of so many young people through migration. For instance, in the Ashbourne, Bingham and Southwell Districts the population was lower in 1911 than it had been in 1841.

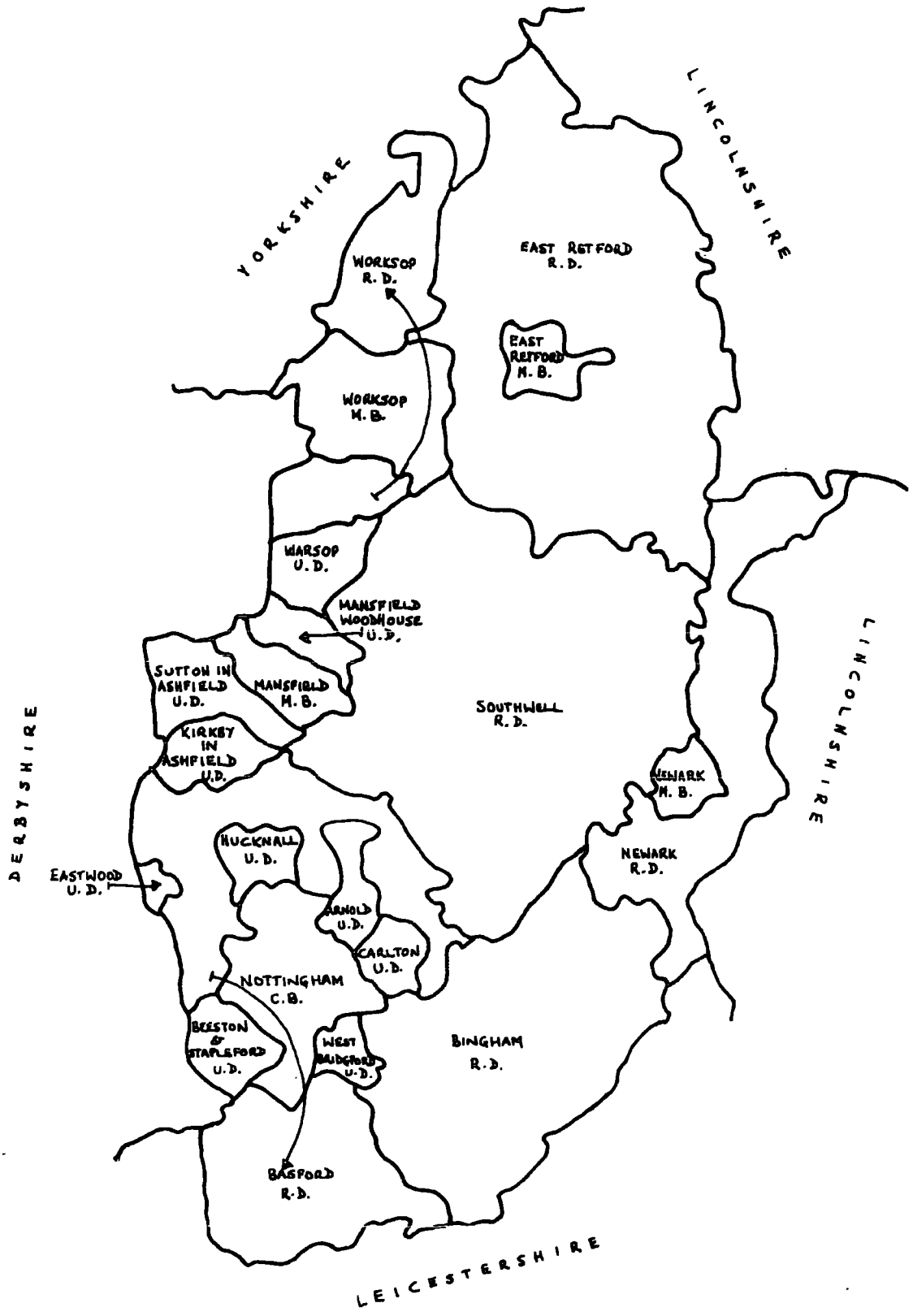


Figure 2. Location and size of present-day county boroughs and county districts of Nottinghamshire

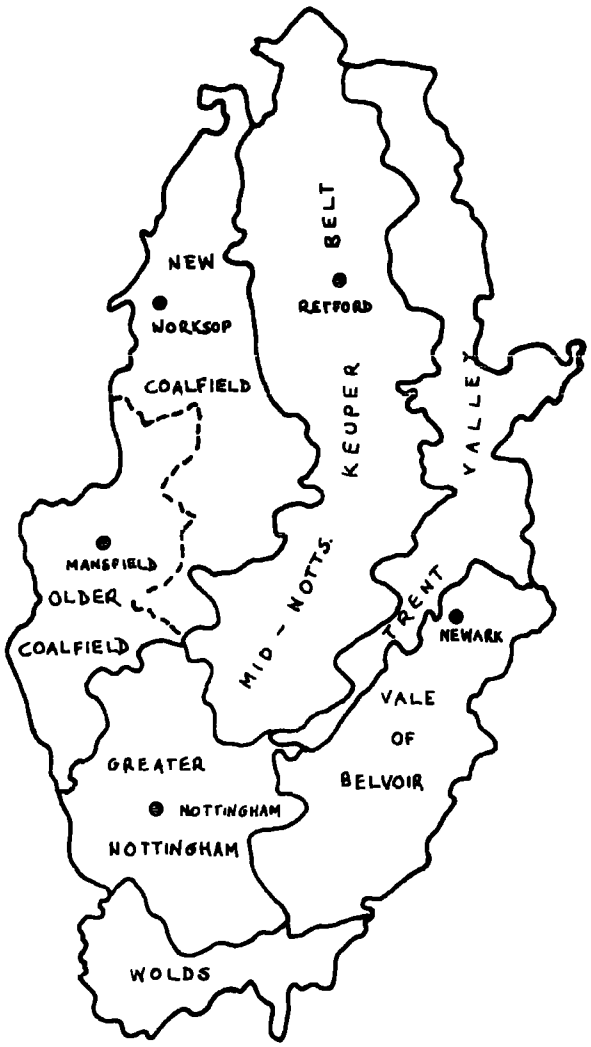


Figure 3. Regional divisions of the Nottingham area.

During the inter-war period, the arrival of the car and motor bus enabled people to live still further from their place of employment and this gave new life to certain villages which now drew their livelihood from employment in nearby towns. Nevertheless, many parishes still had lower populations than one hundred years previously - these lay chiefly in the rural east.

Immediately after the war renewed migration from the rural area was inhibited by the severe housing shortage, but by 1951 rural population decline was again apparent. However, there are a number of villages where substantial growth has taken place, especially where suburban development has occurred.

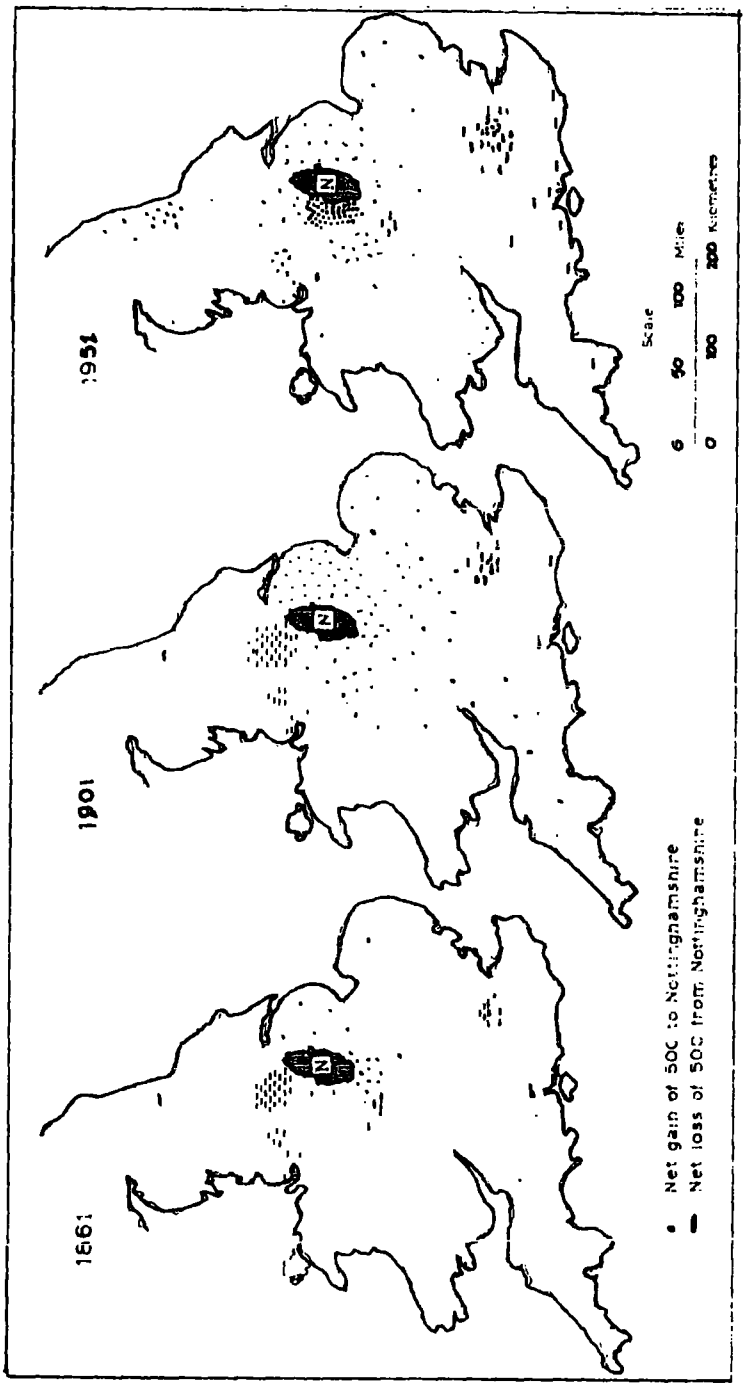
In Nottingham, the general trend of increasing mining activity has been from west to east through the years, and the population has moved in response. Thus, since the end of the nineteenth century, there has been some movement of Derbyshire mining families into Nottinghamshire. After World War I there was considerable development of new mining villages in the Bunter Sandstone district east of a line running approximately through Worksop, Mansfield and Nottingham (for example, Bilsthorpe, Blidworth, Clipstone, Edwinstowe and Ollerton). But even these new villages experienced economic difficulties. In many mining communities it is possible to discern a cycle of population change extending over several decades. A new mine is manned, often in an agricultural or a former knitwear village, and a reasonably youthful population moves in. Then follows a high rate of natural increase. However, unless the mine is still expanding locally this second generation may experience difficulties in finding employment and thus outward migration results. When the mining employment ceases altogether the population then stagnates or declines unless nearby mines or industries can absorb the displaced labour or school-leavers.

During this century there has been a net outward migration movement from the coalfield as a whole, thus illustrating the dangers of comparatively high rates of natural increase and an absence of alternative employment. However, in those coalfield towns where more varied industry was available the rates of emigration were at a minimum. For example, there was 0.5% emigration from Mansfield; 0.3% immigration into Worksop, an example of a coalfield town with market functions and a considerable range of industry, and 21% emigration from Warsop where there was no other employment except the local collieries. Migration rates of 6% or 7% are common throughout the Nottinghamshire coalfield, but being areas of substantial natural increase there is a tendency for the overall change to be one of modest increase.

Virtually all the districts south and east of the Trent, including the whole of greater Nottingham, have increased by immigration, whilst those north of the river are characterized by emigration or by lower than average rates of immigration. Generally speaking, there is a broad area of immigration centred on Nottingham and one of emigration covering the coalfields.

On a national level, in the nineteenth century Nottingham was losing population to the northern industrial counties, whilst in the first half of the twentieth century it was gaining from them. In both periods it was losing to London. Using the 'county of birth' data published in the 1861, 1901 and 1951 census (see Fig. 4) we can see that in the years before 1861 Nottinghamshire had been gaining from Leicestershire but losing to Lancashire and Yorkshire, the London area and the West Midlands. On balance the county had lost nearly 14,000 people. By 1901, Lincolnshire had become the chief single source of immigrants (13,900 on balance), the loss to the West Midlands had been reversed and the movements to London and the North had increased. On balance, however, there was a net gain from the rest of the country of 13,100. By 1951, the gain from Staffordshire had increased (decline in the Potteries) and a large

Figure 4.



Net migration to and from Nottinghamshire, 1861, 1901 and 1951

The net balances are in respect of English and Welsh persons enumerated in each of these years. Only balances of over 250 persons are shown. Movements from Nottinghamshire are assumed, for cartographical purposes, to have been directed to the nearer major urban concentrations in the counties concerned. Movements to Nottinghamshire are assumed to have originated either from such concentrations or from widespread places if the population of the county concerned was well distributed

number of people had moved into the county from Derbyshire, while the flow from Lincolnshire had diminished and there was now a loss to Warwickshire (including Birmingham and Coventry). There was also a striking reversal of the pre-existing movement north. These counties now showed substantial losses to Nottinghamshire, presumably owing to their less prosperous conditions and the N.C.B. encouragement given to Durham and Scottish miners to move south. Similarly, Welsh people were also affected. There was no great increase in southward migration although the number of counties which gained from Nottinghamshire increased. The outcome of all these movements was a net gain of 52,000 to which Derbyshire was the leading contributor.

Modern Nottingham may thus be divided into an industrial and urban west and an east which still remains predominantly rural. The same division is revealed between the relative rates of natural increase. Virtually the whole of the western half of the county has a net increase above the county average while the eastern half is uniformly below average. The general patterns of migration movements in the county are almost the reverse of this.

Modern Nottingham has a variety of industries and a well-balanced economic structure. The textile and clothing industry is no longer of such importance and the famous lace industry is now quite small. Hosiery still plays an important role but more markedly so in Leicester. Tobacco, chemical and pharmaceutical products have maintained the strong position they reached between the two World Wars and there has been a considerable expansion in a variety of engineering trades. However, more than 50 per cent of the working population are employed in the service trades, including building and contracting.

Nottingham in 1971 had a population of 956,410, and for the purpose of this study it was found profitable to divide Nottinghamshire into three areas, the older inner city area, the surrounding conurbation of the

five urban districts of Arnold, Carlton, West Bridgford, Beeston and Stapleford and Hucknall and the remaining area of rural districts and pit villages. However, none of these form distinct, clear-cut units.

Within the first area of study, Nottingham City, there are many service centres from which the population under study came, the four largest being Sneinton, Hyson Green, Sherwood and Bulwell. All were affected in the early nineteenth century by the development associated with the Industrial Revolution.

Sneinton lies half a mile to the east of the old Anglo-Saxon town and in the nineteenth century became a densely populated artisan quarter with its own market and gradually more and more shops have moved into the area. Another important area is Hyson Green which also resulted from the overspill housing of the nineteenth century from the district of Radford and the spread of industries along the Leen valley. Bulwell was another industrial township of the nineteenth century which grew with the increased numbers of factory workers and which also had an influx of miners living there from the local collieries.

Sherwood is an extension of Carrington, another location for a few small lace and framework-knitters' factories. It has however become a largely middle-class residential district.

Another area where there was considerable development with artisan houses interspersed with lace and hosiery factories and engineering works in the nineteenth century was Arkwright Street and this expanded to form the Meadows, a working-class housing area, now cleared of slum housing and in the process of redevelopment. Other similarly important areas are those centred on the older parts of St Anne's Well Road and Alfreton Road.

The second area, the five urban districts of Arnold (1971, population of approx. 33,000), Carlton (pop. 45,000), West Bridgford (pop. 28,000), Beeston and Stapleford (pop. 63,000) and Hucknall (pop. 26,000)

which together with Nottingham form the Nottingham conurbation, a true conurbation which forms a coherent and closely-knit economic and social entity; did not develop into major industrial and residential satellites until the late nineteenth century. Although these districts differ from one another in the degree to which they are dependent on the city, only a small percentage of the total working population finds employment outside the conurbation.

Arnold was an industrial village which with the building of factories in the late nineteenth century became a satellite of Nottingham. People are employed locally in the hosiery and engineering trades together with a few light industries.

Carlton developed likewise and one third of the working population is still concerned with hosiery-making; another source of employment is the colliery at Gedling. Even so, over 50 per cent of the working population is employed in Nottingham. In the older part of Carlton there is still a tight-knit community of hosiery-workers, miners and tradesmen.

West Bridgford, by contrast, is mainly a residential area for those of the middle-income group who earn their livelihood in the City. It is the district most dependent on the City.

Beeston and Stapleford are the largest of the urban districts and the most independent from Nottingham. This was brought about by the merging in 1935 of the urban districts of Beeston and the rural district of Stapleford. There has been a great deal of housing developments and there are many sources of employment, for instance, lace, hosiery and clothing factories, engineering works together with the Beeston Boiler works, Boots (pharmaceuticals) and Ericssons, such that there is an inward daily movement of workers chiefly from Nottingham.

Finally, 7 miles from the centre of Nottingham is Hucknall, primarily a coal-mining and hosiery-making centre.

The remaining area includes the chiefly rural districts of Newark, East Retford, Bingham and Southwell and the urbanized area of the twelve large collieries on the Coal Measures. From 1870-1920s the gradual eastwards advancement of mining led to separate settlements which have gradually expanded and merged to form an almost continuously built up area from Annesley in the south to New Clipstone in the north-east, and includes such places as Mansfield, Worksop, Eastwood, Kirkby-in-Ashfield, Mansfield Woodhouse, Sutton-in-Ashfield, Warsop and Basford. It is worth noting that the rural areas to the east and south now attract people from the city and there is a rapidly increasing commuter population dispersed among the larger villages.

2. RED CELL ANTIGEN SYSTEMS

2.1 Introduction

At the turn of the century a new group of physical characters entered the field of anthropology, namely the blood factors. Such blood factors are under precise genetic control for they are determined at conception and remain fixed for life, and thus are very reliable taxonomic tools. Their mode of inheritance is simple, straightforward and usually follows Mendelian laws and the gene frequencies in the populations tested may easily be computed from the observed phenotypic frequencies. For most systems the gene frequencies vary significantly from one population to another.

In this chapter I shall review the basic genetics of the red cell groups used in the present study.

2.2 Red cell antigen systems

2.2.1 The ABO blood group system

Landsteiner (1900) first observed a regular pattern of agglutination when cells and serum from different normal individuals were mixed. On the basis of these reactions Landsteiner (1901) was able to divide human individuals into three distinct categories:- O, A and B. Due to the fact that AB was generally rare, it was not until one year later that his pupils von Decastello and Sturli (1902) discovered this group.

Epstein and Ottenburg (1908) suggested that the ABO blood groups were inherited and this was demonstrated by von Dungern and Hirszfeld (1910). However, it was not until fourteen years later that, on the basis of an analysis of the association between A and B antigens, Bernstein (1924) recognized that the genetics of the ABO system were controlled by three

alleles A, B and O (sometimes referred to as p, q and r), at each of two loci. As both A and B are dominant to O, the six possible genotypes give rise to four phenotypes: A, B, O, AB.

Thomsen *et al.* (1930) discovered that blood type A could be further subdivided into two groups A_1 and A_2, so that there are six types: O, A_1, A_2, B, A_1B, A_2B. A_1 is determined by an allele A_1 which is dominant to A_2 and O. Thus genotypes A_1A_1, A_1A_2, A_1O are all type A_1 and only genotypes A_2A_2 and A_2O are of type A_2. A_2 is in general a weak antigen which is recognized by the use of an antibody which only reacts with A_1 and A_1B cells. Within the United Kingdom the gene frequencies for the A, B and O genes are approximately 68, 26 and 6 per cent frequency, respectively.

A number of other blood groups are now also known to be related to the ABO system, such as the P, Lewis and MNSs systems.

2.22 The MNSs blood group system

Landsteiner and Levine (1927a and b) were the first to describe the existence of two human antigens, which they called M and N. Unlike the discovery of most other blood group systems made by investigating an antibody found in human serum, the antibodies were prepared by injecting human blood into rabbits. The immune serum of the rabbit injected with blood type M agglutinated both M and MN erythrocytes and the serum treated with blood type N agglutinated N and MN erythrocytes.

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

Walsh and Montgomery (1947) reported the existence of another antigen in an Australian blood sample which was shown to be serologically different from M and N. This new antigen was called S (Sanger and Race 1947). Family studies indicated that persons possessing S were homozygous or heterosygous for one allele and persons who did not have S were homozygous for another allele. Levine *et al.* (1951) discovered the antithetical antibody, anti-s, which agglutinated the red cells of homozygotes as well as heterozygotes, thus indicating that there are two antigens, S and s, and three blood types, S, Ss and s.

The relationships of the M and N gene locus to that for S and s is considered to be very close and since crossing over occurs only very occasionally, Race and Sanger (1970) consider they occupy closely linked loci on the same chromosome; whereas Wiener and Wexler (cf. Race and Sanger 1950) believe they are multiple alleles at a single gene locus (see Table 2).

Table 2. The Genetical Interpretation of the reactions of anti-M, anti-N, anti-S and anti-s sera

Anti M	Anti N	Anti S	Anti 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

(After Race and Sanger 1950)

2.23 The Rhesus blood group system

Investigations which led to the discovery of the Rh factor were based on the assumption that antigens similar to undiscovered antigens of human blood might be present in a purer form in the blood of rhesus monkeys. Landsteiner and Wiener (1940) demonstrated that antigens produced by immunizing rabbits and guinea pigs with the red cells of the Monkey *Macacus rhesus* not only agglutinated the red cells of the monkey but also approximately those of 85 per cent of people of European descent. The red cells agglutinated by the anti-rhesus serum were classified as Rh positive, the remaining 15 per cent Rh negative.

It was soon discovered that the antibody previously described by Levine and Stetson (1939) in a case report of an unusual complication during childbirth had essentially the same specificity as anti-Rh and that many cases of haemolytic disease of the newborn could be attributed to its activity.

Two theories have been proposed to explain the inheritance of the rhesus factors, one by Fisher and Race (cf. Race and Sanger 1962), the other by Wiener (cf. Wiener and Wexler 1963).

The Fisher-Race concepts postulate three closely linked genes, C, D and E arranged in a linear sequence, but with C lying between E and D, on the same chromosome, which are inherited as a unit. At each gene locus there are two main alternative genes named C and c, D and d, E and e. The occurrence of the d antigen is presumed but has not yet been demonstrated. As only one of each pair can be carried on each chromosome, there are eight alternative rhesus gene combinations.

In the English population these Rh gene complexes occur in three orders of frequency. Frequent (12 per cent or over) CDe, cde and cDE; infrequent (less than 3 per cent) cDe, cdE, Cde and CDE; very infrequent CdE, CDE (see Table 3). According to Fisher and Race, genes on a chromosome

Table 3. Rhesus gene complexes and their U.K. frequencies

Gene	Complex	Percentage Frequency in U.K.
CDe	(R_1)	41
cde	(r)	39
cDE	(R_2)	14
cDe	(R_o)	3
C^WDe	(R^W_1)	1
Cde	(r')	1
cdE	(r")	1
CDE	(R_z)	0.2

are not absolutely linked and occasional crossing over from the common heterozygote is suggested to explain some of the rare gene combinations. For example, a cross-over occurring between C and D in the CDe/cde would produce cDe and Cde and in this way all four second order combinations could be produced. The production of CdE would require a cross-over from a heterozygote, for example, cDE/Cde which involves a second order complex Cde, itself a cross-over, a rare event.

Wiener and Wexler contend that there are multiple alleles each controlling the appearance of characteristic antigens. Rh-Hr factors are transmitted by a series of eight 'standard' allelic pairs which give rise to a possible thirty six different phenotypes. Wiener proposes that the gene be symbolized as a unit controlling an agglutinogen which may have multiple blood factor specificities - the serological properties by which agglutinogens may be recognized.

For a comparison of the Fisher-Race linked gene theory and the Wiener multiple allele theory see Table 4.

Table 4. Comparison of the Fisher-Race linked gene theory and the Wiener multiple allele theory

CDE System			Rh-Hr System		
Gene Complex	Symbol	Antigens	Gene	Agglutinogen	Blood Factors
CDE	R_z	CDE	R^Z	Rh_z	rh' Rh_o rh"
CDe	R_1	CDe	R'	Rh_1	rh' Rh_o hr"
cDE	R_2	cDE	R^2	Rh_2	hr' Rh_o rh"
cde	r	cde	r	rh	hr' hr" hr
cDe	R_o	cDe	R^o	Rh_o	hr' Rh_o hr" hr
cdE	R"	cdE	r"	rh"	hr' hr"
Cde	R'	Cde	r'	rh'	rh' hr"
CdE	r_y	CdE	ry	rh^y	rh' rh"

In the present investigation the nomenclature of Fisher and Race was used.

Various investigators have reported a number of additional alleles occurring at the Rh locus. They are labelled D^U, C^W, C^U, C^X, E^W, E^U, e^S and e^1. However, with the exception of D^U and C^W, all these alleles are unusual or rare in Caucasian populations.

2.24 The Kell blood group system

Coombs, Mourant and Race (1946) first encountered the existence of the Kell system. The red cells of a child thought to be suffering from haemolytic disease of the newborn gave a positive direct reaction which could not be explained by Rh. In the mother's serum there was an antibody

which sensitized her husband's and eldest child's cells and approximately seven per cent of random blood samples. Her name was Mrs Kell.

Levine *et al.* (1949) discovered the expected antithetical antibody anti-k. Inheritance of the Kell system is governed by a pair of allelomorphic genes K and k which determine the production of the corresponding antigens. Antigen K is determined by a dominant gene K with a frequency of about ninety five per cent.

Allen and Lewis (1957) reported a new antigen called Penney Kp^a which, through family studies, was found to be associated with the Kell system. They also described the antithetical Rautenberg Kp^b.

Thus the Kell system of blood groups is now defined by three pairs of antithetical antibodies, anti-K and anti-k, anti-Kp^a and anti-Kp^b and the Sutter system (anti-Js^a and anti-Js^b) found in Negroes. The complex locus controlling the three series of allelomorphic antigens appears to be similar to that controlling the Rh system.

The alleles Kk make useful distinctions and cause haemolytic disease of the newborn in Whites but not in Negroes. The Kp^a Kp^b alleles very rarely make useful distinctions and very occasionally may cause a mild haemolytic disease in Whites.

2.25 The Duffy blood group system

Cutbush *et al.* (1950) reported the discovery of the Duffy blood group system. 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. It was demonstrated that the antigen was inherited by means of a gene expressing itself in single and double dose. The gene which gave rise to the recognized antigen was called Fy^a and its allele, not recognized at that time, Fy^b. Ikin *et al.* (1951) discovered the antithetical antibody anti-Fy^b. The frequency of Fy^a in the British population is 42 per cent.

Sanger *et al.* (1955) found in their studies that the majority of American Negroes were of the phenotype Fy (a-b-), a situation previously unheard of, amongst Whites. The symbol Fy was used for this amorph. Since then Chown *et al.* (1965) have recorded cases of 2 Fy alleles in Whites.

2.26 The P blood group system

As a result of similar experimentation to that carried out for the MNS blood groups, Landsteiner and Levine (1927b) discovered the P blood group system. The P antigen was demonstrated to be inherited as a Mendelian dominant character, and the two types of blood were called P+ and P-. Levine *et al.* (1951) discovered a new antigen Tja but it was Sanger (1955) who recognised that in fact this antigen was also part of the P system. P- people, instead of lacking an antigen, were found to share a powerful one with P+ people, and a third extremely rare group was defined in which the antigen was lacking.

Thus the phenotype P+ became P_1, the phenotype P- became P_2 and anti-P became anti-P_1.

The P system demonstrates biochemical linkage with the A_1 A_2 O system. The reactions are graded in this system and variations occur in results for different types of antisera. In the British population the gene P_1 has a frequency of slightly over 50 per cent and similar frequencies are found throughout Europe.

2.27 The Lewis groups

Mourant (1946) described the antibody, anti-Le^a which agglutinated the red cells of approximately 22% of Europeans. Andresen (1948) described the almost antithetical antibody anti-Le^b. However, our understanding of this system is by no means complete.

Grubb (1951) proposed a general theory of the Lewis groups based on the presence or absence of antigens, not of the red cell, but of the saliva. Le^a substance in saliva is present in over 90% of Europeans but in greater amounts in those people whose red cells are Le (a+ b-); those lacking this substance have Le (a-b-) red cells. Le^b substance is present in the saliva of people whose red cells are Le (a-b+).

Sneath and Sneath (1955) observed that when suspended in plasma containing Le^a or Le^b, red cells which lacked either of these substances would take up these antigens. Therefore we must not regard the Lewis antigens of the red cell as being directly genetically predetermined but rather as being passively acquired from antigens of the plasma.

Ceppellini (1955) showed that the presence of Le^a in the saliva was controlled by a gene dominant in effect but independent of the secretor genes. He called the genes L and ℓ, but he and later workers have changed to Le and ℓe.

Lawler and Marshall (1961) have found that the Lewis antigens are present at birth in saliva and serum although they are absent from the red cell.

3. MATERIALS AND METHODS

3.1 Blood and Data Collection

3.11 Blood collection

The blood specimens used in this study were collected on four separate occasions from the central drawing station in the city of Nottingham and on one occasion from the village of Edwinstowe, a small coal mining community several miles to the north. A point was made to collect blood from all areas of Nottingham and in fact this was achieved by using the Castle House drawing station. A maximum of 180 people was taken during each session in order to gain a wide range of age groups and social classes and to eliminate population bias. The blood sample was taken into liquid citrate and received in Durham within 50 hours of collection.

3.12 Demographic data collection

Demographic information was gathered for each subject. This included where the individual was interviewed, his date of birth, sex, present address and occupation; whether the person was born in Nottinghamshire or had moved there after birth and, if so, when; also, where his parents were born and their occupations and, similarly, where his grandparents were born and their occupations. All areas refer to the 'old' counties before the reorganization which took effect in April 1974.

Three residential types are clearly defined in Nottinghamshire, namely urban Nottingham, pit villages in the Nottinghamshire coalfield and the rural areas in between. These residential categorizations were applied to the birthplaces of the entire sample.

Occupations were classified according to the Classification of Occupations 1970 from the Office of Population Censuses and Surveys. The basic common factor of all groups is the kind of work done and the nature of the operation performed.

Since the Census of 1911 it has been the practice, for certain analytical purposes, to arrange the large number of unit groups of the Occupational Classification into a smaller number of broad categories called Social Classes, and this was done for the present study.

These social classes are:

I Professional etc. occupations

II Intermediate occupations

III Skilled occupations

(N) non-manual

(M) manual

IV Partly skilled occupations

V Unskilled occupations

As far as possible the occupational unit groups included in each of these categories have been selected to ensure that as far as possible each category is homogeneous in relation to the basic criterion of the general standing within the community of the occupations concerned.

The Social Class appropriate to any combination of occupation and status is derived by the following rules:

a. each occupation is given a basic Social Class.

b. persons of foreman status whose basic Social Class is IV or V are allotted to Social Class III.

c. persons of manager status are allocated either to Social Class II or III, the latter applying if the basic class is IV or V, except those in occupation groups 003 (agricultural workers not elsewhere classified), 004 (agricultural machinery drivers) and 006 (foresters and woodmen) who are allocated to Social Class II.

3.2 Red Cell Preparation

Once in the laboratory at Durham the red cells were separated by centrifugation at 2,000 r.p.m. for 10 minutes and the serum was decanted and frozen to -20°C and stored for future use. The red cells were washed three times in normal saline (0.9% NaCl) in order to remove the anti-coagulant and a 4% suspension of the cells in saline was prepared for red cell grouping. The remaining red cells were frozen and stored for haemolysate preparation.

The sera and lysates were used in tests for further polymorphisms, not included in the present study.

3.3 Red Cell Serology

Three different methods were used for grouping the blood, depending on the type of antiserum employed.

3.31 The Tile Technique

Equal volumes of antiserum and 4% red blood cell suspension were placed on a scored glass tile. The red cell-serum mixture was left for a specific length of time at a certain temperature. The tile was intermittently rotated to mix the cells and antiserum, and after the specific time period had elapsed the mixture was inspected for agglutination. For investigations carried out at 4°C, the tile had been previously cooled to this temperature.

3.32 The Tube Technique

This technique involved placing equal volumes of red blood cells and antiserum in a precipitin tube. The serum-cell mixture was then incubated for a specific length of time at a certain temperature and the results were read microscopically.

The addition of bovine albumen or papain was required to enhance the reaction of some antisera.

For the antisera requiring an albumen displacement method the albumen was run gently down the inside of the precipitin tube after the cell suspension had been incubated with the antiserum for 1½ hours, and the serum-cell mixture was then re-incubated for a period of 30 minutes.

For the papain method used with anti-e, the red cells were initially sensitized with 1% papain solution by agitating one drop of the 4% saline suspension with two drops of the papain solution. The resulting suspension was spun so that the excess papain could be removed and the sensitized cells were re-suspended in two drops of saline. One drop of anti-e was added and the test read after incubation for one hour at 37°C.

3.33 The Indirect Coombs Technique

This technique involved placing equal volumes of 6% red blood cell suspension and antiserum in a precipitin tube. The serum-cell mixture was then incubated at 37°C for one hour. After incubation the cells were washed three times with saline. A drop of shaken red cells was mixed with one drop of anti-human globulin on a scored tile and the tile was then rotated gently for 5-10 minutes and agglutination was observed over a strong light.

3.34 Antisera

The following blood transfusion centres kindly provided antisera for this study. They are listed, together with the method employed for each particular antiserum (Table 5).

Table 5. List of antisera, its source, and method employed for testing

Antiserum	Source	Method		
anti-A	C.B.G.R.L.	Tile Room Temp.		10 mins
anti-A_1	Newcastle B.T.S.	Tile	4°C	10 mins
anti-A_{hel}	Biotest	Tile Room Temp.		30 secs
anti-B	C.B.G.R.L.	Tile Room Temp.		10 mins
anti-A+B	C.B.G.R.L.	Tile Room Temp.		10 mins
anti-M	Newcastle B.T.S.	Tile Room Temp.		10 mins
anti-M	C.B.G.R.L.	Tile Room Temp.		10 mins
anti-N	C.B.G.R.L.	Tile Room Temp.		10 mins
anti-N	C.B.G.R.L.	Dil. 1 in 1 saline		
		Tube	18°C	2 hrs
anti-S	C.B.G.R.L.	I.D.C.	37°C	1 hr
anti-S	Biotest Diagnostics	I.D.C.	37°C	1 hr
anti-S	Sheffield B.T.S.	I.D.C.	37°C	1 hr
anti-s	Biotest Diagnostics	I.D.C.	37°C	1 hr
anti-s	Ortho Diagnostics	I.D.C.	37°C	1 hr
anti-C	Newcastle B.T.S.	Tube alb. disp.	37°C	2 hrs
anti-c	Newcastle B.T.S.	Tube alb. disp.	37°C	2 hrs
anti-D	Newcastle B.T.S.	Tube alb. disp.	37°C	2 hrs
anti-E	Newcastle B.T.S.	Tube alb. disp.	37°C	2 hrs
anti-e	Newcastle B.T.S.	Papainised cell tech.	37°C	50 mins
anti-C^W	C.B.G.R.L.	Tube alb. disp.	37°C	2 hrs
anti-K	Newcastle B.T.S.	I.D.C.	37°C	1 hr
anti-Cellano	Biotest Diagnostics	I.D.C.	37°C	1 hr
anti-Cellano	Sheffield B.T.S.	Dilute 1 in 6 AB sera		
		I.D.C.	37°C	1 hr
anti-Kp^a	C.B.G.R.L.	I.D.C.	37°C	1 hr
anti-Kp^b	Biotest Diagnostics	I.D.C.	37°C	1 hr
anti-Fy^a	Hyland Laboratories	I.D.C.	37°C	1 hr
anti-Fy^a	Newcastle B.T.S.	I.D.C.	37°C	1 hr
anti-Fy^b	Biotest Diagnostics	I.D.C.	37°C	1 hr
anti-Le^a	Lancaster B.T.S.	EDTA Replacement tech.	16°C	1 hr
anti-P_1	Lancaster B.T.S.	Tile	4°C	10 mins

C.B.G.R.L. = Central Blood Group Reference Laboratories

3.35 System of Testing

The Lewis, P and S groups were always tested first. Each result was checked by at least one other person and wherever possible the same batch of antiserum was used for the whole series. Anti-P_1 was an exceptionally strong antiserum and therefore these results may only be compared with similarly tested series.

Appropriate controls (homozygous positive, heterozygote and homozygous negative) from Ortho, Biotest or Newcastle B.T.S. were set up with each run. However, in the case of testing with anti-Kp^a, no suitable controls were ever available.

In the case of unusual phenotypes (for the rhesus and Kell systems) corroboration was obtained with the use of commercial antisera.

For the ABO blood group system all 1,068 bloods were tested with anti-A, anti-B and anti-A+B. All A positives were tested with anti-A_1, A_1 negatives were tested with anti-A_{hel} to confirm the A_2 results; all cases were confirmed.

For the MNSs system all 1,068 bloods were tested with anti-M, anti-N and anti-S. S positives were tested with anti-s.

For the Rhesus system all 1,068 bloods were tested with anti-C, anti-D, anti-E and anti-c. E positives were tested with anti-e, C positives were tested with anti-C^W.

For Kell and Kp^a all 1,068 bloods were tested with anti-K but only Kell positives were tested with anti-Cellano. However, due to a shortage of antisera only 350 were tested with anti-Kp^a, and only Kp^a positives were tested with anti-Kp^b.

All the bloods were tested with anti-Fy^a and anti-Fy^b and anti-P, but there was only sufficient antiserum for the testing of 620 individuals for Le^a. This antiserum contained anti-B and consequently only individuals of groups A and O were tested.

3.4 Statistical Analysis

Gene frequencies were computed by the gene counting technique unless otherwise stated below, and populations were tested for accordance with the predictions of the Hardy-Weinberg law in the standard way. The standard errors of the gene frequency estimates were computed according to the formula:

$$\text{Standard error} = \sqrt{\frac{p(1-p)}{N}}$$

where p is the gene frequency concerned and

where N is the total sample size

and were calculated for each system to introduce a measure of sample size variation into the values obtained.

The ABO gene frequencies were calculated using the formulae given by Mourant *et al.* (1976) and which are attributable to Bernstein.

The formulae are:

$$p_1^1 = 1 - (\bar{O} + \bar{A}_2 + \bar{B} + \overline{A_2B})$$

$$p_2^1 = (\bar{O} + \bar{A}_2 + \bar{B} + \overline{A_2B} - (\bar{O} + \bar{B})$$

$$q^1 = 1 - (\bar{O} + \bar{A})$$

$$r^1 = \bar{O}$$

where p_1^1 p_2^1 q^1 and r^1 are initial estimates of the A_1 A_2 B and O gene frequencies respectively.

From these initial estimates can be calculated a correction factor D:

$$D = 1 - (p_1^1 + p_2^1 + q^1 + r^1)$$

and final values are given by:

$$p_1 = p_1^1 \ (1 + D/2)$$

$$p_2 = p_2^1 \ (1 + D/2)$$

$$q = q^1 \ (1 + D/2)$$

$$r = (r^1 + D/2)(1 + D/2)$$

These final values of p_1 p_2 q and r approximate to the maximum likelihood estimates for the gene frequencies.

All statistics were coded, carded and computerized using an IBM 360/67 and 370/168 computer (NUMAC) using the SPSS (Nie *et al.* 1975) packaged programmes.

3.5 Sources of Error in the Data

Although all precautions were taken to avoid mistakes, the possibility that some errors may have occurred must not be denied. All the red cell grouping procedures are open to observer error.

In the present study, the frequency of the heterozygote MN is greater than the theoretical maximum of 50% predicted by the Hardy-Weinberg Law. This may well be the result of the mis-typing of some M individuals as MN because of the agglutination with anti-N, as discussed by Mourant *et al.* (1976).

Similarly, the frequency of the heterozygote Cc was greater than the 50% theoretical maximum. Again it is probable that this was the result of typing errors, although it does not conform to the most usual error (cited by Mourant *et al.* 1976) of false negative results with anti-c which give an excess of CC homozygotes and a deficit of heterozygotes.

With the P system of graded reactions there is more scope for observer error than any other system, especially as the continual freezing and thawing of the antiserum over the time interval of the study may have affected the titre. Thus for gene frequency calculation all the results were classified as either positive or negative reactions and did not include the grading from one to five of the strength of reaction.

There is also the possibility, although slight, of transcription errors in the computerization even though double checking was used at all stages.

Furthermore, there may be some errors in the demographic information, particularly in the replies given for the occupational status of the donor, since this could in no way be corroborated. However any dubious replies were discounted and the cards removed from the study.

Finally, on a more basic level is the underlying problem of whether or not blood donors may be regarded as a random sample of the population under study. Kopeć (1970) found that blood donors did not differ significantly from a large number of Royal Air Force recruits for ABO and Rhesus D frequencies, although this does not mean that either group is representative of the general population.

4. RESULTS

Complete tables of all the gene frequency results may be found in Appendix A. The standard error of the arithmetic mean is given together with the number tested for each system. Gene frequencies were used as they provide a more direct way of comparing the blood group content of different populations than do the phenotype frequencies. As mentioned in 3.12 gene frequencies were computed for each donor with respect to his sex, his birthplace (both area and type, that is, whether born in the city, the conurbation or the remaining area and whether it was an urban, pit or rural area) and that of his spouse, his parents and his grandparents; also, his area and type of residence and his own and his father's occupation. Immigration tables were also compiled for each decade of this century, but due to small sample sizes for the years 1901-1940 it was decided to include all the results from these years together as one sample. Similarly, paucity of numbers made comparisons unprofitable for the results from Edwinstowe as a separate unit and so for all distinctions other than sex of donor there are two main groups of results, those from Nottingham taken from the sessions held at Castle House and a second group for all the sessions, which thus includes the Edwinstowe results.

The tables were studied to discover whether the sub-samples differed significantly or whether they formed a homogeneous group with regard to area and type, sex and social class for all the blood group systems. Twice standard error was regarded as a significant difference since this corresponds to the 5% level of probability.

Despite variations between all sub-samples there was only one result which proved significant for 2 standard errors, namely gene frequencies segregated on the basis of donor's occupation for the ABO blood group system. For the gene p^1 there was a significant difference of 2 S.E. between the

professional classes and the intermediate, the skilled non-manual, the skilled manual and the unskilled classes:

Table 6. P Gene frequencies segregated on the basis of donor's occupation

	Nottingham and Edwinstowe					
	Professional	Intermediate	Skilled non-manual	Skilled manual	Partly unskilled	Unskilled
p^1	0.032 ±.044	0.205 ±.030	0.233 ±.030	0.207 ±.024	0.187 ±.041	0.317 ±.065
No.tested	16	178	201	296	92	51

All other comparisons did not demonstrate any significant differences.

5. DISCUSSION

5.1 Introduction

Population genetics is a vast subject which is concerned with understanding both the nature and source of inherited differences, with the changes in the relative frequencies of these differences within a population, and with considering the conditions under which equilibrium between the various forces acting upon the different types may be obtained. The various forces which contribute to changes in gene, genotype and phenotype frequencies are those which have a directional effect, such as mutation, gene flow and selection and their random variability which may occur either spatially or with time, and the stochastic forces such as genetic drift. Gene flow which occurs only through some form of movement is the great homogenizing influence in evolution.

The fact that a population does not deviate significantly from a Hardy-Weinberg equilibrium does not necessarily mean that all the possible causes of divergence are absent, it simply indicates that either they are not present at a level that can be detected with the number of individuals tested or that disturbing forces have opposite effects which cancel each other.

Although there is increasing evidence to support the view that factors of drift have been underestimated in influencing the differences in populations, it is still generally believed that the genetic composition of human populations is determined by the action of differential selection; both the natural and social environment exerting the selection pressure on the gene pool. Thus population variability in gene frequencies is seen as the result of adaptation, either in the past or currently, to different climatic, nutritional, disease and similar natural environmental factors, and also possibly to different social and technological systems and ways of life.

Every population is in some sense unique. Every population has a history of its own - migration, admixture and all sorts of historical accidents have taken place, and still are taking place, and it must be recognized that cultural factors can clearly affect biological structures. The proper study of the biology of modern man of necessity should be multi-disciplinary in character, there is a need for a combined approach which, for instance, incorporates the demography of the population under study, for patterns of fertility and mortality, emigration and immigration and the effect of these on the age and sex composition are all important factors to be taken into consideration when looking at the measurable variability of the population under study.

Unfortunately there has been very little research undertaken on these lines and so published comparative material is scanty, also, few urban population studies have been carried out. Clearly there is a need for a great deal more research and compilation of data, and a recent publication outlining the known world distributions for many genetic polymorphisms (Mourant A.E. *et al.* 1976) highlights the paucity of knowledge on regional polymorphic distributions in the United Kingdom.

5.2 Nottinghamshire

Except for the work on the ABO and Rh(D) blood group antigens (Kopeć A.C. 1970) and one study by Dunsford (cited in Mourant A.E. *et al.* 1976) on Rh(D) blood group antigens, there has been no research into genetic distributions in the Nottinghamshire region. Due to the scarcity of genetic information, all the results from this study are included in Appendix A so that the data may be readily available for future detailed inter-regional comparisons.

As mentioned in 3.5 the most serious potential bias in the sample lies in the ABO and Rh(D) results. The donors were called in the usual

fashion and do not necessarily represent the entire panel or the normal population of the area. However, the closeness of the ABO and Rh(D) results to those of Kopeć (1970) may be taken to indicate that the sample is representative of the whole region. Kopeć's main data from Nottingham was analyzed with that from Worksop, Retford, Newark, Sutton-in-Ashfield and Mansfield to approximate to the area under comparison in the present study. It must be mentioned, however, that although Nottinghamshire has a multi-racial population, only 10 donors were either West Indian or Indian, and thus a fairly sizeable coloured population has not been represented. It was also unfortunate for comparative purposes that permission to collect data from schoolchildren could not be obtained from the Director of Education.

Out of the total sample, 42% resided in the City, 25% in the conurbation and 33% in the remaining area. When considering type of residential area, of the total sample 24% resided in a pit type of residence, 8% in a rural and 68% in an urban residential type. When looking at birthplaces of the Nottinghamshire-born sample there is very little difference in distribution, namely of the total sample 54% were born in the city, 13% in the conurbation and 33% in the remaining area. Of these, 25% were born in a pit residential type, 7% in the rural type and 68% in the urban type.

In view of the present geographical distribution and historical background of the Nottinghamshire people it was thought worthwhile to investigate the amount of genetic distinctiveness of the area, to see whether or not there were any significant differences between the three areas under study and in particular to undertake a comparison of the pit and rural areas with the main urban complex. However, although cumulative changes over the last few generations must have produced considerable gene frequency changes between and within areas, the region proved to be a homogeneous unit with regard to the blood group gene frequencies.

Variation in the gene frequencies did exist but not at a 95% significance level. Of the various blood group systems studied, the A and O genes of the ABO blood group system and the D and E genes of the Rh system and the P_1 gene of the P blood group system exhibited the greatest variation, and by far the most variation occurred between the rural and urban areas, although again not at the significant level of twice standard error. The only significant difference in the present series of comparisons was that found between the different social classes with regard to the ABO blood group system. This raises an interesting point that with further investigation the region may prove to be structurally heterogeneous with regard to occupational groupings and social class, and this in turn emphasizes the need for the inclusion of demographic information in genetic studies of different populations. For if there is an heritable element to the attributes which influence whether or not an individual moves up or down in the social hierarchy, then social mobility becomes an important example of selective movement which can have profound effects on the genetic structure of populations. Of all the possible structures of a population, the occupational or social class structure has been widely recognized by demographers as important in studies of differential fertility and mortality in populations.

Thus the population of Nottinghamshire may be considered as an homogeneous unit genetically, although some significant variability does exist within the social class structure. Perhaps this conclusion is understandable when the history of the region is taken into account. Unlike Ireland or Wales, for instance, Nottinghamshire has no history of Celtic ancestry or cataclysmic population disasters. The native population has readily assimilated the foreigner or outsider, from the very early days when it preferred the beneficial aspects of trading rather than fighting with the enemy. With the Industrial Revolution (see 1.6) agricultural workers

readily moved to the towns to gain employment. In the nineteenth century (see 1.7) many semi-agricultural settlements were transformed into thriving industrial village suburbs which were all later incorporated into Nottingham. During this century there was a constant flow of immigrants from both outside the region and abroad who came into the area in search of work in the new factories. Further mobility was encouraged by the opening of pits to satisfy the demand for coal and many Derbyshire mining families moved into the area. Again, there was immigration from Coventry when skilled mechanics moved into the area to work in the Raleigh Cycle Factory. In the twentieth century (see 1.8) we find the inception of a commuter population bringing new life to village communities, a youthful emigration from the coalfield area to find alternative employment, and an influx in the 1930s of many Staffordshire pottery families, and in the 1950s Durham and Scottish mining families, to find employment in this prosperous region, for Nottingham has always been favoured by a reasonably low unemployment problem. Thus it was a population accustomed to movement both to and from the area, and it would be interesting to see how these gene frequency figures compared with the other regions which constitute the English Midlands.

5.3 Comparisons with other areas in the United Kingdom

When looking at the relevant results from Nottinghamshire as a whole compared with other studies in the United Kingdom, it must be emphasized that the populations with which the entire sample is contrasted are not particularly appropriate, but they do represent the only other British series available. All the comparative material together with the results from Nottinghamshire are located in Tables 7-15.

As with the Nottinghamshire results, differences in gene frequencies are compared on the basis of standard errors (see 3.4). In order to establish the degree of confidence with which standard error may be used in such a comparison, it is necessary to determine whether the results have a normal distribution. To test for normality of the frequency distribution the Rankit method was used. The gene frequencies p, q amd r for the ABO blood group system from Table 7 were plotted and the best curve fitted (see Fig. 5). It can be seen from Figure 5 that the curves for p and q approximate well to a straight line, thus indicating normal distribution. The curve for r was slightly skewed and thus did not approximate so well to a straight line. It is probable that the reason for this skewed curve is due to the summation of the sampling errors from p and q. Nevertheless, the graphs indicate that the use of standard error to compare the Nottinghamshire results with those from other areas is justified.

When comparing the ABO gene frequencies (see Table 7), it was found that the Nottinghamshire data for the p gene were significantly different for 2 standard errors from the data from Lancaster and Northern Ireland. For the remaining data there were no comparisons that were statistically significant. The Nottinghamshire p results were slightly higher than those from all the other areas except for the Isle of Man, Fishguard, Tenby, Worcestershire and Warwickshire, and Oxfordshire and the nearest approximation in frequency was that from Derbyshire. For the gene q there were no differences of any significance, the Nottinghamshire result demonstrating a higher gene frequency than the other areas except for Caernarvon, Fishguard, Tenby and S.W. Scotland. For the gene r there were 2 results which were significant at twice standard error, namely S. Cumberland and Lancaster. Nottinghamshire had a lower r gene frequency result than all the other areas except Fishguard and Tenby and again the nearest comparable gene frequency result was that from Derbyshire.

Figure 5 Graphic test for normality of ABO gene frequency distributions

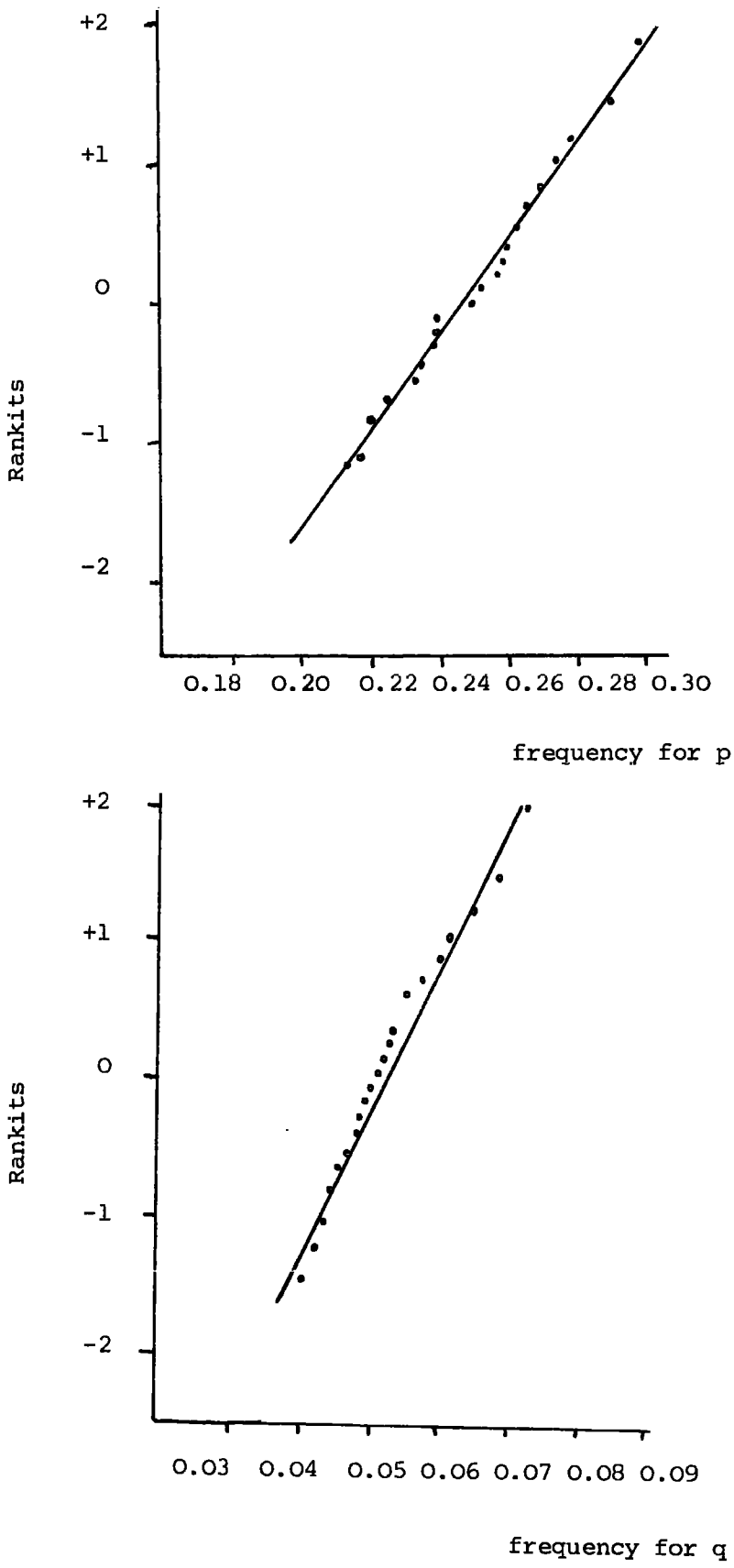


Figure 5 (contd.)

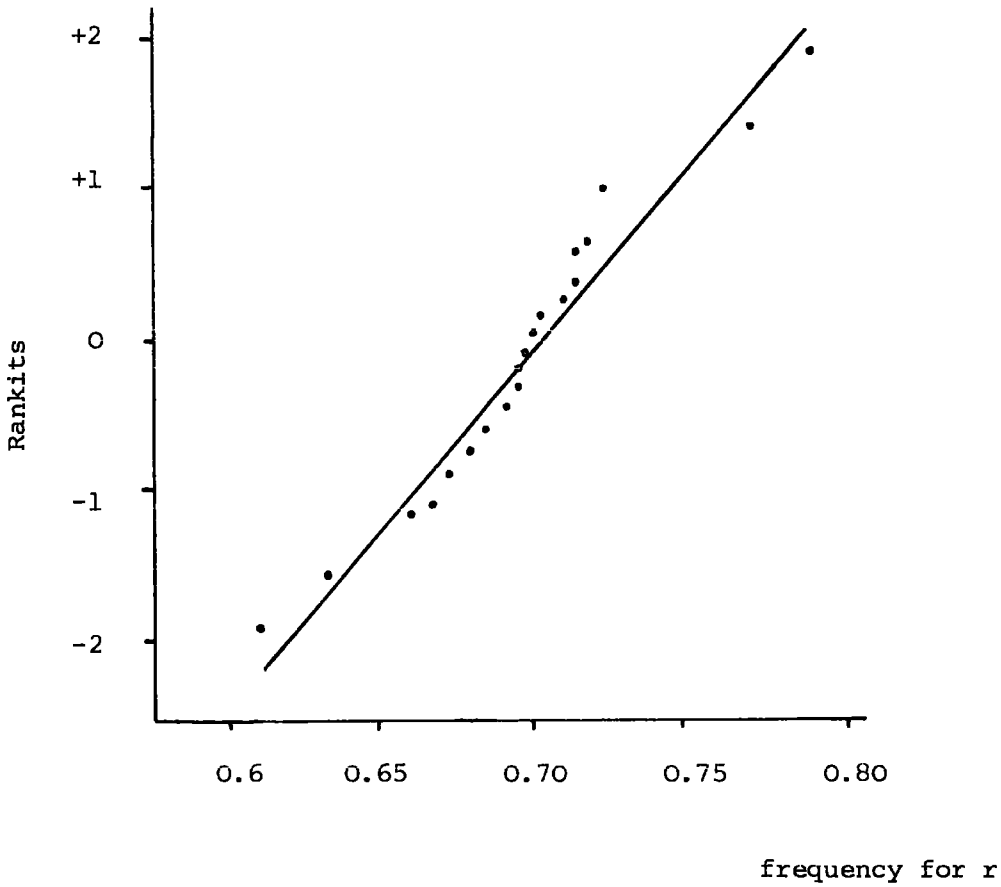


Table 7. ABO gene frequencies - comparable data in the U.K.

Area	Gene frequency			Standard Error	Sample Size
	p	q	r		
1. Nottinghamshire (present study)	0.264	0.068	0.668	±0.014	898
2. Nottinghamshire (Kopec A.C. 1970)	0.257	0.060	0.683	±0.005	6,749
3. Durham (Williams D.R.R. 1977)	0.235	0.063	0.702	±0.020	515
4. N. Northumberland (Cartwright R.A. 1973)	0.190	0.083	0.727	±0.033	144
5. Oxfordshire (Hiorns R.W.*et al.* 1977)	0.277	0.053	0.670	±0.015	805
6. North East	0.239	0.061	0.700	±0.006	6,247
7. Durham	0.220	0.062	0.718	±0.014	1,025
8. Stockton	0.250	0.059	0.691	±0.006	6,735
9. N. Cumberland	0.234	0.054	0.712	±0.009	2,408
10. S. Cumberland	0.233	0.053	0.714	±0.008	3,312
11. Lancaster	0.223	0.061	0.716	±0.001	1,864
12. Lancashire	0.251	0.054	0.695	±0.004	5,578
13. Derbyshire	0.259	0.057	0.684	±0.006	5,995
14. Anglesey	0.238	0.040	0.722	±0.025	314
15. Caernarvon	0.215	0.071	0.714	±0.020	533
16. Swansea	0.258	0.063	0.679	±0.010	2,161
17. Fishguard	0.299	0.083	0.618	±0.015	1,018
18. Tenby	0.294	0.079	0.627	±0.022	463
19. S.W. Scotland (Mitchell R.J. *et al.* 1976)	0.214	0.070	0.716	±0.016	
20. Isle of Man (Mitchell R.J. 1974)	0.266	0.051	0.683	±0.016	
21. N. Ireland (Teesdale and Tills 1970)	0.168	0.056	0.776	±0.018	318
22. Herefordshire (Watkin I.M. 1965)	0.260	0.049	0.691	±0.007	4,525
23. Worcestershire and Warwickshire (Watkin I.M. 1967)	0.269	0.055	0.676	±0.004	11,811

References 6-18 cited in Sunderland E. and Cartwright R.A. (1974)

Reference 21 cited in Mourant A.E. *et al.* (1976)

When comparing the Rh(d) gene frequencies (Table 8) there was a significant difference of 2 standard errors between the Nottinghamshire result and those from Swansea, Derbyshire and Yorkshire; the Nottinghamshire d gene frequency being higher than all the other results, the closest result was that from S.W. Scotland and the Isle of Man. For the other genes in the Rhesus system, comparative material was more scanty. Nottinghamshire had a higher C gene frequency than the other regions (see Table 9) and lay between Durham and North Northumberland for the Rhesus E gene frequencies (see Table 10), but there were no differences of any significance.

As regards the MNSs gene frequencies (see Tables 11 and 12) Nottinghamshire had a slightly lower gene frequency figure for the gene M and a lower gene frequency for the gene S than the other areas. However, there was very little comparative material available.

When comparing Fy gene frequencies (see Table 13) it was found that the combined Nottinghamshire results were higher in Fy^a than those from Scotland and N. Ireland and 2 of the English results, but lower than those from Wales, Oxfordshire and N. Northumberland and Cleghorn's English material.

For the Kell series of gene frequency results (see Table 14) the Nottinghamshire results approximate closest to those from Scotland, Wales, N. Ireland and N. Northumberland, having a slightly higher K gene frequency than the data from Durham, Oxfordshire and England. Again, there were no differences of any significance.

For the only similarly tested P gene frequency data (see 3.35), the P_1 gene frequency was much higher than the other 2 results, being in fact significantly different by 2 standard errors from the Durham result (see Table 15).

Thus for the two major comparative series, it was found that Nottinghamshire had a higher A and B gene frequency and lower O gene

Table 8. Rhesus d gene frequencies - comparable data in the U.K.

Area	Gene frequency d	Standard Error	Sample size
1. Nottinghamshire (Present study)	0.458	±0.016	921
2. Nottinghamshire (Kopec 1970)	0.444	±0.006	6,749
3. Nottinghamshire (Dunsford 1953)	0.425	±0.014	1,206
4. Derbyshire (Dunsford 1953)	0.380	±0.014	1,258
5. Leicestershire (Dunsford 1953)	0.415	±0.010	2,583
6. Lincolnshire (Dunsford 1953)	0.410	±0.018	787
7. Yorkshire (Dunsford 1953)	0.384	±0.016	916
8. Durham (Williams D.R.R. 1977)	0.414	±0.022	515
9. North East	0.444	±0.006	6,247
10. Durham	0.428	±0.015	1,025
11. Stockton	0.427	±0.006	6,735
12. N. Cumberland	0.420	±0.010	2,408
13. S. Cumberland	0.428	±0.009	3,312
14. Lancaster	0.432	±0.012	1,864
15. Lancashire	0.432	±0.004	5,428
16. Derbyshire	0.424	±0.006	5,995
17. Anglesey	0.408	±0.030	270
18. Caernarvon	0.431	±0.026	350
19. Swansea	0.400	±0.011	2,161
20. Fishguard	0.409	±0.015	1,018
21. Tenby	0.397	±0.023	463
22. S.W. Scotland (Mitchell R.J. 1976)	0.453	±0.018	625
23. Isle of Man (Mitchell R.J. 1974)	0.448	±0.020	499

References 3-7 cited in Mourant A.E. *et al.* (1976)

References 9-21 cited in Sunderland E. and Cartwright R.A. (1974)

Table 9. Rhesus C and c gene frequencies - comparable date in the U.K.

Area	Gene frequency C	Gene frequency c	Standard Error	Sample size
1. Nottinghamshire (Present study)	0.441	0.559	±0.020	640
2. Durham (Williams D.R.R. 1977)	0.437	0.563	±0.022	515
3. N. Northumberland (Cartwright R.A. 1973)	0.395	0.605	±0.044	123
4. England (Race *et al.* 1948)	0.422	0.578	±0.015	

Table 10. Rhesus E and e gene frequencies - comparable data in the U.K.

Area	Gene frequency E	Gene frequency e	Standard Error	Sample size
1. Nottinghamshire (Present study)	0.140	0.860	±0.012	910
2. Durham (Williams D.R.R. 1977)	0.153	0.847	±0.016	515
3. N. Northumberland (Cartwright R.A. 1973)	0.138	0.862	±0.031	123
4. England (Race *et al.* 1948)	0.163	0.837	±0.011	

Table 11. MN gene frequencies - comparable data in the U.K.

Area	Gene frequency M	Gene frequency N	Standard Error	Sample size
1. Nottinghamshire (Present study)	0.525	0.475	±0.016	920
2. Durham (Williams D.R.R. 1977)	0.551	0.449	±0.023	515
3. N. Northumberland (Cartwright R.A. 1973)	0.573	0.427	±0.041	144
4. Hertfordshire (Thomas J.C. and Hewitt E.J.C. 1939)	0.552	0.448	±0.017	900
5. Wales (Boyd and Boyd 1937)	0.583	0.417	±0.036	192
6. Belfast (Macafee 1964)	0.550	0.450	±0.035	202

References 5 and 6 cited in Mourant A.E. *et al.* (1976)

Table 12. Ss gene frequencies - comparable data in the U.K.

Area	Gene frequency S	Gene frequency s	Standard Error	Sample size
1. Nottinghamshire (Present study)	0.286	0.714	±0.015	915
2. Durham (Williams D.R.R. 1977)	0.321	0.679	±0.028	515
3. N. Northumberland (Cartwright R.A. 1973)	0.309	0.691	±0.043	115

Table 13. Duffy gene frequencies - comparable data in the U.K.

Area	Gene frequency Fy^a	Gene frequency Fy^b	Standard Error	Sample size
1. Nottinghamshire (present study)	0.431	0.569	±0.018	789
2. Durham (Williams D.R.R. 1977)	0.408	0.592	±0.030	515
3. N. Northumberland (Cartwright R.A. 1973)	0.445	0.555	±0.050	100
4. Oxfordshire (Hiorns R.W. *et al.* 1977)	0.459	0.541	±0.019	679
5. England (Ikin 1954)	0.413	0.587	±0.014	1,166
6. England (Cleghorn)	0.443	0.557	±0.019	656
7. England (Race 1968)	0.407	0.593	±0.031	250
8. Scotland (Ikin 1954)	0.424	0.576	±0.022	527
9. Wales (Ikin 1954)	0.451	0.549	±0.046	116
10. N. Ireland (Teesdale and Tills 1970)	0.412	0.588	±0.028	318

References 5-10 cited in Mourant *et al.* (1976)

Table 14. Kell gene frequencies - comparable data in the U.K.

Area	Gene frequencies K	Gene frequencies k	Standard Error	Sample size
1. Nottinghamshire (Present study)	0.043	0.957	±0.007	916
2. Durham (Williams D.R.R. 1977)	0.035	0.965	±0.008	515
3. N. Northumberland (Cartwright R.A. 1973)	0.042	0.958	±0.017	144
4. Oxfordshire (Hiorns R.W. *et al.* 1977)	0.036	0.964	±0.005	652
5. England (Ikin *et al.* 1954)	0.039	0.961	±0.006	1,166
6. Scotland (Ikin *et al.* 1954)	0.045	0.955	±0.009	527
7. Wales (Ikin *et al.* 1954)	0.043	0.957	±0.019	116
8. N. Ireland (Teesdale and Tills 1970)	0.045	0.955	±0.012	319

References 5-8 cited in Mourant A.E. *et al.* (1976)

Table 15. P gene frequencies - comparable data in the U.K.

Area	Gene frequencies P_1	Gene frequencies $P_2 + p$	Standard Error	Sample size
1. Nottinghamshire (Present study)	0.614	0.386	±0.016	919
2. Durham (Williams D.R.R. 1977)	0.499	0.502	±0.022	515
3. N. Northumberland (Cartwright R.A. 1973)	0.551	0.449	±0.041	144

frequency than most other areas, in fact Sheffield B.T.S. had noted a higher percentage of A donors in their donor panel. Also, Nottinghamshire exhibited a higher Rh(d) gene frequency.

5.4 Further research considerations.

Comparison of the Nottinghamshire data with that from other available series shows that genetic variability does exist between the different geographical regions, but comparison on the basis of differences in standard errors between gene frequencies is very limiting, for standard errors are only useful when one genetic system is being considered at any one time. Distance statistics may be used in an attempt to find underlying patterns amongst arrays of genetic data. Distance statistics combine all the gene frequencies from one population sample in order to effect comparisons with similar data from another population in pairwise fashion, and there are various ways in which distance statistics may be computed (see Sunderland E. and Cartwright R.A. 1974). However, distance statistics have two disadvantages; first the results are usually in several dimensions, and secondly they lack the usual forms of significance test.

It would be profitable to analyse the Nottinghamshire results by using distance statistics and to explore the interrelationships between them and the other series of comparative material available. Other comparisons which might be undertaken would be to investigate the differences between those born in the county and those born elsewhere, the differences in gene frequencies between those who move type of residential area between birth and the present time and of those who do not move between the three settlement areas, and the differences between the gene frequencies at birth and at the time of the survey in the three types of residence.

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Appendix A

Gene frequency distribution by sex of donor

	NOTTM.			EDWIN.			NOTTM. + EDWIN.		
	Male	Female	Both	Male	Female	Both	Male	Female	Both
pl	0.192± 0.020	0.206± 0.023	0.198± 0.015	0.243± 0.040	0.271± 0.050	0.255± 0.031	0.204± 0.018	0.218± 0.021	0.210± 0.014
p2	0.066	0.056	0.062	0.018	0.037	0.025	0.055	0.052	0.054
q	0.060	0.066	0.062	0.074	0.104	0.086	0.063	0.073	0.068
r	0.682	0.672	0.678	0.665	0.588	0.634	0.678	0.657	0.668
No.tested	406	302	708	113	77	190	519	379	898
M	0.513± 0.024	0.523± 0.028	0.518± 0.019	0.531± 0.047	0.585± 0.056	0.553± 0.036	0.517± 0.022	0.535± 0.025	0.525± 0.016
N	0.487	0.477	0.482	0.469	0.415	0.447	0.483	0.465	0.475
No.tested	419	311	730	113	77	190	532	388	920
S	0.279± 0.022	0.309± 0.026	0.292± 0.017	0.261± 0.041	0.273± 0.051	0.265± 0.032	0.276± 0.019	0.302± 0.076	0.286± 0.015
s	0.721	0.691	0.708	0.739	0.727	0.735	0.724	0.698	0.714
No.tested	415	310	725	113	77	190	528	387	215
D	0.561± 0.024	0.543± 0.028	0.553± 0.019	0.494± 0.047	0.517± 0.057	0.504± 0.036	0.547± 0.022	0.538± 0.025	0.542± 0.016
d	0.439	0.457	0.447	0.506	0.483	0.496	0.453	0.462	0.458
No.tested	420	311	731	113	77	190	533	388	921
C	0.447± 0.029	0.438± 0.035	0.444± 0.022	0.380± 0.057	0.372± 0.072	0.376± 0.045	0.433± 0.026	0.358± 0.029	0.440± 0.020
C^W	0.012	0.007	0.009	0.014	0.011	0.013	0.012	0.075	0.001
c	0.541	0.555	0.547	0.606	0.617	0.611	0.555	0.567	0.559
No.tested	303	220	523	72	45	117	375	265	640
E	0.130± 0.017	0.143± 0.020	0.136± 0.013	0.165± 0.035	0.136± 0.039	0.153± 0.026	0.139± 0.015	0.142± 0.018	0.140± 0.012
e	0.870	0.857	0.864	0.835	0.864	0.847	0.163	0.858	0.860
No.tested	415	306	721	112	77	189	527	383	910
Fya	0.462± 0.027	0.412± 0.031	0.441± 0.020	0.429± 0.047	0.355± 0.055	0.399± 0.036	0.454± 0.023	0.399± 0.027	0.431± 0.018
Fyb	0.538	0.588	0.559	0.571	0.645	0.601	0.546	0.601	0.569
No.tested	344	256	600	113	76	189	457	332	789
K	0.037± 0.009	0.058± 0.010	0.045± 0.007	0.035± 0.017	0.032± 0.020	0.034± 0.013	0.037± 0.008	0.052± 0.011	0.043± 0.007
k	0.963	0.942	0.955	0.965	0.968	0.966	0.963	0.948	0.957
No.tested	419	307	726	113	77	190	532	384	916
Kpa+	0.0	0.0	0.0	0.013± 0.013	0.0	0.009± 0.008	0.006± 0.006	0.015± 0.011	0.010± 0.006
Kpa-	0.0	0.0	0.0	0.987	1.00	0.991	0.994	0.985	0.990
No. tested	0	0	0	81	46	127	180	103	283

Gene frequency distribution by sex of donor (Continued)

	NOTTM.			EDWIN.			NOTTM + EDWIN.		
	Male	Female	Both	Male	Female	Both	Male	Female	Both
Lea+	0.189± 0.043	0.104± 0.035	0.147± 0.028	0.182± 0.033	0.148± 0.045	0.169± 0.030	1.185± 0.029	0.124± 0.028	0.158± 0.020
Lea-	0.811	0.896	0.853	0.818	0.852	0.831	0.815	0.876	0.842
No.tested	82	76	158	97	62	159	179	138	317
P+	0.600± 0.024	0.611± 0.028	0.606± 0.018	0.689± 0.044	0.606± 0.056	0.653± 0.035	0.617± 0.021	0.611± 0.025	0.614± 0.016
P-	0.400	0.389	0.394	0.311	0.394	0.347	0.383	0.389	0.386
No.tested	419	310	729	113	77	190	532	387	919

Gene frequency distribution by area of residence of donor

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
p1	0.206± 0.021	0.201± 0.027	0.177± 0.037	0.205± 0.021	0.201± 0.027	0.226± 0.024
p2	0.049	0.070	0.065	0.049	0.070	0.039
q	0.071	0.052	0.063	0.071	0.052	0.077
r	0.674	0.677	0.695	0.675	0.677	0.658
No.tested	383	226	106	384	226	299
M	0.516± 0.025	0.544± 0.033	0.455± 0.047	0.516± 0.025	0.544± 0.033	0.513± 0.029
N	0.484	0.456	0.545	0.484	0.456	0.487
No.tested	396	230	112	397	230	305
S	0.305± 0.023	0.292± 0.030	0.236± 0.040	0.306± 0.023	0.292± 0.030	0.254± 0.025
s	0.695	0.708	0.764	0.694	0.708	0.746
No.tested	393	228	112	394	228	305
D	0.566± 0.025	0.522± 0.033	0.567± 0.047	0.566± 0.025	0.522± 0.033	0.528± 0.029
d	0.434	0.478	0.433	0.434	0.478	0.472
No.tested	396	231	112	397	231	305
C	0.468± 0.029	0.388± 0.040	0.479± 0.054	0.468± 0.029	0.388± 0.040	0.414± 0.034
C^W	0.008	0.010	0.012	0.008	0.010	0.012
c	0.524	0.602	0.509	0.524	0.602	0.574
No.tested	288	152	86	289	152	204
E	0.125± 0.017	0.167± 0.025	0.125± 0.031	0.126± 0.017	0.167± 0.025	0.143± 0.020
e	0.875	0.833	0.875	0.874	0.833	0.857
No.tested	392	224	112	393	224	304
Fya	0.446± 0.028	0.460± 0.036	0.406± 0.051	0.444± 0.028	0.460± 0.036	0.401± 0.029
Fyb	0.554	0.540	0.594	0.556	0.540	0.599
No.tested	324	191	91	325	191	283
K	0.044± 0.010	0.045± 0.014	0.040± 0.019	0.044± 0.010	0.045± 0.014	0.036± 0.011
k	0.956	0.955	0.960	0.956	0.955	0.964
No.tested	393	231	111	394	231	304
Kpa+	0.019± 0.015	0.0	0.0	0.019± 0.015	0.0	0.007± 0.007
Kpa-	0.981	1.0	1.0	0.981	1.0	0.993
No.tested	80	47	27	81	47	157

Gene frequency distribution by area of residence of donor (contd.)

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
Lea+	0.176± 0.037	0.124± 0.044	0.183± 0.091	0.174± 0.037	0.124± 0.044	0.167± 0.028
Lea-	0.824	0.876	0.817	0.826	0.876	0.833
No.tested	106	56	18	107	56	180
P+	0.620± 0.024	0.600± 0.032	0.588± 0.047	0.621± 0.024	0.600± 0.032	0.629± 0.028
P-	0.380	0.400	0.412	0.379	0.400	0.371
No.tested	394	231	112	395	231	305

Gene frequency distribution by type of residence of donor

	NOTTM.			NOTTM. + EDWIN		
	Pit	Rural	Urban	Pit	Rural	Urban
pl	0.089± 0.044	0.216± 0.053	0.204± 0.016	0.226± 0.028	0.225± 0.049	0.204± 0.016
p2	0.112	0.055	0.057	0.037	0.045	0.057
q	0.089	0.051	0.064	0.085	0.057	0.065
r	0.710	0.678	0.675	0.652	0.673	0.674
No.tested	42	60	611	216	73	616
M	0.386± 0.073	0.500± 0.063	0.526± 0.020	0.514± 0.034	0.520± 0.057	0.529± 0.020
N	0.614	0.500	0.474	0.486	0.480	0.473
No.tested	44	64	628	218	77	633
S	0.227± 0.063	0.257± 0.055	0.299± 0.018	0.268± 0.030	0.240± 0.049	0.298± 0.018
s	0.773	0.743	0.701	0.732	0.760	0.702
No.tested	44	64	623	218	77	628
D	0.574± 0.075	0.550± 0.062	0.551± 0.020	0.512± 0.034	0.544± 0.057	0.553± 0.020
d	0.426	0.450	0.449	0.488	0.456	0.447
No.tested	44	64	629	218	77	634
C	0.523± 0.084	0.448± 0.073	0.439± 0.024	0.396± 0.041	0.456± 0.067	0.439± 0.023
C^W	0.0	0.021	0.009	0.007	0.018	0.009
c	0.477	0.531	0.552	0.597	0.526	0.552
No.tested	35	47	443	141	55	446
E	0.113± 0.048	0.125± 0.041	0.140± 0.014	0.152± 0.024	0.110± 0.036	0.142± 0.014
e	0.887	0.875	0.860	0.848	0.890	0.858
No.tested	44	64	618	217	77	623
Fya	0.391± 0.080	0.411± 0.069	0.452± 0.022	0.404± 0.034	0.390± 0.061	0.451± 0.022
Fyb	0.608	0.589	0.548	0.596	0.610	0.549
No.tested	37	51	518	210	64	522
K	0.011± 0.016	0.063± 0.031	0.044± 0.008	0.032± 0.012	0.052± 0.025	0.044± 0.008
k	0.989	0.937	0.956	0.968	0.948	0.956
No.tested	44	63	626	218	76	631
Kpa+	0.0	0.0	0.012± 0.010	0.004± 0.005	0.025± 0.035	0.012± 0.010
Kpa-	1.0	1.0	0.988	0.996	0.975	0.988
No.tested	14	13	127	132	20	131

Gene frequency distribution by type of residence of donor (contd.)

	NOTTM.			NOTTM. + EDWIN.		
	Pit	Rural	Urban	Pit	Rural	Urban
Lea+	0.183± 0.158	0.147± 0.107	0.161± 0.029	0.165± 0.030	0.174± 0.081	0.159± 0.028
Lea-	0.817	0.853	0.839	0.835	0.826	0.841
No.tested	6	11	164	152	22	167
P+	0.663± 0.071	0.550± 0.062	0.616± 0.019	0.669± 0.032	0.544± 0.057	0.613± 0.019
P-	0.337	0.450	0.384	0.331	0.456	0.387
No.tested	44	64	627	218	77	632

Gene frequency distribution for Nottingham+ Edwinstone by occupation of donor

	Professional	Intermediate	Skilled Non-Manual	Skilled Manual	Part-Skilled	Unskilled
p1	0.032± 0.044	0.205± 0.030	0.233± 0.030	0.207± 0.024	0.187± 0.041	0.317± 0.065
p2	0.137	0.070	0.040	0.056	0.041	0.015
q	0.098	0.061	0.082	0.063	0.075	0.073
r	0.783	0.664	0.645	0.674	0.697	0.595
No.tested	16	178	201	296	92	51
M	0.563± 0.124	0.563± 0.037	0.475± 0.035	0.528± 0.029	0.538± 0.052	0.539± 0.069
N	0.437	0.437	0.525	0.472	0.462	0.461
No.tested	16	184	204	305	93	52
S	0.532± 0.125	0.288± 0.034	0.255± 0.031	0.280± 0.026	0.342± 0.049	0.308± 0.064
s	0.468	0.712	0.745	0.720	0.658	0.692
No.tested	16	182	204	303	92	52
D	0.752± 0.108	0.500± 0.037	0.522± 0.035	0.561± 0.028	0.504± 0.052	0.661± 0.066
d	0.248	0.500	0.478	0.439	0.496	0.339
No.tested	16	184	205	305	93	52
C	0.500± 0.144	0.389± 0.045	0.410± 0.042	0.457± 0.034	0.407± 0.061	0.490± 0.080
C^W	0.0	0.012	0.007	0.007	0.039	0.0
c	0.500	0.598	0.583	0.536	0.554	0.510
No.tested	12	120	136	215	64	39
E	0.156± 0.091	0.151± 0.027	0.135± 0.024	0.140± 0.020	0.088± 0.030	0.160± 0.052
e	0.844	0.849	0.865	0.860	0.912	0.840
No.tested	16	182	204	303	91	50
Fya	0.428± 0.132	0.465± 0.040	0.430± 0.037	0.394± 0.031	0.482± 0.056	0.488± 0.077
Fyb	0.572	0.535	0.570	0.606	0.518	0.512
No.tested	14	157	180	256	81	42
K	0.031± 0.043	0.033± 0.013	0.037± 0.013	0.045± 0.012	0.043± 0.021	0.057± 0.032
k	0.969	0.969	0.963	0.955	0.957	0.943
No.tested	16	183	203	305	92	52
Kpa+		0.0	0.008± 0.011	0.014± 0.011	0.0	0.031± 0.043
Kpa-		1.0	0.992	0.986	1.0	0.969
No.tested		50	63	110	34	16

Gene frequency distribution for Nottingham + Edwinstone

by occupation of donor (contd.)

	Professional	Intermediate	Skilled Non-Manual	Skilled Manual	Part-Skilled	Unskilled
Lea+		0.183± 0.054	0.132± 0.041	0.156± 0.033	0.244± 0.081	0.078± 0.060
Lea-		0.817	0.868	0.844	0.756	0.922
No.tested	51	69	122	28	20	
P+	0.567± 0.124	0.625± 0.036	0.637± 0.034	0.603± 0.028	0.560± 0.051	0.585± 0.068
P-	0.433	0.375	0.363	0.397	0.440	0.415
No.tested	16	184	205	303	93	52

Gene frequency distribution for Nottingham + Edwinstone

by occupation of donor's father

	Professional	Intermediate	Skilled Non-Manual	Skilled Manual	Part-Skilled	Unskilled
pl	0.096± 0.089	0.157± 0.038	0.216± 0.044	0.220± 0.020	0.225± 0.040	0.215± 0.050
p2	0.052	0.076	0.052	0.048	0.035	0.079
q	0.047	0.058	0.058	0.081	0.075	0.054
r	0.805	0.709	0.674	0.651	0.665	0.652
No.tested	11	90	89	425	110	68
M	0.454± 0.150	0.462± 0.052	0.522± 0.052	0.538± 0.024	0.527± 0.047	0.521± 0.059
N	0.546	0.538	0.478	0.462	0.473	0.479
No.tested	11	93	91	436	111	71
S	0.350± 0.151	0.231± 0.044	0.286± 0.047	0.281± 0.022	0.326± 0.045	0.316± 0.055
s	0.650	0.769	0.714	0.719	0.674	0.684
No.tested	10	93	91	436	109	71
D	0.478± 0.151	0.626± 0.050	0.509± 0.052	0.522± 0.024	0.526± 0.047	0.573± 0.059
d	0.522	0.374	0.491	0.478	0.474	0.427
No.tested	11	93	91	437	111	71
C	0.545± 0.176	0.480± 0.059	0.415± 0.062	0.425± 0.029	0.374± 0.056	0.446± 0.068
C^W	0.0	0.014	0.008	0.006	0.013	0.019
c	0.455	0.506	0.577	0.569	0.613	0.535
No.tested	8	72	63	289	75	53
E	0.045± 0.063	0.151± 0.037	0.111± 0.033	0.136± 0.016	0.173± 0.036	0.127± 0.040
e	0.955	0.849	0.889	0.864	0.827	0.873
No.tested	11	93	90	433	110	71
Fya	0.318± 0.140	0.367± 0.054	0.468± 0.056	0.446± 0.026	0.435± 0.051	0.491± 0.067
Fyb	0.682	0.633	0.532	0.554	0.565	0.509
No.tested	11	79	80	372	93	56
K	0.141± 0.105	0.032± 0.018	0.039± 0.020	0.044± 0.010	0.051± 0.021	0.049± 0.026
k	0.959	0.968	0.961	0.956	0.949	0.951
No.tested	11	93	90	437	108	71
Kpa+		0.0	0.019± 0.026	0.012± 0.008	0.0	0.0
Kpa-		1.0	0.981	0.988	1.0	1.0
No.tested		29	27	169	28	18

Gene frequency distribution for Nottingham + Edwinstone

by occupation of donor's father (contd.)

	Professional	Intermediate	Skilled Non-Manual	Skilled Manual	Part-Skilled	Unskilled
Lea+		0.220± 0.086	0.123± 0.062	0.160± 0.029	0.069± 0.046	0.225± 0.093
Lea-		0.780	0.887	0.840	0.931	0.775
No.tested		23	28	160	30	20
P+	0.574± 0.149	0.560± 0.051	0.704± 0.048	0.626± 0.023	0.576± 0.047	0.573± 0.059
P-	0.426	0.440	0.296	0.374	0.424	0.427
No.tested	11	93	91	436	111	71

Gene frequency distribution by area of birthplace of donor

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
p1	0.220± 0.025	0.239± 0.051	0.165± 0.019	0.219± 0.025	0.230± 0.051	0.259± 0.019
p2	0.069	0.061	0.102	0.069	0.059	0.053
q	0.068	0.039	0.074	0.069	0.037	0.057
r	0.643	0.661	0.659	0.643	0.674	0.631
No.tested	269	67	57	272	69	172
M	0.512± 0.029	0.537± 0.061	0.482± 0.066	0.517± 0.029	0.528± 0.060	0.520± 0.040
N	0.488	0.468	0.518	0.483	0.472	0.480
No.tested	281	68	58	284	70	173
S	0.309± 0.027	0.242± 0.052	0.267± 0.058	0.310± 0.028	0.236± 0.051	0.265± 0.035
s	0.691	0.758	0.733	0.690	0.764	0.735
No.tested	278	68	58	281	70	173
D	0.558± 0.023	0.486± 0.061	0.607± 0.064	0.560± 0.029	0.494± 0.060	0.544± 0.038
d	0.442	0.514	0.393	0.440	0.506	0.456
No.tested	281	68	58	284	70	173
C	0.482± 0.035	0.411± 0.073	0.508± 0.075	0.480± 0.035	0.414± 0.072	0.427± 0.047
C^W	0.012	0.011	0.OLL	0.012	0.010	0.014
c	0.516	0.588	0.491	0.518	0.586	0.729
No.tested	205	45	45	208	47	114
E	0.113± 0.018	0.159± 0.044	0.123± 0.044	0.114± 0.019	0.151± 0.043	0.146± 0.072
e	0.887	0.843	0.877	0.886	0.849	0.854
No.tested	278	67	57	281	69	171
Fya	0.420± 0.033	0.509± 0.065	0.416± 0.071	0.414± 0.032	0.500± 0.064	0.428± 0.039
Fyb	0.580	0.491	0.584	0.586	0.500	0.577
No.tested	230	59	48	233	61	163
K	0.033± 0.011	0.030± 0.021	0.079± 0.036	0.032± 0.011	0.028± 0.020	0.050± 0.017
k	0.967	0.970	0.921	0.968	0.972	0.950
No.tested	278	68	57	281	70	172
Kpa+	0.025± 0.020	0.0	0.0	0.025± 0.020	0.0	0.006± 0.008
Kpa-	0.975	1.0	1.0	0.975	1.0	0.994
No.tested	62	10	15	63	12	95

Gene frequency distribution by area of birthplace of donor (continued)

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
Lea+	0.154± 0.044	0.156± 0.079	0.156± 0.137	0.149± 0.042	0.141± 0.073	0.184± 0.037
Lea-	0.846	0.844	0.844	0.851	0.859	0.816
No.tested	69	21	7	71	23	111
P+	0.628± 0.029	0.500± 0.061	0.586± 0.066	0.620± 0.029	0.494± 0.029	0.620± 0.037
P-	0.372	0.500	0.414	0.380	0.506	0.380
No.tested	280	68	58	283	70	173

Gene frequency distribution by type of birthplace of donor

	NOTTM.			NOTTM. + EDWIN		
	Pit	Rural	Urban	Pit	Rural	Urban
p1	0.162± 0.071	0.165± 0.072	0.220± 0.022	0.284± 0.040	0.181± 0.063	0.219± 0.022
p2	0.095	0.096	0.069	0.033	0.089	0.069
q	0.038	0.121	0.061	0.040	0.132	0.061
r	0.705	0.618	0.650	0.643	0.598	0.651
No.tested	27	27	341	131	37	347
M	0.536± 0.094	0.444± 0.056	0.515± 0.027	0.526± 0.043	0.500± 0.082	0.520± 0.026
N	0.464	0.556	0.485	0.474	0.500	0.480
No.tested	28	27	354	132	37	360
S	0.321± 0.088	0.222± 0.080	0.295± 0.023	0.272± 0.039	0.256± 0.071	0.292± 0.024
s	0.679	0.778	0.705	0.728	0.744	0.708
No.tested	28	27	351	132	37	357
D	0.622± 0.092	0.570± 0.095	0.547± 0.026	0.524± 0.044	0.598± 0.081	0.550± 0.026
d	0.378	0.430	0.453	0.476	0.402	0.450
No.tested	28	27	354	132	37	360
C	0.572± 0.105	0.439± 0.108	0.455± 0.031	0.414± 0.054	0.439± 0.092	0.456± 0.031
C^W	0.0	0.024	0.012	0.006	0.034	0.011
c	0.428	0.537	0.533	0.580	0.527	0.533
No.tested	22	21	254	83	29	259
E	0.107± 0.058	0.115± 0.063	0.128± 0.018	0.149± 0.031	0.097± 0.049	0.125± 0.018
e	0.893	0.885	0.877	0.851	0.903	0.875
No.tested	28	26	350	131	36	356
Fya	0.369± 0.101	0.477± 0.106	0.435± 0.029	0.421± 0.044	0.453± 0.088	0.428± 0.029
Fyb	0.631	0.523	0.565	0.579	0.547	0.572
No.tested	23	22	294	127	32	300
K	0.018± 0.025	0.134± 0.067	0.033± 0.009	0.031± 0.015	0.112± 0.053	0.033± 0.009
k	0.982	0.866	0.967	0.969	0.888	0.967
No. tested	28	26	351	132	36	357
Kpa+	0.0	0.0	0.021± 0.017	0.007± 0.009	0.0	0.020± 0.016
Kpa-	1.0	1.0	0.979	0.993	1.0	0.980
No.tested	9	5	73	85	9	76

Gene frequency distribution by type of birthplace of donor (continued)

	NOTTM.			NOTTM. + EDWIN.		
	Pit	Rural	Urban	Pit	Rural	Urban
Lea+	0.134± 0.170	0.184± 0.224	0.154± 0.038	0.194± 0.040	0.106± 0.097	0.145± 0.036
Lea-	0.866	0.816	0.846	0.806	0.894	0.855
No.tested	4	3	90	100	10	95
P+	0.622± 0.092	0.570± 0.095	0.599± 0.026	0.641± 0.042	0.566± 0.082	0.592± 0.026
P-	0.378	0.430	0.401	0.359	0.434	0.408
No.tested	28	27	353	132	37	359

Gene frequency distribution by area of birthplace of donor's spouse

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
pl	0.200± 0.033	0.132± 0.050	0.150± 0.054	0.203± 0.033	0.129± 0.049	0.228± 0.038
p2	0.090	0.067	0.042	0.089	0.065	0.026
q	0.089	0.070	0.123	0.088	0.069	0.089
r	0.621	0.731	0.685	0.620	0.737	0.657
No.tested	148	45	44	149	46	125
M	0.484± 0.040	0.511± 0.074	0.522± 0.074	0.487± 0.040	0.511± 0.073	0.528± 0.044
N	0.516	0.489	0.478	0.513	0.489	0.472
No.tested	154	46	46	155	47	127
S	0.288± 0.036	0.315± 0.068	0.206± 0.060	0.287± 0.036	0.308± 0.067	0.252± 0.039
s	0.712	0.685	0.794	0.713	0.692	0.748
No.tested	154	46	46	155	47	127
D	0.589± 0.040	0.640± 0.071	0.489± 0.074	0.591± 0.039	0.643± 0.070	0.539± 0.044
d	0.411	0.360	0.511	0.409	0.357	0.461
No.tested	154	46	46	155	47	127
C	0.444± 0.046	0.464± 0.084	0.456± 0.089	0.445± 0.046	0.464± 0.083	0.433± 0.054
C^W	0.013	0.014	0.0	0.013	0.014	0.0
c	0.543	0.522	0.544	0.542	0.522	0.567
No.tested	115	35	31	116	36	84
E	0.127± 0.027	0.193± 0.059	0.087± 0.042	0.135± 0.028	0.189± 0.058	0.151± 0.032
e	0.863	0.807	0.913	0.865	0.811	0.849
No.tested	150	44	46	151	45	126
Fya	0.418± 0.044	0.486± 0.082	0.425± 0.078	0.414± 0.043	0.477± 0.081	0.429± 0.045
Fyb	0.582	0.514	0.575	0.586	0.503	0.571
No.tested	128	37	40	129	38	121
K	0.035± 0.015	0.054± 0.033	0.021± 0.021	0.035± 0.015	0.053± 0.033	0.039± 0.017
k	0.965	0.946	0.979	0.965	0.947	0.961
No.tested	154	46	46	155	47	127
Kpa+	0.026± 0.025	0.0	0.0	0.026± 0.025	0.0	0.008± 0.011
Kpa-	0.974	1.0	1.0	0.974	1.0	0.992
No.tested	39	10	4	39	11	66

Gene frequency distribution by area of birthplace of donor's spouse (contd.)

Lea+	0.134± 0.051	0.118± 0.108	0.057± 0.077	0.130± 0.050	0.106± 0.097	0.173± 0.043
Lea-	0.866	0.882	0.943	0.870	0.894	0.827
No.tested	44	9	9	45	10	79
P+	0.572± 0.040	0.670± 0.069	0.583± 0.073	0.566± 0.040	0.674± 0.068	0.624± 0.043
P-	0.428	0.330	0.417	0.434	0.326	0.376
No.tested	153	46	46	154	47	127

Gene frequency distribution by type of birthplace of donor's spouse

	NOTTM.			NOTTM. + EDWIN.		
	Pit	Rural	Urban	Pit	Rural	Urban
pl	0.164± 0.071	0.116± 0.086	0.183± 0.028	0.248± 0.044	0.149± 0.070	0.186± 0.028
p2	0.047	0.041	0.082	0.021	0.047	0.081
q	0.120	0.115	0.085	0.077	0.126	0.084
r	0.669	0.728	0.650	0.654	0.678	0.649
No.tested	27	14	197	95	26	200
M	0.518± 0.094	0.567± 0.128	0.485± 0.035	0.526± 0.051	0.574± 0.095	0.485± 0.035
N	0.482	0.433	0.515	0.474	0.426	0.515
No.tested	28	15	204	96	27	207
S	0.196± 0.075	0.233± 0.109	0.291± 0.032	0.276± 0.046	0.185± 0.075	0.288± 0.031
s	0.804	0.767	0.709	0.724	0.815	0.712
No.tested	28	15	204	96	27	207
D	0.500± 0.094	0.423± 0.128	0.604± 0.034	0.555± 0.050	0.456± 0.096	0.606± 0.034
d	0.500	0.577	0.396	0.445	0.544	0.394
No.tested	28	15	204	96	27	207
C	0.464± 0.114	0.400± 0.166	0.453± 0.040	0.411± 0.062	0.463± 0.012	0.456± 0.039
C^W	0.0	0.0	0.013	0.0	0.0	0.012
c	0.536	0.600	0.534	0.589	0.537	0.532
No.tested	19	9	154	63	17	157
E	0.125± 0.063	0.033± 0.046	0.146± 0.025	0.179± 0.039	0.074± 0.051	0.144± 0.025
e	0.875	0.967	0.854	0.821	0.926	0.856
No.tested	28	15	198	95	27	201
Fya	0.374± 0.099	0.539± 0.138	0.432± 0.038	0.418± 0.051	0.500± 0.100	0.427± 0.038
Fyb	0.626	0.461	0.568	0.582	0.500	0.573
No.tested	24	13	169	95	25	172
K	0.0	0.066± 0.064	0.039± 0.014	0.041± 0.020	0.037± 0.036	0.039± 0.014
k	1.00	0.934	0.961	0.959	0.963	0.961
No.tested	28	15	204	96	27	207
Kpa+	0.0	0.0	0.021± 0.021	0.009± 0.013	0.0	0.020± 0.019
Kpa-	1.0	1.0	0.979	0.991	1.0	0.980
No.tested	12	2	49	58	8	50

Gene frequency distribution by type of birthplace of donor's spouse (contd.)

Lea+	0.0	0.183± 0.223	0.129± 0.046	0.186± 0.048	0.134± 0.098	0.121± 0.043
Lea-	1.0	0.817	0.871	0.814	0.866	0.879
No.tested	5	3	54	65	12	57
P+	0.622 0.092	0.553± 0.128	0.591± 0.035	0.646± 0.049	0.570± 0.095	0.588± 0.034
P-	0.378	0.447	0.409	0.354	0.430	0.412
No.tested	28	15	203	96	27	206

Gene frequency distribution by area of birthplace of donor's father

	NOTTM.			NOTTM + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
pl	0.229± 0.036	0.141± 0.049	0.205± 0.061	0.228± 0.037	0.141± 0.049	0.221± 0.042
p2	0.067	0.074	0.061	0.066	0.074	0.034
q	0.072	0.052	0.109	0.075	0.052	0.075
r	0.632	0.733	0.625	0.631	0.733	0.670
No.tested	131	50	44	132	50	97
M	0.511± 0.044	0.462± 0.069	0.489± 0.075	0.515± 0.043	0.462± 0.068	0.505± 0.051
N	0.489	0.538	0.511	0.485	0.538	0.495
No.tested	132	53	45	133	53	98
S	0.389± 0.042	0.245± 0.059	0.227± 0.063	0.341± 0.041	0.245± 0.059	0.262± 0.042
s	0.661	0.755	0.773	0.659	0.755	0.738
No.tested	131	53	44	132	53	97
D	0.565± 0.043	0.611± 0.067	0.605± 0.073	0.566± 0.043	0.611± 0.067	0.596± 0.049
d	0.435	0.389	0.395	0.434	0.389	0.404
No.tested	132	53	45	133	53	98
C	0.471± 0.051	0.471± 0.078	0.534± 0.084	0.481± 0.051	0.471± 0.078	0.466± 0.059
C^W	0.010	0.0	0.0	0.010	0.0	0.014
c	0.519	0.529	0.466	0.519	0.529	0.521
No.tested	97	41	35	98	41	70
E	0.095± 0.026	0.130± 0.048	0.122± 0.048	0.094± 0.025	0.130± 0.048	0.158± 0.037
e	0.905	0.870	0.878	0.906	0.780	0.842
No.tested	132	53	45	133	53	98
Fya	0.444± 0.048	0.442± 0.076	0.404± 0.076	0.440± 0.048	0.442± 0.076	0.421± 0.051
Fyb	0.556	0.558	0.596	0.560	0.558	0.579
No.tested	108	43	42	109	43	95
K	0.038± 0.017	0.056± 0.032	0.057± 0.035	0.038± 0.016	0.056± 0.032	0.041± 0.020
k	0.962	0.944	0.943	0.962	0.944	0.959
No.tested	131	53	44	132	53	97
Kpa+	0.033± 0.032	0.0	0.0	0.033± 0.032	0.0	0.009± 0.013
Kpa-	0.967	1.0	1.0	0.967	1.0	0.991
No.tested	31	13	15	31	13	56

Gene frequency distribution by area of birthplace of donor's father (contd.)

Lea+	0.120± 0.051	0.047± 0.064	0.163± 0.120	0.120± 0.051	0.047± 0.064	0.149± 0.050
Lea-	0.880	0.953	0.837	0.880	0.953	0.851
No.tested	40	11	10	40	11	58
P+	0.590± 0.043	0.588± 0.068	0.553± 0.046	0.591± 0.043	0.588± 0.054	0.571± 0.049
P-	0.410	0.412	0.447	0.409	0.412	0.429
No.tested	131	53	45	132	53	98

Gene frequency distribution by type of birthplace of donor's father

	NOTTM.			NOTTM. + EDWIN.		
	Pit	Rural	Urban	Pit	Rural	Urban
pl	0.155± 0.073	0.232± 0.100	0.198± 0.029	0.203± 0.052	0.250± 0.074	0.200± 0.023
p2	0.049	0.081	0.065	0.021	0.040	0.070
q	0.132	0.093	0.122	0.062	0.108	0.067
r	0.664	0.593	0.615	0.714	0.602	0.663
No.tested	25	17	184	58	34	187
M	0.480± 0.099	0.500± 0.120	0.498± 0.036	0.500± 0.066	0.500± 0.085	0.503± 0.036
N	0.520	0.500	0.502	0.500	0.500	0.497
No.tested	25	18	188	58	35	191
S	0.188± 0.080	0.305± 0.110	0.309± 0.034	0.237± 0.056	0.300± 0.068	0.311± 0.034
s	0.812	0.695	0.693	0.763	0.700	0.689
No.tested	24	18	187	57	35	190
D	0.553± 0.094	0.763± 0.100	0.574± 0.036	0.545± 0.065	0.830± 0.064	0.572± 0.036
d	0.447	0.237	0.426	0.455	0.170	0.428
No.tested	25	18	188	58	35	191
C	0.520± 0.120	0.556± 0.099	0.474± 0.042	0.448± 0.082	0.571± 0.087	0.469± 0.042
C^W	0.0	0.0	0.007	0.0	0.031	0.007
c	0.480	0.444	0.519	0.552	0.428	0.524
No.tested	17	17	140	37	32	141
E	0.120± 0.065	0.139± 0.082	0.105± 0.023	0.155± 0.047	0.157± 0.062	0.409± 0.023
e	0.880	0.861	0.895	0.845	0.843	0.891
No.tested	25	18	185	58	35	188
Fya	0.354± 0.098	0.470± 0.120	0.445± 0.039	0.421± 0.065	0.427± 0.085	0.440± 0.039
Fyb	0.646	0.530	0.555	0.579	0.573	0.560
No.tested	24	17	155	57	34	158
K	0.0	0.147± 0.085	0.042± 0.015	0.025± 0.021	0.073± 0.045	0.042± 0.012
k	1.0	0.853	0.958	0.975	0.927	0.958
No.tested	25	17	187	58	34	190
Kpa+	0.0	0.0	0.022± 0.022	0.0	0.036± 0.049	0.022± 0.022
Kpa-	1.0	1.0	0.978	1.0	0.964	0.978
No.tested	9	5	45	39	14	46

Gene frequency distribution by type of birthplace of donor's father (contd.)

Lea+	0.293	0.0	0.107	0.157	0.160	0.108
	0.186		0.043	0.060	0.089	0.042
Lea-	0.707	1.0	0.893	0.843	0.840	0.892
No.tested	6	4	52	38	17	54
P+	0.654	0.423	0.593	0.606	0.521	0.584
	0.095	0.120	0.036	0.064	0.084	0.036
P-	0.346	0.577	0.407	0.394	0.479	0.416
No.tested	25	18	187	58	35	190

Gene frequency distribution by area of birthplace of donor's mother

	NOTTM.			NOTTM. + EDWIN		
	City	Conurbation	Remainder	City	Conurbation	Remainder
p1	0.210± 0.036	0.135± 0.065	0.203± 0.054	0.212± 0.036	0.130± 0.063	0.290± 0.039
p2	0.051	0.066	0.059	0.051	0.062	0.046
q	0.062	0.076	0.106	0.064	0.073	0.076
r	0.677	0.723	0.632	0.673	0.735	0.608
No.tested	126	28	55	130	29	124
M	0.512± 0.044	0.500± 0.093	0.510± 0.067	0.523± 0.043	0.500± 0.091	0.512± 0.045
N	0.488	0.500	0.490	0.477	0.500	0.488
No.tested	130	29	55	134	30	124
S	0.360± 0.043	0.206± 0.075	0.209± 0.055	0.361± 0.042	0.233± 0.077	0.234± 0.088
s	0.640	0.794	0.791	0.639	0.767	0.766
No.tested	129	29	55	133	30	124
D	0.589± 0.043	0.629± 0.089	0.514± 0.067	0.595± 0.042	0.591± 0.089	0.525± 0.045
d	0.411	0.371	0.486	0.405	0.409	0.475
No.tested	130	29	55	134	30	124
C	0.480± 0.050	0.431± 0.100	0.481± 0.078	0.480± 0.049	0.416± 0.100	0.420± 0.055
C^W	0.005	0.0	0.0	0.005	0.0	0.012
c	0.515	0.569	0.519	0.515	0.584	0.568
No.tested	99	21	40	103	21	80
E	0.100± 0.026	0.250± 0.082	0.122± 0.045	0.104± 0.026	0.241± 0.079	0.159± 0.045
e	0.900	0.750	0.878	0.896	0.759	0.841
No.tested	130	28	53	134	29	122
Fya	0.398± 0.047	0.500± 0.100	0.369± 0.071	0.392± 0.046	0.500± 0.098	0.378± 0.045
Fyb	0.602	0.500	0.631	0.608	0.500	0.622
No.tested	108	25	46	112	26	115
K	0.043± 0.018	0.051± 0.041	0.028± 0.022	0.041± 0.017	0.050± 0.040	0.036± 0.017
k	0.957	0.949	0.972	0.959	0.950	0.964
No.tested	129	29	54	133	30	123
Kp^{a+}	0.041± 0.033	0.0	0.0	0.039± 0.031	0.0	0.0
Kpa-	0.959	1.0	1.0	0.961	1.0	1.0
No.tested	37	6	16	39	7	61

Gene frequency distribution by area of birthplace of donor's mother (contd.)

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
Lea+	0.109± 0.049	0.074± 0.099	0.465± 0.190	0.115± 0.048	0.065± 0.087	0.220± 0.050
Lea-	0.891	0.926	0.535	0.885	0.935	0.780
No.tested	41	7	7	44	8	69
P+	0.617± 0.043	0.545± 0.092	0.595± 0.066	0.603± 0.042	0.517± 0.091	0.641± 0.043
P-	0.383	0.455	0.405	0.397	0.483	0.359
No.tested	129	29	55	133	30	124

Gene frequency distribution by type of birthplace of donor's mother

	NOTTM.			NOTTM. + EDWIN.		
	Pit	Rural	Urban	Pit	Rural	Urban
pl	0.195± 0.068	0.196± 0.089	0.192± 0.031	0.280± 0.047	0.241± 0.077	0.191± 0.031
p2	0.056	0.065	0.053	0.040	0.044	0.055
q	0.093	0.135	0.063	0.068	0.103	0.063
r	0.656	0.604	0.692	0.612	0.612	0.691
No.tested	34	20	157	91	31	163
M	0.485± 0.086	0.575± 0.111	0.503± 0.039	0.489± 0.052	0.581± 0.089	0.515± 0.039
N	0.515	0.425	0.497	0.511	0.419	0.485
No.tested	34	20	162	91	31	168
S	0.205± 0.069	0.225± 0.093	0.326± 0.037	0.253± 0.046	0.194± 0.071	0.329± 0.036
s	0.795	0.775	0.674	0.747	0.806	0.671
No.tested	34	20	161	91	31	167
D	0.485± 0.086	0.553± 0.111	0.600± 0.038	0.497± 0.052	0.599± 0.088	0.599± 0.038
d	0.515	0.447	0.400	0.503	0.401	0.401
No.tested	34	20	162	91	31	168
C	0.530± 0.010	0.400± 0.131	0.474± 0.045	0.420± 0.065	0.428± 0.108	0.469± 0.044
C^W	0.0	0.0	0.004	0.008	0.024	0.004
c	0.470	0.600	0.522	0.572	0.548	0.527
No.tested	25	14	123	58	21	127
E	0.088± 0.049	0.166± 0.088	0.127± 0.026	0.148± 0.037	0.155± 0.067	0.135± 0.026
e	0.912	0.834	0.873	0.852	0.845	0.865
No.tested	34	18	161	91	29	167
Fya	0.321± 0.088	0.441± 0.120	0.423± 0.042	0.371± 0.052	0.411± 0.093	0.416± 0.041
Fyb	0.679	0.559	0.577	0.629	0.589	0.584
No.tested	28	17	137	85	28	143
K	0.0	0.050± 0.049	0.046± 0.017	0.027± 0.017	0.048± 0.038	0.415± 0.016
k	1.00	0.950	0.954	0.973	0.952	0.955
No.tested	33	20	161	90	31	167
Kpa+	0.0	0.0	0.035± 0.028	0.0	0.0	0.033± 0.026
Kpa-	1.0	1.0	0.965	1.0	1.0	0.967
No.tested	9	5	44	47	13	47

Gene frequency distribution by type of birthplace of donor's mother (contd.)

	NOTTM.			NOTTM. + EDWIN.		
	Pit	Rural	Urban	Pit	Rural	Urban
Lea+	0.592± 0.201	0.0	0.104± 0.044	0.239± 0.056	0.147± 0.108	0.105± 0.042
Lea-	0.408	1.0	0.896	0.761	0.853	0.895
No.tested	6	1	48	57	11	53
P+	0.547± 0.085	0.684± 0.104	0.591± 0.039	0.621± 0.051	0.689± 0.083	0.576± 0.038
P-	0.453	0.316	0.409	0.378	0.311	0.424
No.tested	34	20	161	91	31	167

Gene frequency distribution by area of birthplace of donor's father's father

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
p1	0.238± 0.059	0.136± 0.064	0.234± 0.065	0.238± 0.059	0.136± 0.064	0.250± 0.052
p2	0.065	0.087	0.065	0.065	0.087	0.039
q	0.080	0.087	0.090	0.080	0.087	0.068
r	0.617	0.790	0.611	0.617	0.790	0.643
No.tested	52	29	42	52	29	69
M	0.481± 0.068	0.433± 0.090	0.534± 0.075	0.481± 0.068	0.433± 0.090	0.529± 0.059
N	0.519	0.567	0.466	0.519	0.567	0.471
No.tested	54	30	44	54	30	71
S	0.305± 0.063	0.233± 0.077	0.227± 0.063	0.305± 0.063	0.233± 0.077	0.288± 0.054
s	0.695	0.767	0.773	0.695	0.767	0.712
No.tested	54	30	44	54	30	71
D	0.570± 0.067	0.592± 0.090	0.602± 0.074	0.570± 0.067	0.592± 0.090	0.589± 0.058
d	0.430	0.408	0.398	0.430	0.408	0.411
No.tested	54	30	44	54	30	71
C	0.454± 0.079	0.450± 0.106	0.546± 0.083	0.454± 0.079	0.450± 0.106	0.491± 0.068
C^W	0.0	0.0	0.0	0.0	0.0	0.009
c	0.546	0.550	0.454	0.546	0.550	0.500
No.tested	40	22	36	40	22	54
E	0.111± 0.043	0.148± 0.068	0.113± 0.047	0.111± 0.043	0.148± 0.068	0.141± 0.041
e	0.889	0.852	0.887	0.889	0.852	0.859
No.tested	54	27	44	54	27	71
Fya	0.500± 0.074	0.420± 0.099	0.410± 0.080	0.500± 0.074	0.420± 0.099	0.446± 0.062
Fyb	0.500	0.580	0.490	0.500	0.580	0.554
No.tested	46	25	38	46	25	65
K	0.018± 0.018	0.066± 0.045	0.058± 0.036	0.018± 0.018	0.066± 0.045	0.064± 0.029
k	0.982	0.934	0.942	0.982	0.934	0.936
No.tested	54	30	43	54	30	70
Kpa+	0.042± 0.058	0.0	0.0	0.042± 0.058	0.0	0.015± 0.021
Kpa-	0.958	1.0	1.0	0.958	1.0	0.985
No.tested	12	7	12	12	7	33

Gene frequency distribution by area of birthplace of donor's father's father (contd.)

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
Lea+	0.134± 0.085	0.094± 0.099	0.057± 0.077	0.134± 0.085	0.074± 0.099	0.160± 0.063
Lea-	0.866	0.926	0.943	0.866	0.926	0.840
No.tested	16	7	9	16	7	34
P+	0.640± 0.065	0.553± 0.091	0.601± 0.074	0.640± 0.065	0.553± 0.091	0.607± 0.058
P-	0.360	0.447	0.398	0.360	0.447	0.393
No.tested	54	30	44	54	30	71

Gene frequency distribution by type of birthplace of donor's father's father

	NOTTM.			NOTTM. + EDWIN		
	Pit	Rural	Urban	Pit	Rural	Urban
p1	0.270± 0.095	0.115± 0.085	0.210± 0.043	0.253± 0.068	0.201± 0.085	0.210± 0.043
p2	0.124	0.131	0.075	0.076	0.090	0.075
q	0.0	0.037	0.077	0.0	0.047	0.077
r	0.606	0.717	0.638	0.671	0.662	0.638
No.tested	22	14	88	41	22	88
M	0.614± 0.104	0.437± 0.124	0.467± 0.052	0.574± 0.077	0.458± 0.102	0.467± 0.052
N	0.386	0.563	0.533	0.426	0.542	0.533
No.tested	22	16	91	41	24	91
S	0.227± 0.089	0.312± 0.116	0.264± 0.046	0.292± 0.071	0.354± 0.098	0.264± 0.046
s	0.773	0.688	0.736	0.708	0.646	0.736
No.tested	22	16	91	41	24	91
D	0.524± 0.106	0.752± 0.108	0.581± 0.052	0.507± 0.078	0.796± 0.082	0.581± 0.052
d	0.476	0.248	0.419	0.493	0.204	0.419
No.tested	22	16	91	41	24	91
C	0.500± 0.125	0.594± 0.123	0.447± 0.060	0.463± 0.093	0.541± 0.111	0.447± 0.060
C^W	0.0	0.0	0.014	0.0	0.0	0.014
c	0.500	0.406	0.539	0.537	0.459	0.539
No.tested	16	15	68	29	20	68
E	0.113± 0.067	0.125± 0.083	0.119± 0.035	0.134± 0.053	0.166± 0.076	0.119± 0.035
e	0.887	0.875	0.881	0.866	0.834	0.881
No.tested	22	16	88	41	24	88
Fya	0.425± 0.111	0.533± 0.129	0.467± 0.057	0.475± 0.080	0.413± 0.103	0.467± 0.057
Fyb	0.575	0.467	0.533	0.525	0.587	0.533
No.tested	20	15	76	39	23	76
K	0.067± 0.053	0.067± 0.065	0.033± 0.019	0.085± 0.044	0.043± 0.042	0.033± 0.019
k	0.933	0.933	0.967	0.915	0.957	0.967
No.tested	22	15	91	41	23	91
Kpa+	0.0	0.0	0.025± 0.035	0.0	0.057 0.077	0.025± 0.035
Kpa-	1.0	1.0	0.975	1.0	0.943	0.975
No.tested	7	4	20	23	9	20

Gene frequency distribution by type of birthplace of donor's father's father (contd.)

Lea+	0.106± 0.138	0.0	0.110± 0.064	0.166± 0.078	0.163± 0.117	0.110± 0.064
Lea-	0.894	1.0	0.890	0.834	0.837	0.890
No.tested	5	3	24	23	10	24
P+	0.699± 0.098	0.441± 0.124	0.608± 0.051	0.687± 0.072	0.460± 0.102	0.622± 0.051
P-	0.301	0.559	0.392	0.313	0.540	0.378
No.tested	22	16	91	41	24	91

Gene frequency distribution by area of birthplace of donor's father's mother

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
p1	0.233± 0.070	0.135± 0.070	0.313± 0.097	0.226± 0.069	0.135± 0.070	0.332± 0.069
p2	0.075	0.131	0.032	0.072	0.131	0.016
q	0.072	0.043	0.046	0.069	0.043	0.033
r	0.620	0.691	0.609	0.633	0.691	0.619
No.tested	36	24	23	37	24	47
M	0.459± 0.082	0.480± 0.100	0.631± 0.100	0.474± 0.081	0.480± 0.100	0.617± 0.071
N	0.541	0.520	0.369	0.526	0.520	0.383
No.tested	37	25	23	38	25	47
S	0.243± 0.071	0.320± 0.093	0.174± 0.079	0.250± 0.070	0.320± 0.093	0.287± 0.066
s	0.757	0.680	0.826	0.750	0.680	0.713
No.tested	37	25	23	38	25	47
D	0.536± 0.082	0.553± 0.099	0.583± 0.103	0.541± 0.081	0.553± 0.099	0.588± 0.072
d	0.464	0.447	0.417	0.459	0.447	0.412
No.tested	37	25	23	38	25	47
C	0.365± 0.098	0.440± 0.120	0.587± 0.113	0.368± 0.096	0.440± 0.120	0.496± 0.083
C^W	0.0	0.0	0.0	0.0	0.0	0.014
c	0.635	0.560	0.413	0.632	0.560	0.490
No.tested	24	17	19	25	17	36
E	0.135± 0.056	0.108± 0.065	0.087± 0.059	0.144± 0.057	0.108± 0.065	0.127± 0.049
e	0.865	0.892	0.913	0.856	0.892	0.873
No.tested	37	23	23	38	23	47
Fya	0.448 0.092	0.479 0.102	0.368 0.111	0.433 0.090	0.479 0.102	0.395 0.075
Fyb	0.552	0.521	0.632	0.567	0.521	0.605
No.tested	29	24	19	30	24	43
K	0.067± 0.041	0.080± 0.054	0.021± 0.03	0.066± 0.040	0.080± 0.054	0.053± 0.033
k	0.933	0.920	0.979	0.934	0.920	0.947
No.tested	37	25	23	38	25	47
Kpa+	0.0	0.0	0.0	0.0	0.0	0.0
Kpa-	1.0	1.0	1.0	1.0	1.0	1.0
No.tested	10	6	8	11	6	26

Gene frequency distribution by area of birthplace of donor's father's mother (contd.)

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
Lea+	0.262± 0.132	0.087± 0.115	0.106± 0.138	0.236± 0.122	0.087± 0.115	0.191± 0.074
Lea-	0.738	0.913	0.894	0.764	0.913	0.802
No.tested	11	6	5	12	6	28
P+	0.598± 0.081	0.510± 0.100	0.793± 0.084	0.613± 0.079	0.510± 0.100	0.748± 0.063
P-	0.402	0.490	0.207	0.397	0.490	0.252
No.tested	37	25	23	38	25	47

Gene frequency distribution by type of birthplace of donor's father's mother

NOTTM. + EDWIN

	Pit	Rural	Urban
p1	0.295± 0.089	0.434± 0.124	0.201± 0.049
p2	0.028	0.0	0.080
q	0.040	0.031	0.054
r	0.635	0.535	0.665
No.tested	26	16	66
M	0.674± 0.091	0.563± 0.124	0.478± 0.061
N	0.326	0.437	0.522
No.tested	26	16	68
S	0.308± 0.091	0.281± 0.112	0.272± 0.054
s	0.692	0.719	0.728
No.tested	26	16	68
D	0.562± 0.097	0.752± 0.108	0.530± 0.061
d	0.438	0.248	0.470
No.tested	26	16	68
C	0.500 0.112	0.563 0.138	0.397 0.073
C^W	0.0	0.0	0.0
c	0.500	0.437	0.603
No.tested	20	13	45
E	0.134± 0.067	0.156± 0.091	0.121± 0.040
e	0.866	0.844	0.879
No.tested	26	16	66
Fya	0.400 0.098	0.357 0.128	0.457 0.065
Fyb	0.600	0.643	0.543
No.tested	25	14	58
K	0.057± 0.045	0.062± 0.060	0.066± 0.030
k	0.943	0.938	0.934
No.tested	26	16	68
Kpa+	0.0	0.0	0.0
Kpa-	1.0	1.0	1.0
No.tested	5	1	17

Gene frequency distribution by type of birthplace of donor's father's mother (contd.)

	NOTTM. + EDWIN.		
	Pit	Rural	Urban
Lea+	0.196±	0.225±	0.173±
Lea-	0.804	0.775	0.827
No.tested	17	10	19
P+	0.806±	0.752±	0.563±
	0.078	0.108	0.060
P-	0.194	0.248	0.437
No.tested	26	16	68

Gene frequency distribution by area of birthplace of donor's mother's father

	NOTTM.			NOTTM. + EDWIN		
	City	Conurbation	Remainder	City	Conurbation	Remainder
pl	0.216± 0.060	0.098± 0.074	0.166± 0.055	0.224± 0.060	0.118± 0.076	0.209± 0.044
p2	0.042	0.035	0.082	0.041	0.032	0.060
q	0.033	0.098	0.091	0.032	0.087	0.080
r	0.709	0.769	0.661	0.703	0.763	0.651
No.tested	47	16	46	48	18	86
M	0.450± 0.070	0.440± 0.120	0.522± 0.073	0.460± 0.070	0.474± 0.115	0.523± 0.054
N	0.550	0.560	0.478	0.540	0.526	0.477
No.tested	50	17	47	51	19	87
S	0.360± 0.068	0.176± 0.092	0.191± 0.057	0.353± 0.067	0.210± 0.093	0.229± 0.045
s	0.640	0.824	0.809	0.647	0.790	0.771
No.tested	50	17	47	51	19	87
D	0.576± 0.070	0.758± 0.104	0.495± 0.073	0.581± 0.069	0.770± 0.097	0.510± 0.054
d	0.424	0.242	0.505	0.419	0.230	0.490
No.tested	50	17	47	51	19	87
C	0.478± 0.079	0.500± 0.139	0.457± 0.084	0.478± 0.078	0.500± 0.129	0.415± 0.063
C^W	0.012	0.0	0.0	0.012	0.0	0.016
c	0.510	0.500	0.543	0.510	0.500	0.569
No.tested	40	13	35	41	15	61
E	0.090± 0.040	0.218± 0.103	0.113± 0.048	0.088± 0.040	0.222± 0.098	0.143± 0.038
e	0.910	0.782	0.887	0.912	0.778	0.857
No.tested	50	16	44	51	18	84
Fya	0.440± 0.079	0.468± 0.125	0.378± 0.076	0.430± 0.075	0.416± 0.116	0.407± 0.055
Fyb	0.560	0.532	0.622	0.570	0.584	0.593
No.tested	42	16	41	43	18	81
K	0.040± 0.028	0.029± 0.041	0.043± 0.030	0.039± 0.027	0.026± 0.037	0.052± 0.024
k	0.960	0.971	0.957	0.961	0.974	0.948
No.tested	50	17	46	51	19	86
Kpa+	0.047± 0.064	0.0	0.0	0.047± 0.064	0.0	0.0
Kpa-	0.953	1.0	1.0	0.953	1.0	1.0
No.tested	11	5	11	11	7	39

Gene frequency distribution by area of birthplace of donor's mother's father (contd.)

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
Lea+	0.061± 0.058	0.183± 0.223	0.368± 0.216	0.057± 0.055	0.225± 0.187	0.242± 0.068
Lea-	0.939	0.817	0.632	0.943	0.775	0.758
No.tested	17	3	5	18	5	40
P+	0.756± 0.061	0.581± 0.120	0.539± 0.073	0.721± 0.063	0.603± 0.112	0.586± 0.053
P-	0.244	0.419	0.461	0.279	0.397	0.414
No.tested	50	17	47	51	19	87

Gene frequency distribution by type of birthplace of donor's mother's father

	NOTTM.			NOTTM. + EDWIN.		
	Pit	Rural	Urban	Pit	Rural	Urban
pl	0.205± 0.078	0.138± 0.086	0.166± 0.045	0.231± 0.054	0.177± 0.081	0.176± 0.045
p2	0.023	0.163	0.045	0.032	0.119	0.043
q	0.097	0.101	0.044	0.068	0.122	0.043
r	0.675	0.598	0.745	0.669	0.582	0.738
No.tested	27	16	69	61	22	72
M	0.463± 0.096	0.618± 0.118	0.452± 0.058	0.500± 0.064	0.587± 0.103	0.467± 0.057
N	0.537	0.382	0.548	0.500	0.413	0.533
No.tested	27	17	73	61	23	76
S	0.185± 0.075	0.176± 0.032	0.308± 0.054	0.229± 0.054	0.217± 0.086	0.309± 0.053
s	0.815	0.824	0.692	0.771	0.783	0.691
No.tested	27	17	73	61	23	76
D	0.491± 0.096	0.458± 0.121	0.630± 0.057	0.521± 0.064	0.449± 0.104	0.637± 0.055
d	0.509	0.542	0.370	0.479	0.551	0.363
No.tested	27	17	73	61	23	76
C	0.481± 0.112	0.382± 0.140	0.499± 0.065	0.406± 0.076	0.404± 0.123	0.499± 0.063
C^W	0.0	0.0	0.008	0.012	0.031	0.008
c	0.519	0.618	0.493	0.582	0.565	0.493
No.tested	20	12	59	42	16	62
E	0.111± 0.060	0.035± 0.049	0.132± 0.040	0.163± 0.047	0.025± 0.035	0.133± 0.039
e	0.889	0.965	0.868	0.837	0.975	0.867
No.tested	27	14	72	61	20	75
Fya	0.369± 0.101	0.433± 0.128	0.422± 0.062	0.412± 0.065	0.428± 0.108	0.403± 0.060
Fyb	0.631	0.567	0.578	0.588	0.572	0.597
No.tested	23	15	64	57	21	67
K	0.0	0.059 ± 0.057	0.054 ± 0.026	0.042 ± 0.026	0.043 ± 0.042	0.052 ± 0.025
k	1.0	0.941	0.946	0.958	0.957	0.948
No.tested	26	17	73	60	23	76
Kpa+	0.0	0.0	0.031± 0.043	0.0	0.0	0.028± 0.039
Kpa-	1.0	1.0	0.969	1.0	1.0	0.972
No.tested	8	3	16	34	5	18

Gene frequency distribution by type of birthplace of donor's mother's father (contd.)

	NOTTM.			NOTTM. + EDWIN.		
	Pit	Rural	Urban	Pit	Rural	Urban
Lea+	0.423± 0.285	0.293± 0.322	0.078± 0.060	0.231± 0.072	0.293± 0.186	0.091± 0.060
Lea-	0.577	0.707	0.922	0.769	0.707	0.909
No.tested	3	2	20	34	6	23
P+	0.570± 0.095	0.514± 0.121	0.691± 0.054	0.616± 0.062	0.535± 0.104	0.676± 0.054
P-	0.430	0.486	0.309	0.384	0.465	0.324
No.tested	27	17	73	61	23	76

Gene frequency distribution by area of birthplace of donor's mother's mother

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
pl	0.158± 0.059	0.107± 0.098	0.183± 0.060	0.170± 0.060	0.081± 0.076	0.218± 0.049
p2	0.049	0.058	0.045	0.047	0.088	0.037
q	0.041	0.052	0.088	0.040	0.040	0.073
r	0.752	0.783	0.684	0.743	0.791	0.672
No.tested	38	10	42	39	13	72
M	0.475± 0.079	0.500± 0.158	0.524± 0.077	0.475± 0.078	0.539± 0.138	0.507± 0.059
N	0.525	0.500	0.476	0.525	0.461	0.493
No.tested	40	10	42	41	13	72
S	0.312± 0.073	0.200± 0.126	0.214± 0.063	0.317± 0.073	0.192± 0.109	0.229± 0.050
s	0.688	0.800	0.786	0.683	0.808	0.771
No.tested	40	10	42	41	13	72
D	0.613± 0.077	0.684± 0.147	0.513± 0.077	0.618± 0.076	0.723± 0.124	0.544± 0.059
d	0.387	0.316	0.487	0.382	0.277	0.456
No.tested	40	10	42	41	13	72
C	0.462± 0.088	0.450± 0.188	0.476± 0.088	0.463± 0.087	0.423± 0.165	0.436± 0.069
C^W	0.0	0.0	0.0	0.0	0.0	0.001
c	0.438	0.550	0.524	0.537	0.577	0.563
No.tested	32	7	32	33	9	51
E	0.100± 0.047	0.277± 0.149	0.100± 0.047	0.110± 0.049	0.291± 0.131	0.135± 0.041
e	0.900	0.723	0.900	0.890	0.709	0.865
No.tested	40	9	40	41	12	70
Fya	0.364± 0.084	0.500± 0.158	0.405± 0.081	0.382± 0.083	0.500± 0.139	0.425± 0.060
Fyb	0.636	0.500	0.595	0.618	0.500	0.575
No.tested	33	10	37	34	13	67
K	0.050± 0.034	0.050± 0.069	0.049± 0.033	0.048± 0.033	0.038± 0.053	0.035± 0.022
k	0.950	0.950	0.951	0.952	0.962	0.965
No.tested	40	10	41	41	13	71
Kpa+	0.106± 0.138	0.0	0.0	0.087± 0.115	0.0	0.0
Kpa-	0.894	1.0	0.0	0.913	1.0	1.0
No.tested	5	2	11	6	4	33

Gene frequency distribution by area of birthplace of donor's mother's mother (contd.)

	NOTTM.			NOTTM. + EDWIN.		
	City	Conurbation	Remainder	City	Conurbation	Remainder
Lea+	0.074± 0.070	0.293± 0.322	0.462± 0.188	0.106± 0.079	0.106± 0.138	0.314± 0.080
Lea-	0.926	0.707	0.538	0.894	0.894	0.686
No.tested	14	2	7	15	5	34
P+	0.729± 0.071	0.452± 0.157	0.538± 0.077	0.730± 0.069	0.520± 0.139	0.544± 0.059
P-	0.273	0.548	0.462	0.270	0.480	0.456
No.tested	40	10	42	41	13	72

Gene frequency distribution by type of birthplace of donor's mother's mother

	NOTTM.			NOTTM. + EDWIN.		
	Pit	Rural	Urban	Pit	Rural	Urban
pl	0.182± 0.074	0.243± 0.124	0.133± 0.047	0.225± 0.059	0.244± 0.099	0.133± 0.045
p2	0.022	0.123	0.044	0.025	0.073	0.052
q	0.097	0.090	0.039	0.052	0.145	0.036
r	0.699	0.544	0.784	0.698	0.538	0.779
No.tested	27	12	53	50	19	57
M	0.500± 0.096	0.584± 0.142	0.472± 0.067	0.490± 0.071	0.553± 0.114	0.483± 0.065
N	0.500	0.416	0.528	0.510	0.447	0.517
No.tested	27	12	55	50	19	59
S	0.166± 0.072	0.250± 0.125	0.291± 0.061	0.210± 0.058	0.236± 0.097	0.288± 0.059
s	0.834	0.750	0.709	0.790	0.764	0.712
No.tested	27	12	55	50	19	59
D	0.456± 0.096	0.592± 0.142	0.644± 0.065	0.553± 0.070	0.487± 0.115	0.656± 0.062
d	0.544	0.408	0.356	0.447	0.513	0.344
No.tested	27	12	55	50	19	59
C	0.444± 0.114	0.500± 0.158	0.472± 0.075	0.410± 0.084	0.439± 0.133	0.466± 0.073
C^W	0.0	0.0	0.0	0.0	0.035	0.0
c	0.556	0.500	0.528	0.590	0.526	0.534
No.tested	19	10	44	34	14	47
E	0.092± 0.056	0.050± 0.069	0.139± 0.049	0.150± 0.050	0.059± 0.057	0.195± 0.048
e	0.908	0.950	0.861	0.850	0.941	0.845
No.tested	27	10	54	50	17	58
Fya	0.375± 0.099	0.450± 0.157	0.406± 0.071	0.393± 0.071	0.500± 0.121	0.422± 0.068
Fyb	0.625	0.550	0.594	0.607	0.500	0.578
No.tested	24	10	48	47	17	52
K	0.057± 0.045	0.041± 0.057	0.045± 0.028	0.041± 0.028	0.026± 0.037	0.042± 0.026
k	0.943	0.959	0.955	0.959	0.974	0.958
No.tested	26	12	55	49	19	59
Kpa+	0.0	0.0	0.074± 0.099	0.0	0.0	0.051± 0.070
Kpa-	1.0	1.0	0.926	1.0	1.0	0.949
No.tested	9	2	7	29	4	10

Gene frequency distribution by type of birthplace of donor's mother's mother (contd.)

	NOTTM.			NOTTM. + EDWIN.		
	Pit	Rural	Urban	Pit	Rural	Urban
Lea+	0.500± 0.250	0.423± 0.285	0.099± 0.075	0.280± 0.086	0.465± 0.189	0.106± 0.069
Lea-	0.500	0.577	0.901	0.720	0.535	0.894
No.tested	4	3	16	27	7	20
P+	0.570± 0.095	0.500± 0.144	0.644± 0.065	0.531± 0.071	0.603± 0.112	0.656± 0.062
P-	0.430	0.500	0.356	0.469	0.397	0.344
No.tested	27	12	55	50	19	59

Gene frequency distribution for Nottingham + Edwinstone

by immigration

	1901-1940	1941-1950	1951-1960	1961-1970	1971-
p1	0.172± 0.050	0.170± 0.060	0.181± 0.050	0.193± 0.030	0.151± 0.041
p2	0.053	0.0	0.062	0.058	0.064
q	0.129	0.082	0.077	0.086	0.076
r	0.646	0.748	0.680	0.663	0.709
No.tested	55	39	61	174	76
M	0.490± 0.066	0.513± 0.080	0.475± 0.063	0.535± 0.037	0.559± 0.057
N	0.510	0.487	0.525	0.465	0.441
No.tested	57	39	62	178	76
S	0.263± 0.058	0.230± 0.067	0.311± 0.059	0.281± 0.034	0.289± 0.052
s	0.737	0.770	0.689	0.719	0.711
No.tested	57	39	61	178	76
D	0.551± 0.065	0.423± 0.079	0.560± 0.063	0.490± 0.037	0.572± 0.057
d	0.449	0.577	0.440	0.510	0.428
No.tested	57	39	62	179	76
C	0.417± 0.076	0.421± 0.101	0.447± 0.073	0.411± 0.045	0.412± 0.067
C^W	0.0	0.0	0.021	0.0	0.009
c	0.583	0.579	0.532	0.590	0.579
No.tested	42	24	47	118	54
E	0.143± 0.046	0.064± 0.039	0.139± 0.044	0.144± 0.026	0.164± 0.042
e	0.857	0.936	0.861	0.856	0.836
No.tested	56	39	61	177	76
Fya	0.384± 0.068	0.419± 0.081	0.368± 0.066	0.438± 0.040	0.525± 0.064
Fyb	0.616	0.581	0.632	0.562	0.475
No.tested	50	37	53	154	61
K	0.058± 0.030	0.038± 0.031	0.041± 0.025	0.042± 0.015	0.065± 0.028
k	0.942	0.962	0.959	0.958	0.935
No.tested	57	39	61	179	76
Kpa+	0.0	0.039± 0.054	0.0	0.0	0.0
Kpa-	1.0	0.961	1.0	1.0	1.0
No.tested	22	13	17	57	24

Gene frequency distribution for Nottingham + Edwinstone by immigration (contd.)

	1901-1940	1941-1950	1951-1960	1961-1970	1971
Lea+	0.155± 0.072	0.376± 0.114	0.134± 0.070	0.109± 0.038	0.087± 0.081
Lea-	0.845	0.624	0.866	0.891	0.913
No.tested	25	18	24	68	12
P+	0.532± 0.066	0.680± 0.075	0.641± 0.061	0.666± 0.035	0.657± 0.054
P-	0.468	0.320	0.359	0.334	0.343
No.tested	57	39	62	179	76