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**Anna Christie**

## **Are there intervention-generated inequalities in type 2 diabetes care? A systematic review and analysis of routine data**

### **Abstract**

This thesis aimed to contribute to current understanding of *'intervention-generated inequalities'*, that is, the concern that processes in the planning or delivery of an intervention may create or exacerbate the health differences between population groups. This was done by examining the impact of secondary and tertiary preventive interventions for type 2 diabetes by socio-economic status (SES). Previous research has shown that the condition places a disproportionate burden on individuals from disadvantaged backgrounds. In addition, managing the condition involves a range of health care; all potentially exacerbating existing health inequalities.

A systematic review was conducted and secondary data analyses of patient data collected by a hospital diabetes register. The Index of Multiple Deprivation 2004 was used as an indicator of patients' SES. Multilevel models were fitted using repeated measurements, with patients nested within general practices. Interaction effects were used to determine inequalities over time and if interventions were associated with differential health outcomes by SES.

The multilevel analyses showed that high SES patients were more likely to have lower blood glucose over time, but higher levels of cholesterol compared to low SES patients. In contrast, there were few differences in long-term health complications by SES over time. High SES patients were more likely to receive higher quality of care and shared care than low SES patients over time. Furthermore, there were significant inequalities in health by SES were found in patients receiving the same care. There were also significant inequalities in prescriptions for treatments, conditional on other relevant covariates.

The results in this thesis indicate that there were intervention generated inequalities which are particularly important for practitioners. As these were either a result of interventions not being appropriately accessed and/or administered based on need or the efficacy of these interventions differed by SES. Further analyses are needed to unpick the direction of these associations.

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'Are there intervention-generated inequalities in type 2 diabetes care? A systematic review and analysis of routine data'

PhD

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## List of Abbreviations

ACEI	Angiotensin-converting enzyme inhibitor
ADS	Attribution Dataset
ASSIA	Applied Social Sciences Index and Abstracts
BMI	Body mass index
BP	Blood pressure
CCG	Clinical Commissioning Group
CINALH	Cumulative Index to Nursing and Allied Health Literature
CPS	Community Preventive Services
CV	Cardiovascular
CVD	Cardiovascular disease
dBp	Diastolic blood pressure
DIC	Deviance Information Criterion
EBM	Evidence based medicine
eGFR	Estimated glomerular filtration rate
EMBASE	Excerpta Medica database
GEE	Generalized Estimating Equations
GFR	Glomerular filtration rate
HbA	Haemoglobin A
HbA1c	Glycated haemoglobin
HDL-c	High-density lipoprotein cholesterol
HES	Hospital Episode Statistics
ICD	Ischaemic Cardiac Disease
ICC	Intraclass correlation coefficients
ICL	Inverse Care Law
IGI	Intervention generated inequalities
IMD	Index of Multiple Deprivation
Kg	Kilograms
LA	Local authority
LDL-c	Low-density lipoprotein cholesterol
LSOA	Lower super output area
MAR	Missing at random
MCAR	Missing completely at random
MCMC	Markov Chain Monte Carlo
MDRD	Modification of diet in renal disease

MeSH	Medical Subject Headings
MI	Multiple imputation
ML	Maximum likelihood
NDA	National Diabetes Audit
NEPHO	North East Public Health Observatory
NHS	National Health Service
NICE	National Institute for Health and Clinical Excellence
NSF	National Service Framework
NS-SEC	National Statistics Socio-Economic Classification
OECD	Organisation for Economic Co-operation and Development
OHA	Oral anti hyperglycaemic agents
ONS	Office for National Statistics
PCT	Primary Care Trust
PHO	Public Health Observatories
PVD	Peripheral vascular disease
QOF	Quality and Outcomes Framework
sBP	Systolic blood pressure
SEP	Socioeconomic position
SES	Socio-economic status
STROBE	STrengthening the Reporting of OBservational studies in Epidemiology
TIA	Transient ischemic attack
UK	United Kingdom
YHPHO	Yorkshire and Humber Public Health Observatory

## **Declaration**

I declare that the work contained in this thesis has not been submitted for any other award and that it is all my own work, except where explicitly stated. I also confirm that this work fully acknowledges opinions, ideas and contributions from the work of others.

## **Statement of Copyright**

The copyright of this thesis rests with the author. No quotation from it should be published without the prior written consent and information derived from it should be acknowledged.

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## Chapter 1: Introduction

The aim of this thesis was to contribute to the understanding of intervention generated inequalities (IGIs), that is, any process in the planning or delivery of an intervention aimed at improving health overall that has different outcomes in different social groupings in the target population [1]. This was achieved through a systematic review and secondary data analyses of inequalities associated with type 2 diabetes.

The background to this study is rooted in both national and international political agendas related to unequal relationship between socio- demographic and economic conditions and health. This chapter discusses the definition and interpretation of this relationship and provides a broad look at the political history of addressing the issue, with a particular focus on England. The chapter then goes on to discuss the rationale for examining IGIs with the focus on type 2 diabetes. Finally, the chapter discusses why the population of the South Tees, an area in the North East of England, was chosen and what was already known about diabetes and health inequalities in this locality by drawing upon routine data and existing analyses.

### Background to the study

The unequal relationship between socio- demographic and economic conditions and health is often referred to as 'health inequalities'. While this term is widely used its precise definition also has a broad interpretation, depending upon which axes of social differentiation are examined and how health is described and measured. In broad terms, it is understood to refer to the differences in health between populations groups [2] according to socio-demographic and economic characteristics defined by, for example, location, race, ethnicity or culture, occupation, gender, religion, age, education or income [1].

It should be acknowledged that 'health inequalities' and 'health inequities' are often used interchangeably when discussing this phenomenon. 'Health inequalities' is used throughout this thesis to ensure consistency, however, 'health inequities' could have easily been chosen instead. 'Health inequities' is more often used to emphasise that differences in health between population groups are unfair and avoidable and tackling the issue requires societal change to redress the systemic failings [3].

In the United Kingdom (UK), there is a long history of seeking explanations and solutions to the issue of health inequalities. Since the nineteenth century Britain has led the rest of the world in systematic data collection and analysis with the Office of National Statistics, and its predecessors, investments in birth cohort and longitudinal studies [4-6]. A report in Liverpool in 1840 by Edwin Chadwick showed that the average age at death was 35 years for gentry and professional classes and 15 years for labourers [7]. These findings led to the introduction of the 1848 Public Health Act which legislated for street cleaning, refuse collection, and establishing and improving water supplies and sewage systems [8].

In the UK, the National Health Service (NHS) was established in 1948 to provide free care for all. While it was not explicitly cited, there was an assumption made by many that inequalities in health would be rectified as a result. Subsequent research, however, has shown that this has not been the case. The Black Report in 1980 [8] was a milestone publication and re-established health inequalities on the political agenda in Britain. The authors of the report attributed the inequalities in health to the inequalities in other social circumstances, such as education and working conditions, and recommended improvements in preventative and primary health care. However, due to the political circumstances at the time, it was not until the Labour party returned to power in 1997 and the publication of the Acheson Report in 1998 that the issues and recommendations raised in these reports were addressed at a national policy level [8].

Following this report, tackling health inequalities formed a major part of the Labour party political agenda. In 1999, 'Reducing health inequalities: an action Report' was published which introduced initiatives such as 'Sure Start', 'Health Action Zones', national minimum wage, improved benefits and pension rates [9]. Spending was also increased on education, housing, urban regeneration and healthcare. This was followed by a cross-cutting review of tackling health inequalities [10] and a revised strategy 'Tackling health inequalities: a Programme for Action'[11]. The strategy included details of how the national public service agreement target set in 2001 to reduce inequalities in health outcomes by 10 per cent as measured by infant mortality and life expectancy at birth was to be achieved [6, 11].

Since then a series of status reports have been published which reveal that despite the scale of work and overall improvements in life expectancy and infant mortality rates, inequalities remain and in some instances have increased [4, 6]. In particular, the 2007 Status Report found that the relative gap in life expectancy between England as a whole and the fifth most deprived areas had increased by 2% for men and by 11% for females between 2003-05 and 2004-06[12]. As such the need for effective action on tackling health inequalities remains pertinent. The Marmot Review 'Fair Society, Healthy Lives' in 2010 reiterated previous assertions that the

need to tackle health inequalities is a matter of social justice. In addition, the 2010 review asserts that there is an economic benefit stating that if health inequalities were eradicated then the same disadvantaged groups would experience a further 2.8 million years of disability and long-term illness free life. This would also save the NHS in England an estimated £5.5 billion plus and other billions more in productivity, taxes and welfare payments losses [4].

In 2010 the new Conservative-Liberal Democrat coalition government published the white paper 'Equity and Excellence: Liberating the NHS' [13]. In this white paper, the coalition outlined its plans to uphold the values and principles of the NHS; increase spending in real terms and making the NHS a world-class health service. Two major changes the coalition sought to introduce were, firstly, handing the majority of the budget to general practitioners and, secondly, the establishment of public health service primarily situated within local authorities (LAs) so that those responsible for commissioning and running services are closer to the population they serve [13]. The coalition also published a public health white paper 'Healthy lives, healthy people: our strategy for public health in England' later in 2010 [14].

Commentators on the white papers welcomed the continued commitment to public health and reducing health inequalities exemplified through the proposal of a 'health premium', which gives LAs additional funds for health improvement services. These funds are to be allocated depending upon improvements in health of the local population [13, 15, 16]. However, critics suggest that allocation based upon performance may actually widen health inequalities by perpetuating the inverse care law. That is, areas that have greater need, but do not achieve significant improvements in the health of the population, would not receive the extra funding. Lack of improvement may be due to initial poor funding therefore again increasing the challenge for these areas [15].

The changes in the health service arrangements introduced by the Conservative-Liberal Democrat coalition government and the responses to them show that tackling health inequalities is still a major political issue. Yet, whilst there is on-going work to improve health and reduce inequalities there are growing concerns that some health strategies could actually lead to the widening of the health differences between population groups.

## Rationale for thesis

As mentioned above the aim of this thesis is to contribute the understanding of 'intervention generated inequalities', that is, how and why interventions aimed at improving population health overall could lead to the widening of the health differences between population groups. This section introduces the rationale for examining the phenomenon of IGIs and the focus of type 2 diabetes using data collected in the South Tees area.

### Intervention generated inequalities

The phrase 'intervention generated inequalities' was coined by White *et al* in 2009 to bring together existing papers and theories which have noted the differences in the access, uptake and impact of intervention by population groups. The authors wanted to emphasise how IGIs can occur at any stage of the intervention process [1].

One of the more famous pieces of work is Tudor-Hart's 'Inverse Care Law' (ICL). Tudor-Hart's paper was published in 1971 and related to primary care. The ICL states that patients' access to good health care is inversely related to need. This has been interpreted to suggest that the most disadvantaged groups have the poorest access to health care as the need for such services is strongly related to socioeconomic position (SEP) [1]. This interpretation has become somewhat accepted and detached from its original inception. In particular, who has the greatest need for health services is not always associated with SEP. Increasing age has been shown to be more closely related to increased morbidity and mortality than deprivation for most conditions. Similarly, what is regarded as 'good medical care' is also debatable. For instance, good medical care that meets the needs of patients with diagnosed health problems or care which reduces risk and prevents illness [17, 18].

Despite the argument that is not a 'law', as it is not based on a systematic review of evidence when it was first purported [19], and some subsequent skewed interpretations the ICL continues to be a widely cited explanation and has been supported by evidence from a wide range of settings. For example, in a recent review of attendance to health check-ups in developed countries, it was found that people from low SEP were less likely to attend but were also the people who were likely to have a greater clinical need or risk factors [20]. In addition, there are a few studies which have found evidence to contradict this law. One study in the North

East of England found that more deprived patients were geographically closer to general practices than the least deprived, however, this study did not measure medical need of the patients, the quality of the care and whether it was appropriately accessed [21]. Work has also been conducted to explain why this law persists. One study did this by conducting a questionnaire study of NHS patients in the West of Scotland. The authors also found that it was patients from more deprived areas who had the greatest clinical need. This increased burden in deprived areas lead to greater demands on primary care which is associated with reduced access to scheduled care, shorter consultations, higher GP stress and lower levels of patients being able to cope with and understand their psychosocial problems [22].

A particular limitation of the ICL which White *et al* [1] highlighted is that it is primarily concerned with the provision of health services. This is only one type of intervention and one way that inequalities could be introduced or exacerbated and therefore has limited capacity to explain the overall phenomenon of IGIs [1].

A more recent theory which White *et al* [1] brought under the IGI term is the ‘inverse equity hypothesis’. It has been described as a corollary to the ICL and could arguably be an evidence-based version of the ‘inverse prevention law’ briefly referred to in the Acheson Report in 1999. The ‘inverse prevention law’ refers to the concept that individuals least likely to receive preventative measures are those most likely to benefit from them. Similarly to the ICL, no evidence was presented to support this theory when it was first purported [1] and in contrast it has been less widely cited. However, this maybe because that the ICL has been expanded to a range of interventions associated with health beyond the formal medical care for which the theory was initially devised [23]. In contrast, Victora *et al* [24] used analyses of time trends in child health statuses in three Brazilian epidemiology datasets to demonstrate how new public health interventions initially show greater utilisation and health improvements in the most advantaged proportion of the population thereby increasing inequalities. These later reduce as utilisation broadens and the health improvements reach a new plateau. This hypothesis has been used to explain the regional inequalities in liver cirrhosis mortality rates in Taiwan with differences in uptake in hepatitis B vaccination programmes [25].

While the ‘inverse equity hypothesis’ provides a testable framework, it has been shown that such trends may support an artefact theory of IGIs [26]. That is, while the existence of inequalities is not disputed the longitudinal trend in terms of whether the inequalities are increasing or decreasing is dependent upon the prevalence of the outcome being measured. For example, if two groups differ in their susceptibility to an outcome, the rarer the outcome the greater the relative inequalities will be and the more common the outcome the smaller the

relative inequalities will be. As such the prevalence of an outcome affects the change in the size of the perceived relative inequalities and is statistically expected to follow the pattern Victora *et al* described. As such it is not known whether increased usage of an initially rare intervention reflects a meaningful reduction in relative inequalities or if the data is just reflecting the expected statistical pattern. Careful reference therefore needs to be made to the prevalence of the outcome measurement when making conclusions about trends in inequalities using binary measures [26]. This is arguably evident in some of the findings in the DH 2007 'Status Report on the Programme for Action' [12]. For instance, even though the overall prevalence of smoking during pregnancy has decreased slightly, between 2000 to 2005 there was a slight increase for 'routine and manual' workers contributing to possible widening in inequalities.

The 'equity-effectiveness loop' [27] was also discussed by White *et al* [1]. This framework and calculation approach was devised by Tugwell *et al* [27]. They emphasised that inequalities as a result of an intervention and its overall effectiveness can be affected by any stage of an intervention. Yet, no direct evidence was depicted to show a multiplicative effect, nor were the aspects of interventions which have an effect on inequalities been identified [1]. Studies elsewhere, however, have identified characteristics of interventions which are likely to increase inequalities. For instance, Capewell and Graham [28] reviewed various approaches to cardiovascular disease (CVD) prevention and found consistent evidence support for the Geoffrey Rose [29] approach to disease prevention through taking a dual strategy of whole-population interventions as well targeting high-risk individuals. The authors found that while whole-population approaches may not reduce inequalities they do not increase either as interventions, such as smoke-free legislation and water fluoridation, work effectively across the social gradient. Whereas "agentic" interventions, that is those which are based on individual behaviour, such as breast screening programmes and primary prevention medications, require material and psychological resources and favour those with more to draw upon. This is usually people from less deprived backgrounds compared to those from the most deprived, thereby increasing inequalities [28]. This is supported by a recent review of reviews by Lorenc *et al* [30] which found 'downstream', non healthcare interventions which focused on individual factors are more likely to increase inequalities compared to 'upstream' interventions which operate on a social or policy level. Nettle [31] theorises that this social gradient in preventive health behaviour takes a behavioural ecological approach and argues that there is an 'exacerbatory dynamic of poverty' explaining that people from lower SEP have greater exposure to unavoidable harms which disincentives them from investing in positive health behaviours. As such agentic interventions relying on individual behaviour change are likely to introduce further inequalities [31]. These hypotheses are not always supported: Toft *et al* [32] found

evidence that a longitudinal, multifactorial lifestyle intervention to change dietary behaviour found a greater improved effect on lower educated and unemployed participations. However, the study's low participation rates and high degree of attrition may have impacted upon the reliability of these results [32].

The way in which health systems operate also has the potential to increase health inequalities. Ali [33] argues that the personalisation of the NHS, exemplified in 'The NHS Plan' and 'The Expert Patient', could actually exacerbate inequalities as it is the already disadvantaged who will be less likely to be able to make an informed choice over which services to access. In turn, public reporting of quality of services could lead to further inequalities as health professionals and organisations avoid serving high-risk patients that hold the potential to reduce quality outcome measures. This is also a criticism of the increased use of private health service providers, which form part of the current coalition government plans, who potentially may 'cherry pick' patients to ensure lower costs [34].

Much of the work investigating IGIs highlights that interventions that are based around individual behaviour are key sources of the emergence of inequalities. For example, choosing services based on quality requires the individual to seek out that information [33]. Preventative health care, such as changing lifestyle, attending screening services, adhering to medications are all dependent upon individual action. The implication from the work of Capewell *et al* [28], Graham *et al* [2] and Nettle [31] that interventions are not designed to overcome the lack of material and psychological resources and the 'exacerbatory of dynamic of poverty', is that individuals from more deprived socio-economic backgrounds are likely to experience poorer health outcomes from the same interventions compared to the least deprived.

Despite this extensive work which has already been undertaken, Macintyre and Petticrew [35] have previously argued that there are widely held misconceptions that interventions aimed at improving health, and other social circumstances, only have the capacity to do good. In addition, there was an assumption that it is enough to know the intervention does good overall and not whether it has an equal, positive impact on all population groups, how it works and at what cost. Macintyre and Petticrew suggest these misconceptions, amongst others, explain why there has been reluctance amongst practitioners and social scientists to use evidence based medicine (EBM) principles in real-life, complex social settings [35]. This reluctance to use EBM principles, in addition to poor planning and limited subsequent evaluation [6, 36], could be regarded as possible reasons for the failure of the Labour government to meet their own targets to reduce inequalities. It could also be argued that this has continued both at national policy level and in interventions aimed at individuals and smaller populations.

In summary, there has been increasing attention to the adverse effects of health interventions yet more research is required to provide a broader picture of the type and nature of interventions which increase or decrease inequalities. A more robust set of evidence will enable the production of practical advice for policy makers, commissioners and practitioners to reduce health inequalities [1, 30].

## Focus of thesis

### *Type 2 diabetes*

Diabetes mellitus, or diabetes, is a condition characterised by hyperglycaemia resulting from defects in insulin secretion, insulin action, or both. Type 2 is one classification of the condition. Patients are considered to have type 2 diabetes when either the body does not produce enough insulin or it does not react effectively to it in order to maintain blood glucose levels at an appropriate level [37, 38]. This section outlines why type 2 diabetes is an increasingly important health issue, both in the UK and worldwide, and why it is also an ideal condition to be the focus of analysis examining IGIs. Chapter two provides a greater description of the health problems associated with type 2 diabetes and how it is managed.

Firstly, diabetes is expected to affect an increasing proportion of the world population, frequently described as being of epidemic proportions. An estimated 246 million people worldwide suffer from diabetes [39]. In 2010, the estimated prevalence of both diagnosed and undiagnosed diabetes in England was 7.4%; 3,099,853 people aged 16 years or older. By 2030 it is estimated about one in ten of the population could have diabetes [40]. Type 2 diabetes accounts for approximately 90 to 95% of the prevalence of diabetes in adults worldwide [41].

Secondly, whilst anyone can develop type 2 diabetes it is overrepresented in certain population groups, particularly those from lower SEP and particular ethnic groups. The burden of diabetes also does not affect everyone equally; the most deprived groups in the UK are two and half times more likely to have diabetes and three and half times more likely to have severe complications [42]. In the North East of England there was a greater prevalence among men and women in the most deprived areas compared to the national average, 28% and 45% higher respectively [43]. Inequalities in type 2 diabetes by SEP have also been shown on an international scale. A systematic review of studies conducted between 1999 and 2009 found

that type 2 diabetes patients in a poorer SEP had a greater incidence, prevalence and mortality rates [44].

Type 2 diabetes is a complex condition and patients can expect to be engaged with a wide range of health services as part of their routine care. The diabetes care pathway is outlined in Figure 1. As such, there should be a wide range of health data collected on a routine basis for all type 2 diabetes patients who are engaged with health care services [45]. Tugwell *et al* [27] purport that the equity effectiveness of health interventions in real settings and systems at community level is dependent upon the extent of awareness, access, or coverage; screening, diagnosis, or targeting; compliance of providers; and adherence of consumers. There is, therefore, a diverse range of processes involved in the management of type 2 diabetes which have the potential to introduce or exacerbate inequalities in outcome by different social groups.

Finally, diagnosed patients are an easily identified population as it is recommended by the National Institute for Health and Clinical Excellence (NICE) that patients have at least one health service visit to receive an annual review of their condition [46]. In addition to primary care and hospital data this is facilitated by several current schemes and policies which encourage the routine registration and collection of laboratory and administrative data on all known diabetic patients. In England, this includes the Quality and Outcomes Framework (QOF) [47], the National Screening Programme for Diabetic Retinopathy [48] which requires an accurate diabetes register in order for it to achieve the target of 100% screening rate set out in the National Service Framework (NSF) for Diabetes [49] and the National Diabetes Audit (NDA) [50]. The audit, designed and delivered by the National Clinical Audit Support Programme, provides quality information and analysis for NHS organisations to implement the Diabetes NSF and ensure that resources are being utilised effectively and where they are most needed [51]. At a local level, the South Tees area there is a diabetes register, hosted by the Diabetes Clinic at James Cook University Hospital. Established in 1987, the register aims to collect demographic and clinical information on all known diabetes patients in the Middlesbrough and Redcar & Cleveland. This dataset is described in more detail in later chapters as it forms the core dataset used for the secondary data analyses for this thesis.

**Figure 1: Diabetes care pathway [52]**

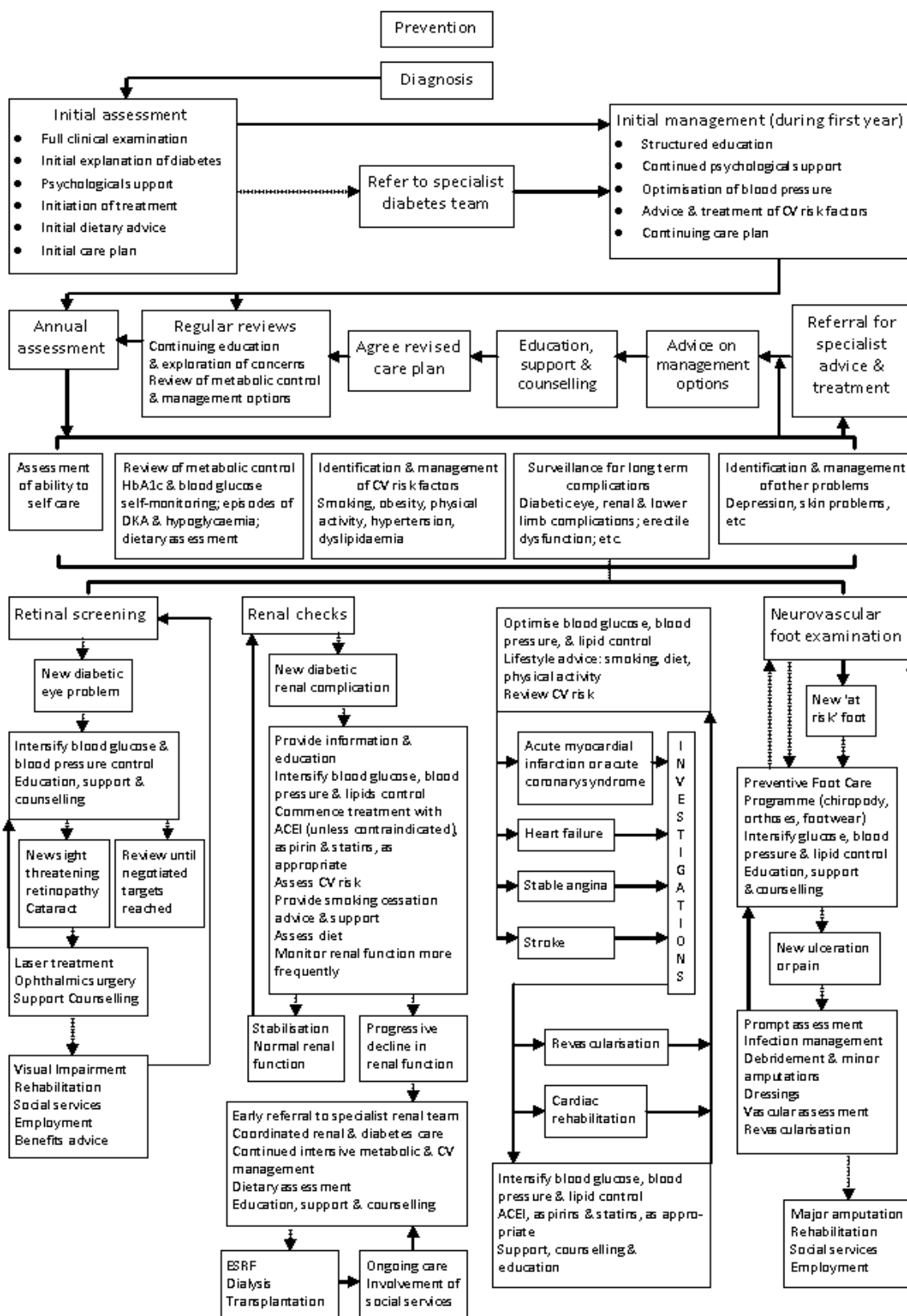
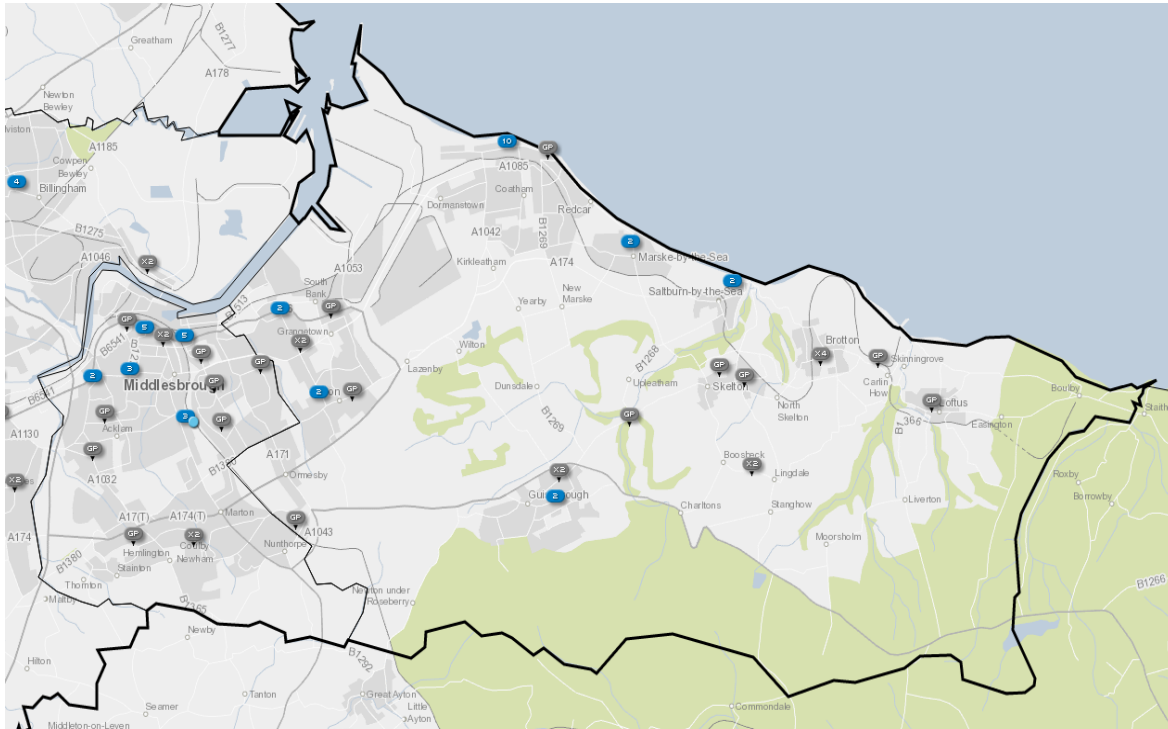


Figure 2: Primary care practices in the South Tees area [53]



This section describes the rationale for having type 2 diabetes patients in the South Tees area as the target population of the analyses described in this thesis. It then goes on to introduce some of the existing analysis illustrating the extent and implications of diabetes on individuals and resources in the area. It also introduces some of the known inequalities between social groups.

South Tees, comprising Middlesbrough Local Authority (LA) and Redcar & Cleveland LA, is a distinct geographical region in the North East of England. The area encompasses the industrial town of Middlesbrough and naturally bordered by the river Tees, the North Sea and the North York Moors [54, 55]. Mid-2010 population estimates recorded Middlesbrough LA as having a population of 142,000 and Redcar & Cleveland as 137,000. In the Public Health Observatories (PHO) for England 2012 Health Profiles, both LAs had higher deprivation and performed worse than England for a range of health indicators, including life expectancy, adult 'healthy eating' and obesity[56].

There are 49 general practices in the South Tees area [57] and one NHS Foundation Trust. South Tees Hospitals NHS Foundation Trust provides hospital and community services for patients in

Middlesbrough, Redcar & Cleveland, Hambleton and Richmondshire and other areas. The majority of patients with diabetes are expected to be managed within primary care [58], however, the trust provides additional services for patients with specific care needs. This includes general diabetes clinics and specialist clinics for pregnant women and those planning a pregnancy, young people, patients treated with continuous subcutaneous insulin infusions using semi-automated infusion and for patients with or at risk of particular diabetes related complications. The Trust also provides a community diabetes service which features a multidisciplinary team which works with the hospital and patients general practitioners. This service operates clinics in primary care hospitals and other locations and provides additional services such as training and support for patients and their primary care team, structured diabetes education programmes and up to date information on diabetes complications, new treatments and other health services [59].

As mentioned at the end of the previous section, the South Tees Hospitals NHS Foundation Trust also maintains a diabetes register hosted by the Diabetes Clinic at James Cook University Hospital. The aim of the register is to collect demographic and clinical information on all known diabetes patients in Middlesbrough and Redcar & Cleveland. Whilst the register has existed in some form since 1987, in 1999 the database was redesigned and data was no longer archived each year. Also due to time constraints in collecting the additional data from primary care, the dataset, in 2010, was only complete up until 2007. There was, therefore, an opportunity to conduct an analysis using repeated measurements at the patient level over a nine year period to compare changes in the rate of intermediate health outcomes and long-term complications [60]. In addition, it is possible to link it with other datasets enabling more features of the diabetes care pathway to be taken into account and measure patients' socio-economic status (SES) allowing for more complex analyses.

### *Type 2 diabetes in South Tees*

This section describes what is currently known about the impact diabetes has on individuals and the resources in the South Tees area, comparing the findings to other local, regional and national trends when appropriate. Due to the nature of the data available in this section diabetes refers to all types unless otherwise stated.

In South Tees in 2010, both the primary care trusts (PCTs) in Middlesbrough and Redcar & Cleveland had an estimated prevalence of 7.9%; higher than the national rate of 7.4% [40]. In

the 2009/10 NDA less than 80% of the predicted registrations were captured [50] therefore a notable proportion of the population appears to be going undiagnosed and consequently untreated.

The Yorkshire and Humber Public Health Observatory (YHPHO), the diabetes lead for PHO for England, used information on diabetes prevalence, population estimates and all-cause mortality to identify diabetes attributable deaths estimates. In England in 2005, there were 26,300 excess deaths among people with diabetes aged between 20 and 79 years. Diabetes accounted for 11.6% of all deaths in this age group. The proportion of excess deaths as a result of diabetes was similar in Redcar & Cleveland PCT to the national rate, in contrast Middlesbrough PCT had a rate of 12.3% [40].

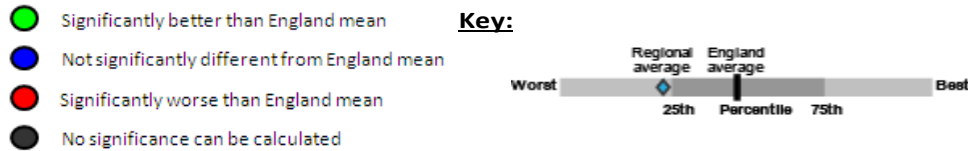
There is a mixed picture of how care has improved since the audits were introduced. Over the six audit periods, 2003/04 to 2008/09, there has been a reduction in the prevalence of ketoacidosis, myocardial infarction and retinopathy treatments for type 2 diabetes in England. However, there has been an increase in the prevalence of angina, cardiac failure, stroke and renal failure. During the same period, there has been an increase in the number of type 2 diabetes patients receiving all nine recommended NICE care processes. The proportion has increased from 10.6% to 50.8%. This was still a low rate and significant variation between population groups exists [61].

Figure 3 and Figure 4 are spine charts that show Middlesbrough PCT and Redcar & Cleveland PCT National Diabetes Audit 2007/08 indicators, respectively, compared with North East and England rates. This year was chosen as it reflects the last year of the data used for the subsequent analysis in the thesis. Earlier periods of data were not readily accessible from the Information Centre [50].

The results in Figure 3 shows that Middlesbrough PCT had statistically significantly lower prevalence of myocardial infarction, stroke, renal failure and major amputations compared to England as a whole. Figure 4 shows that Redcar & Cleveland have statistically significantly lower prevalence of ketoacidosis and myocardial infarction compared to England as a whole. However, in both spine charts where there were statistically significant differences from the national rates for the percentage of care processes and target treatments achieved for patients the South Tees PCTs performed worse. Significantly fewer patients in Middlesbrough PCT had their BMI, albumin, creatinine and smoking status recorded and achieved treatment targets in terms of glycated haemoglobin (HbA1c) and blood pressure (BP) outcomes compared to patients nationally. Whilst there are fewer significant differences between patients in Redcar &

Cleveland compared to patients nationally there were lower proportions of patients having their BP, albumin and smoking status checked and recorded.

Key1

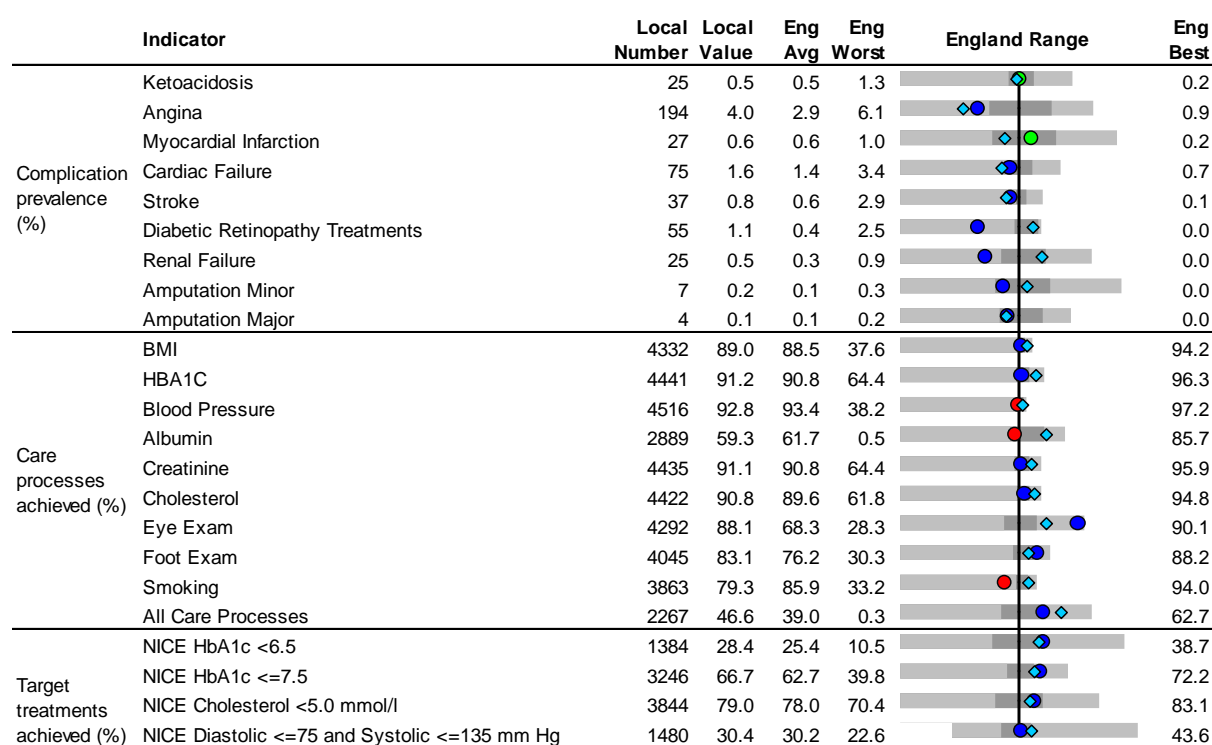


**Figure 3: Middlesbrough PCT performance compared with North East and England rates in the 2007/08 National Diabetes Audit [50]**

	Indicator	Local Number	Local Value	Eng Avg	Eng Worst	England Range	Eng Best
Complication prevalence (%)	Ketoacidosis	35	0.7	0.5	1.3		0.2
	Angina	212	4.1	2.9	6.1		0.9
	Myocardial Infarction	29	0.6	0.6	1.0		0.2
	Cardiac Failure	92	1.8	1.4	3.4		0.7
	Stroke	29	0.6	0.6	2.9		0.1
	Diabetic Retinopathy Treatments	67	1.3	0.4	2.5		0.0
	Renal Failure	13	0.3	0.3	0.9		0.0
	Amputation Minor	8	0.2	0.1	0.3		0.0
	Amputation Major	3	0.1	0.1	0.2		0.0
Care processes achieved (%)	BMI	4571	87.4	88.5	37.6		94.2
	HbA1c	4799	91.8	90.8	64.4		96.3
	Blood Pressure	4893	93.6	93.4	38.2		97.2
	Albumin	3183	60.9	61.7	0.5		85.7
	Creatinine	4587	87.7	90.8	64.4		95.9
	Cholesterol	4697	89.8	89.6	61.8		94.8
	Eye Exam	4713	90.1	68.3	28.3		90.1
	Foot Exam	4168	79.7	76.2	30.3		88.2
	Smoking	4237	81.0	85.9	33.2		94.0
	All Care Processes	2565	49.1	39.0	0.3		62.7
Target treatments achieved (%)	NICE HbA1c <6.5	1304	24.9	25.4	10.5		38.7
	NICE HbA1c <=7.5	3239	61.9	62.7	39.8		72.2
	NICE Cholesterol <5.0 mmol/l	4097	78.3	78.0	70.4		83.1
	NICE Diastolic <=75 and Systolic <=135 mm Hg	1525	29.2	30.2	22.6		43.6

<sup>1</sup> The PCT result for each indicator is shown as a circle. The mean rate for England is shown as a grey bar. A red circle depicts an area significantly worse than England for that indicator, blue depicts no significance differences and green depicts a significantly better result than the national mean. However, the results here should be interpreted with caution as a green circle may still indicate a need for improvement in diabetes care. For example Redcar & Cleveland perform well for the proportion of patients receiving all nine recommended care processes, however, even the best performing PCT only manages to achieve 70%.

**Figure 4: Redcar & Cleveland PCT performance compared with North East and England rates in the 2007/08 National Diabetes Audit [50]**



The descriptive analyses discussed here highlight that there were statistically significant differences in diabetes outcomes and care in the South Tees area. Using individual level data accessed from other sources, this thesis explores whether there are significant differences in patient outcomes and care by SES in the South Tees area. In turn, it also examines whether interventions in the diabetes care pathway are associated with inequalities in patients' health outcomes by SES.

The next chapter provides a more detailed description of type 2 diabetes and the current policy and guidelines. This is then followed by a systematic review of type 2 diabetes and health inequalities which identifies what is currently known and gaps in the evidence. The thesis then moves on to discuss the methodological considerations for undertaking such analyses, which is then followed by the methods and a series of results chapter which address each research question in turn.

## Chapter 2: Type 2 diabetes

Following the introduction about the rationale for this thesis and its focus on type 2 diabetes this chapter provides a detailed description of the condition and how it is managed in general terms. The current policy and guidelines which influence its management in England are also outlined.

### Description

Diabetes is a syndrome of metabolic disorders characterised by inappropriate hyperglycaemia resulting from defects in insulin secretion, insulin action, or both [37, 38]. There are different types of diabetes mellitus with different etiologic classifications:

Type 1 occurs as a result of pancreatic islet  $\beta$ -cell destruction. In the majority of cases this is caused by an autoimmune process, the rest are idiopathic. In adults, type 1 diabetes accounts for 5% to 10% of all diagnosed cases of diabetes [37, 38, 41, 62, 63].

Type 2 refers to a range of defects characterised mostly by insulin resistance or in some cases solely  $\beta$ -cell function, along with an impairment in compensatory insulin secretion. In other words, the body either does not produce enough insulin or the body does not effectively react to the insulin to keep blood glucose levels at a normal level. It is associated with older age, obesity, family history of type 2 diabetes, history of gestational diabetes, impaired glucose metabolism, physical inactivity, and ethnicity, specifically South Asian, Afro-Caribbean and Middle Eastern descent. In adults, type 2 diabetes accounts for about 90% to 95% of all diagnosed cases of diabetes worldwide [37, 38, 41, 62, 63].

Gestational diabetes is a form of glucose intolerance diagnosed during pregnancy occurring more frequently in certain ethnic groups, obese women and those with a family history of diabetes. Immediately after pregnancy 5% to 10% continue to have diabetes, usually type 2. Those who do not have type 2 diabetes immediately, have a 40% to 60% chance of developing type 2 diabetes within the next 5–10 years [37, 38, 41, 62, 63].

Other types of diabetes can also result from specific genetic conditions, surgery, medications, infections, pancreatic disease, and other illnesses. Such types of diabetes account for 1% to 5% of all diagnosed cases [37, 38, 41, 62, 63].

The symptoms of type 2 diabetes include tiredness, frequent urination, increased thirst, weight loss, blurred vision and frequent infections [49]. The symptoms of insulin deficiency which lead to raised blood glucose levels appear more gradually than Type 1 diabetes and usually worsen over time and with increasing age, resulting in the need for therapeutic intervention [46].

## Characteristics of disease progression

Type 2 diabetes is a progressive disorder. The development from pre-diabetes or impaired glucose tolerance stems from  $\beta$ -cell dysfunction which progresses over time.  $\beta$ -cell deterioration can occur up to 12 years prior to diagnosis and can be well advanced by the time a person reaches the diabetes range. It continues to worsen as the disease develops, therefore the next stage in patients' progression of the type 2 diabetes is the need for medication [64].

$\beta$ -cell deterioration leads to worsening glycaemic control. Continued hyperglycaemia can lead to the development of complications, which are discussed in the next section. Medication can lower patients' blood glucose but they do not completely stop the deterioration of  $\beta$ -cell dysfunction as such a patients' condition will continue to worsen over time [64].

## Complications

The greatest risk for diabetes patients is developing CVD which is five times greater than in non-diabetic patients [63]; this increases to ten times greater than the background population if the patient has experienced a previous cardiovascular (CV) event. Cardiovascular diseases include coronary artery disease (myocardial infarction and angina), peripheral artery disease (leg claudication, gangrene) and cerebrovascular disease (accidents/stroke, dementia) [46, 63].

Prolonged hyperglycaemia can also lead to microvascular complications: retinopathy, damage to eyes that can lead to visual impairment; nephropathy, damage to kidneys that can lead to progressive renal failure; neuropathy, damage to nerves that can lead to loss of sensation and function. Nerve damage can lead to foot ulcers, amputation, fainting on standing up, abnormal

sweating, gastrointestinal problems, difficulties in urination and erectile dysfunction. Other problems can include: cataracts, infections, soft tissue conditions, skin conditions and mental health problems. [46, 49, 63].

## Care

Patients' glycated haemoglobin (HbA1c) is measured, as part of the NICE recommended care guidelines, to indicate their blood glucose control over the preceding three-month period. HbA1c is formed when normal haemoglobin A (HbA) reacts with glucose in the blood. The reaction is slow and is dependent upon the amount of HbA and glucose. HbA remains in circulation for about 3 months, therefore HbA1c (%) is the amount of glycated haemoglobin proportional to total HbA over that period. Prolonged, higher levels of blood glucose can lead to atherosclerosis: fatty material building up on the walls of arteries. This can narrow or block the arteries preventing the efficient circulation of blood around the body which is needed to transfer oxygen and fuel to tissues and carry away waste products. This can lead to a number of complications [63]. Therefore, ideally, most patients should aim to have a HbA1c level of approximately 6.5% or less [46].

Patients' risk of developing many of the above complications can be reduced by maintaining optimal blood pressure (BP); as such it is also monitored as part of the NICE recommended care guidelines. Two values make up the overall BP measurement: systolic (sBP) and diastolic (dBP). sBP measures the pressure as the heart contracts to push blood through the body, dBP measures the pressure when the heart relaxes to refill with blood. In the general population elevated dBP is more common in those under 50 whereas sBP becomes a greater problem with increasing age [65]. The higher the blood pressure the more strain the arteries and heart is under increasing risk of complications such as heart attacks, stroke or kidney disease. For diabetes patients, therefore, it is recommended that this should be 130/80 mmHg or lower [46, 63].

Management of a patient's lipid profile, the fatty substances in the blood system, is also a recommended part of diabetes care as it also plays a vital role in reducing risk of complications. There are four aspects a lipid profile: total cholesterol, high-density lipoprotein cholesterol (HDL-c), low-density lipoprotein cholesterol (LDL-c) and triglycerides. HDL-c is often referred to as the 'good' cholesterol as it carries cholesterol away from the cells to the liver where it is broken down or passed out of the body as waste. Higher levels increase this process. LDL-c, the 'bad' cholesterol, carries cholesterol from the liver to the cells. Too much LDL-c leads to more

cholesterol than the cells can use leading to a build-up and the narrowing of the artery walls. This in turn increases risk of the vascular complications; likewise with higher levels of triglycerides [46].

Monitoring patient's kidney function is another recommended part of the annual, routine management of type 2 diabetes. This is to establish as early as possible if poorly controlled blood glucose levels has led to nephropathy. One method is measuring the level of creatinine in the blood. This is a chemical waste product and is usually relatively constant in a person's blood. High levels, therefore, indicate possible kidney damage. Practitioners are advised to estimate the glomerular filtration rate (GFR), that is, how much creatinine is cleared from the blood. This is a more precise measure of kidney function. Normal GFR is 100mls/min/1.73m<sup>2</sup>. A lower rate indicates a greater severity of kidney damage [46, 66].

Practitioners are also advised to measure patients albumin:creatinine ratio annually. This is done by measuring the level of protein in patients' urine. Patients are considered to have microalbuminuria if the ratio is greater than 2.5 mg/mmol for men and greater than 3.5 mg/mmol for women [46].

Routine monitoring of these health indicators on at least an annual basis forms a major part of patients diabetes care. NICE recommend that patients should have the following nine care processes recorded on an annual basis: urinary albumin, BMI, cholesterol, blood creatinine, HbA1c and BP measured, eyes and feet examined and a smoking review [46, 61]. In addition patients should expect to receive individual care planning, the opportunity to attend a diabetes education course, access to specialist healthcare professionals including ophthalmologists, podiatrists and dieticians.

## Treatment

Following a diagnosis of type 2 diabetes, if appropriate, patients are usually encouraged to treat their diabetes through changes in their diet and lifestyle. This includes advice on achieving a healthy balanced diet, increasing physical activity, losing weight and modifying alcohol intake. However, if patients are unable to achieve and/or maintain optimum levels of health outcomes introduction of personalised targets and medication are recommended [46].

Therapies are initiated for patients whose blood glucose is inadequately controlled by lifestyle interventions alone. In the first instance metformin is usually prescribed. Sulphonylureas can be

considered as a first line therapy if patients are not overweight or obese or if their blood glucose levels are particularly high. If blood glucose levels continue to be inadequate or worsens another therapy, usually a sulphonylurea, is added. Rapid-acting insulin secretagogues are considered for those with an erratic lifestyle and acarbose for those who cannot use other oral glucose-lowering medications. Glitazones can be introduced in combination with metformin and/or a sulphonylurea when insulin is either unacceptable or inappropriate for various reasons. Insulin is usually the last blood glucose therapy to be introduced. Education for both patients and their carers should be offered and local arrangements made for the safe disposal of sharps.

Lifestyle modification is also aimed at improving patients BP levels. However, medications are initiated if it is not maintained below 140/80 mmHg. In the first instance a once daily, generic angiotensin-converting enzyme inhibitor (ACEI) should normally be prescribed. For Afro-Caribbean patients this is usually introduced with a diuretic or a generic calcium channel blocker. In patients with a continued intolerance of ACEIs, a substitution for an angiotensin II-receptor antagonist is recommended. Other therapies are introduced if BP levels do not reduce or deteriorate. These include alpha-blockers, beta-blockers, or potassium-sparing diuretic. These therapies are prescribed for patients who have kidney damage.

Unless patients have a low CV risk all patients aged 40 years old and over should be prescribed a statin. Prescription of a fibrate is recommended for patients whose triglyceride levels are continually above 4.5 mmol/l, or between 2.3-4.5 mmol/l despite statin therapy being initiated. Aspirin is offered to patients as an antiplatelet therapy [46].

These medications should be all continually reviewed in reference to how patients' health develops, both in terms of intermediate and long term complications. Patients' personal circumstances are also taken into consideration when treatments are being initiated [46].

In summary this chapter so far shows how the management of type 2 diabetes is very complex [46]. The next section outlines the current English national policies and guidelines, detailing some of the specific healthcare recommendations.

## **National policies and guidelines**

This section describes the recent developments which have led to the current policies and guidelines for diabetes, with a particular focus on type 2 diabetes and the English context.

In 1997 'The New NHS: Modern, Dependable' white paper introduced two initiatives that have influenced diabetes care in the UK in recent years. Firstly, the National Institute for Health and Clinical Excellence (NICE), who now lead on clinical and cost-effectiveness and provide guidelines for health and social care services, and the National Service Frameworks (NSF), which provides evidence-based strategies for consistent access and care quality nationally [67].

The National Institute for Health and Clinical Excellence was established in 1999 known as the National Institute for Clinical Excellence with the remit of reducing variation associated with NHS treatments and care. The organisation then merged with the Health Development Agency in 2005 to become National Institute for Health and Clinical Excellence as the prevention of ill health and the promotion of good health were incorporated into its remit. Following the Health and Social Care Act 2012, it became a Non Departmental Public Body becoming accountable to the Department of Health and taking on responsibility of developed social care guidance and quality standards under its current title [68].

The 'National Service Framework (NSF) for Diabetes: Standards' was published in 2001. Twelve standards and key interventions were outlined in the document designed to improve care by being patient focused. It was developed in partnership with a multidisciplinary team drawing upon skills and knowledge across services. Furthermore, the NSF for Diabetes aims to ensure that services are equitable according to individuals' needs and outcomes, that is narrowing the gap between patients with the worst outcomes and the rest [52]. The 'NSF for Diabetes Delivery Strategy', 2003, was designed to support the achievement of the 'Standards' by 2013 the key elements of which were expected to be undertaken by PCTs. This included setting up a local network to champion the needs of local people, reviewing local baseline data and implementing local arrangements, participation in local and national audits, and developing education and training programmes for staff involved in diabetes care [49].

The delivery strategy is underpinned by the clinical framework for diabetes developed by NICE and it is recommended that they should be used together with the most up to date information [49].

In March 2010, NICE updated their guidelines 'The management of type 2 diabetes'. This replaced those published in 2008. The 2008 guidelines were an updated version of individual guidelines on diabetes care on retinopathy, renal disease, blood glucose and management of BP and blood lipids all published in 2002 and other NICE technology appraisals [69]. The key NICE recommendations, which include aspects of care and treatment targets, form the basis of the National Diabetes Audit (NDA). There are nine care processes which every diabetes patient should have measured and recorded annually. These are as follows: HbA1c, BMI, BP, albumin,

creatinine, cholesterol, eye exam, foot exam and smoking status. Each are chosen on the basis that they are risk factors or indicators of vascular damage and direct the types of interventions a patient requires [46, 63]. In addition, treatment targets are set to ensure that patients' health outcomes are at safe levels. The treatment targets are as follows: HbA1c of either <6.5% or ≤ 7.5% depending upon the health and treatments of the patients, cholesterol of <4.0 mmol/l and a target for BP of ≤ 140/80 for those without recorded eye, kidney or vascular disease or ≤ 130/80 for those with recorded eye, kidney, or vascular disease. The NDA also records the prevalence of the following complications: angina, myocardial infarction, cardiac failure, stroke, diabetic retinopathy treatments, renal failure and amputations [61].

In March 2011, NICE published quality standards for clinical best practice for adults with diabetes. The aim of which was to outline high-quality and cost-effective care to be delivered collectively to improve effectiveness, safety and experience for patients [46]. The introduction of these standards has been praised by patient advocates for emphasis on involving patients in their own care but that they should be wider to incorporate more aspects of patients care and evaluated to see whether high quality is achieved [63]. Other relevant NICE guidelines include TA248: Diabetes (type 2) – exenatide (prolonged release), CG119: Diabetic foot problems – inpatient management, CG87: Type 2 Diabetes – newer agents (partial update of CG66), CG66: Type 2 diabetes (partially updated by CG87) [46] and CG10: Type 2 diabetes – foot care [70].

In the UK, another important initiative associated with diabetes care is the 'Quality and Outcomes Framework' (QOF) which is part of the General Medical Services contract [71]. QOF was one of the consequences of the Labour government's aim to expand chronic disease management into primary care [58]. The framework is a voluntary annual reward and incentive programme established in general practices across the UK during 2004 with the aim to reward the provision of good quality care and improve standards [47, 72]. Whilst QOF has all the same care processes that NICE recommends for the management of type 2 diabetes featured in some form, the care targets vary. In particular, there are lower thresholds of intermediate outcomes in order for patients' results to count towards the payments. Another drawback of QOF as an incentive to improve standards and quality of care is the ability of practices to use 'exception reporting' so that they are not penalised financially for various criteria including patients who do not attend for review, or refuse treatments and/or investigations [47]. The criticism of this approach is that patients could potentially be inappropriately excluded in order for practices to boost their payments. Also it has been shown that exception reporting is associated with the deprivation of patients. As a result the added incentive to improve the quality of care for "exempted" patients, by implication the most deprived patients, circumvented leading to no improvements in standards in comparison to the included patients [73].

This chapter has provided a broad overview of type 2 diabetes and the prominent guidelines and policy recommendations regarding the management of the condition in England. The subsequent secondary analysis examines whether interventions related to the type 2 diabetes care pathway are delivered equitably and whether these intervention differ in their association with health outcomes by patients SES, that is, the identification of potential IGIs. The next chapter features the systematic review which searched for and assessed the current evidence surrounding health inequalities associated with type 2 diabetes.

### **Non-diabetes specific interventions**

Interventions aimed at improving population health, particular increasing healthy behaviours, will also impact on the prevention of type 2 diabetes and the management the condition in diagnosed patients [references].

## **Chapter 3: Are there inequalities associated with interventions to manage and treat Type 2 diabetes?**

Following the introduction to the rationale and contextual information, this chapter presents a systematic review of the current evidence surrounding health inequalities associated with type 2 diabetes. The review had two main aims. The principal aim was to answer the chapter title question: Are there inequalities associated with interventions to manage and treat Type 2 diabetes? The secondary aim of the review was to identify areas where evidence was lacking and if the methodology of previous research could potentially be improved upon in order to develop the research questions outlined at the end of the chapter.

### **Methodological considerations**

A previous systematic review published in 2010 examined inequalities associated with diabetes and concluded that there was evidence of inequalities in treatment, control and service utilisation by ethnicity, socioeconomic inequalities in diagnosis and control, but no evidence of gender inequalities [74]. The present review revises and updates the 2010 review. Here the review focuses on type 2 diabetes only but with a more inclusive search strategy. In addition, a different approach was taken to the critical analysis of the final sample which enabled the quality of the methodology and design to be examined separately and graphically synthesised.

This systematic review focuses on inequalities in the management and treatment of type 2 diabetes from the point of diagnosis. Interventions associated with prevention were not included as they are often provided by organisations and services not specifically orientated to the management and treatment of diabetes [75]. The eligible studies were also limited to patients aged 16 and over with type 2 diabetes as adult services are often delivered differently to those provided for children and adolescents [76]. Studies examining inequalities in mortality were also excluded as this was the subject of recent review [44]. Due to the numerous related health complications that could be investigated as a consequence of type 2 diabetes studies were further limited to those examining outcomes which are routinely monitored, as recommended by NICE Type 2 diabetes guidelines [46].

Like the 2010 review, the search strategy was limited to studies which were undertaken in countries belonging to the Organisation for Economic Co-operation and Development (OECD) with universal healthcare. This approach was chosen as the health systems and high economic development of these countries means that they are in a better position to prevent health inequalities than other countries [74]. The search was limited from 1st January 1998 to the date of abstraction, 6<sup>th</sup> August 2012. The start date was chosen as it was the year of the Acheson Report [77], an influential publication which shaped the subsequent health inequalities agenda both in the UK and worldwide. The 2010 review searched between 1967 and 2007. However, only one study prior to 1998 was included in the final sample and this focused on type 1 diabetes only.

The search strategy adopted here was chosen for its arguably more inclusive approach. The previous review used Medical Subject Headings (MeSH) and their equivalent terms [78]. It has been argued that using these terms leads to a more efficient search strategy than free text searches as they limit the results to specific subjects and their related terms [79]. However, this approach is reliant on these terms being assigned to studies and for this to be undertaken correctly and in a timely manner. There can often be a time delay in adding subject headings of up to three months therefore searches using subject headings may miss the most up to date research. A further problem, particularly with this review, is that the subject matter is quite broad and specific subject headings may not appropriately capture the topic under review [79]. A free text approach, therefore, was used instead. However, to ensure that the hits were relevant, the search was limited to studies which featured “diabetes” or “diabetic” in the title and/or abstract in common with the 2010 review. This is necessary as the drawback to free text searches is its low sensitivity. That is, they tend to generate a lot of hits, due to capturing articles containing the free text words, even if they are unrelated to the subject matter [80].

The data from the studies were extracted and entered into Access 2007 [81] following an adapted version of a data extraction form used in another review examining inequalities associated with health interventions [82]. Study quality was assessed using the ‘Data Collection Instrument and Procedure for Systematic Reviews in the *Guide to Community Preventive Services*’ (CPS). This instrument was chosen as it was designed to be flexible enough to evaluate the reliability and validity of a diverse range of study designs and intervention types [83]. Other common quality assessment tools would have had to be considerably adapted specifically for this review. The 2010 review used the STrengthening the Reporting of OBservational studies in Epidemiology (STROBE) Statement. However, this tool assesses the reporting quality rather than methodological quality of observational studies [84].

The most distinct change from the previous review is in the use of 'harvest plots' to graphically synthesise the data. The key strength of harvest plots is that it can accommodate heterogeneous studies, both in terms of outcomes and quality of study design, therefore making use of all the available evidence. The graphical plots can display multiple of aspects of each of the studies allowing it to retain the immediacy of interpretation of the original 'forest plot' [85].

## Methods

### Search strategy

The search strategies are outlined in Appendix A. The databases searched were PubMed, Excerpta Medica database (EMBASE), Cumulative Index to Nursing and Allied Health Literature (CINALH) and Applied Social Sciences Index and Abstracts (ASSIA). All the 'hits' were stored in Endnote X6 [86].

### Study selection and inclusion criteria

Studies were included if they met the following criteria:

- i. It was a primary study.
- ii. The study analysed the association between one or more population sub-groups based on gender, ethnicity including immigrant versus native populations, or socioeconomic position, including individual and area-based measures, and any one aspect of type 2 diabetes interventions that are part of the patients' usual care available.

These were subsequently grouped into 5 categories: diagnosis, treatment, control, monitoring, services which are normally available for type 2 diabetics. This was done to provide a more coherent discussion of the findings and more concise harvest plots. Prevention, screening and education programmes were excluded as this review focused from the point of diagnosis onwards on services typically delivered within primary and secondary care settings.

- iii. The primary health outcomes, if included in study, were those routinely monitored by health care professionals, as recommended by NICE Type 2 diabetes guidelines [46].
- iv. These are broadly described as follows: blood glucose, plasma glucose, blood pressure, blood lipids, eye damage, nerve damage, kidney function and cardiovascular disease [46].
- v. Had quantitative outcomes in terms of access to, uptake of, or outcome of the interventions
- vi. Patients of the primary studies had to be 16 years old or older with type 2 diabetes
- vii. The study had to be undertaken in community settings, that is, participants were not in residential institutions such as care homes or prisons.
- viii. Carried out in an OCED countries with universal healthcare: Australia, Austria, Belgium, Canada, Denmark, Finland, France, Germany, Greece, Iceland, Ireland, Italy, Japan, Luxembourg, The Netherlands, New Zealand, Norway, Portugal, Spain, South Korea, Sweden, or United Kingdom (England, Northern Ireland, Scotland or Wales) [87].
- ix. Published in English
- x. Published from 1<sup>st</sup> January 1998 to 6<sup>th</sup> August 2012, the date that the databases were searched.

All study designs with original results were included. Articles not related to type 2 diabetes, not original (e.g. narrative reviews, letters, editorials, opinion articles etc.) and qualitative only studies were excluded. Titles and abstracts were initially assessed for inclusion. The full texts of those which met the inclusion criteria were then read; those which did not meet the criteria were then excluded leaving the final sample.

### **Data extraction and quality assessment**

The data from the studies were extracted and entered into Access 2007 [81] following an adapted version of the data extraction form [88]. Study quality was assessed using the 'Data Collection Instrument and Procedure for Systematic Reviews in the Guide to Community Preventive Services' (CPS) [83].

The CPS tool has five components relating to the descriptions, sampling, measurement, data analysis and the interpretation of results. In order to produce graphical syntheses of the data for this review, a scoring system was created in which a point was awarded if the study covered the relevant aspects of each component, with a maximum score of five. The suitability of the study design was assessed using a further adaption of the scale developed by Thomas *et al* [88]: 1 = cross-sectional studies; 2 = more than one measurement at different time periods and no comparative group; 3 = more than one measurement at different time periods with a comparative group.

## Data synthesis

The interventions were grouped into the following categories: diagnosis, monitoring, control, treatment or services. Due to the diverse range of interventions that can affect the management and outcomes of type 2 diabetes, the majority of studies fall into more than one category and therefore many studies were included more than once in the data synthesis. The hypothesis-testing approach to data synthesis devised by Thomas *et al* [88] was used to examine the differential effects of each group of interventions separately. Each study was categorised depending upon which of the following hypotheses its findings most supported for each intervention:

The null hypothesis was that for any given demographic or socio-economic characteristic there are no inequalities in the effectiveness of the intervention.

The hypothesis of negative impact on social inequalities, defined as evidence that groups with a higher SEP gain the greatest benefit from the intervention.

The hypothesis of positive impact on social inequalities, defined as evidence that groups with a lower SEP gain the greatest benefit from the intervention.

Though not ideal, non-white ethnicities and immigrants, women and patients described to be in rural areas were considered the group with the more disadvantaged social group in respect to their counterparts.

The results were graphically synthesised using the harvest plot method developed by Ogilvie *et al* [85] and used in two previous systematic reviews [82, 89]. In a harvest plot the rows consist of different axes of inequalities and three columns reflect the three competing hypotheses. Each

bar refers to one comparison from one study with the height of the bar indicating the suitability of study design and the number annotated above it indicating the methodological quality [85].

In this review a series of harvest plots were produced for each group of interventions using the data visualisation software Tableau Public 6.0 [90]. In the harvest plot each bar refers to one comparison from a study; studies that examined more than one intervention and more than one inequality are represented through multiple bars. The height of the bar represents the suitability of study design; here there are three possible heights with the tallest being the most suitable. Each bar is annotated with a number representing the methodological criteria which could be from zero to five, with five indicating the greatest quality. In this review, the colour of the bar indicates the consistency of the results: dark blue indicates that all or the vast majority of the results in that study support that hypothesis, the light blue indicates that there are findings which conflict with the overall result. If all of the outcomes in the study are in conflict it is marked down as representing the null hypothesis. A judgement was made on a case by case basis as to whether these conflicting findings support the null, negative or positive hypotheses. A narrative review accompanies each graphical display.

## Results

2,758 references were identified: 938 in PubMed, 1737 in EMBASE, 213 in CINAHL and 99 in ASSIA with 1088 duplicates. Of these 33 met the inclusion criteria and were critically assessed. Many of the included analyses examined more than one intervention and/or inequality, and therefore appear in the graphical displays and narrative reviews below multiple times.

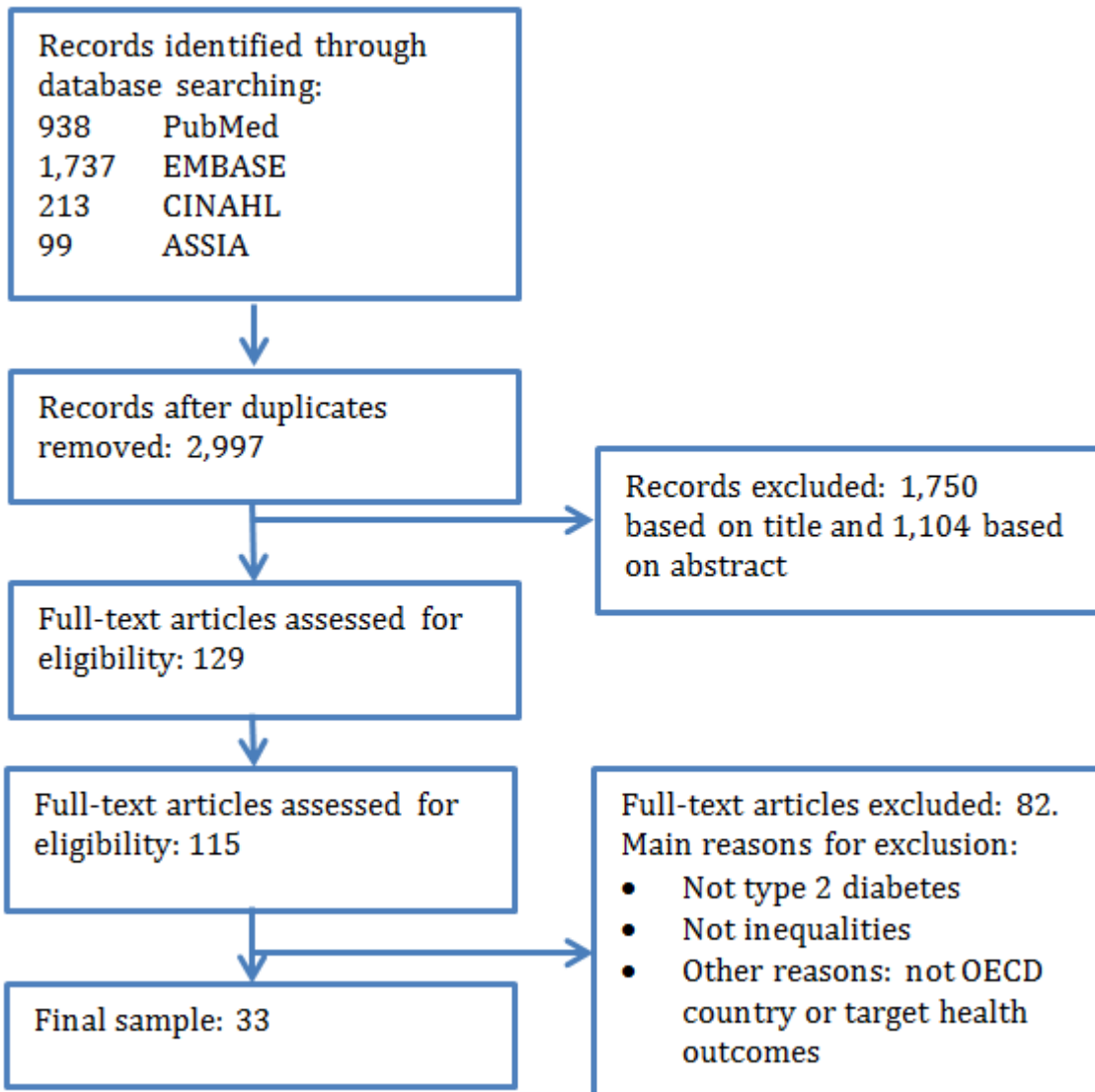


Figure 5: Flow diagram of study selection and exclusion

## Diagnosis

Figure 6 is the first of the five harvest plots and is the least populated. Only three studies met the inclusion criteria which examined if there were inequalities in the diagnosis of type 2 diabetes. The focus of these studies was inequalities in the severity of diabetes related symptoms at diagnosis [91] and being clinically diagnosed or not [92, 93]. Two studies collected data via surveys [92] [93]. One survey randomly recruited participants from general practitioners' lists in the UK [92]. The other survey recruited participants from the resident population of Augsburg, Germany [93]. A health service database held by Southampton University NHS Trust, UK was used for the other study [91].

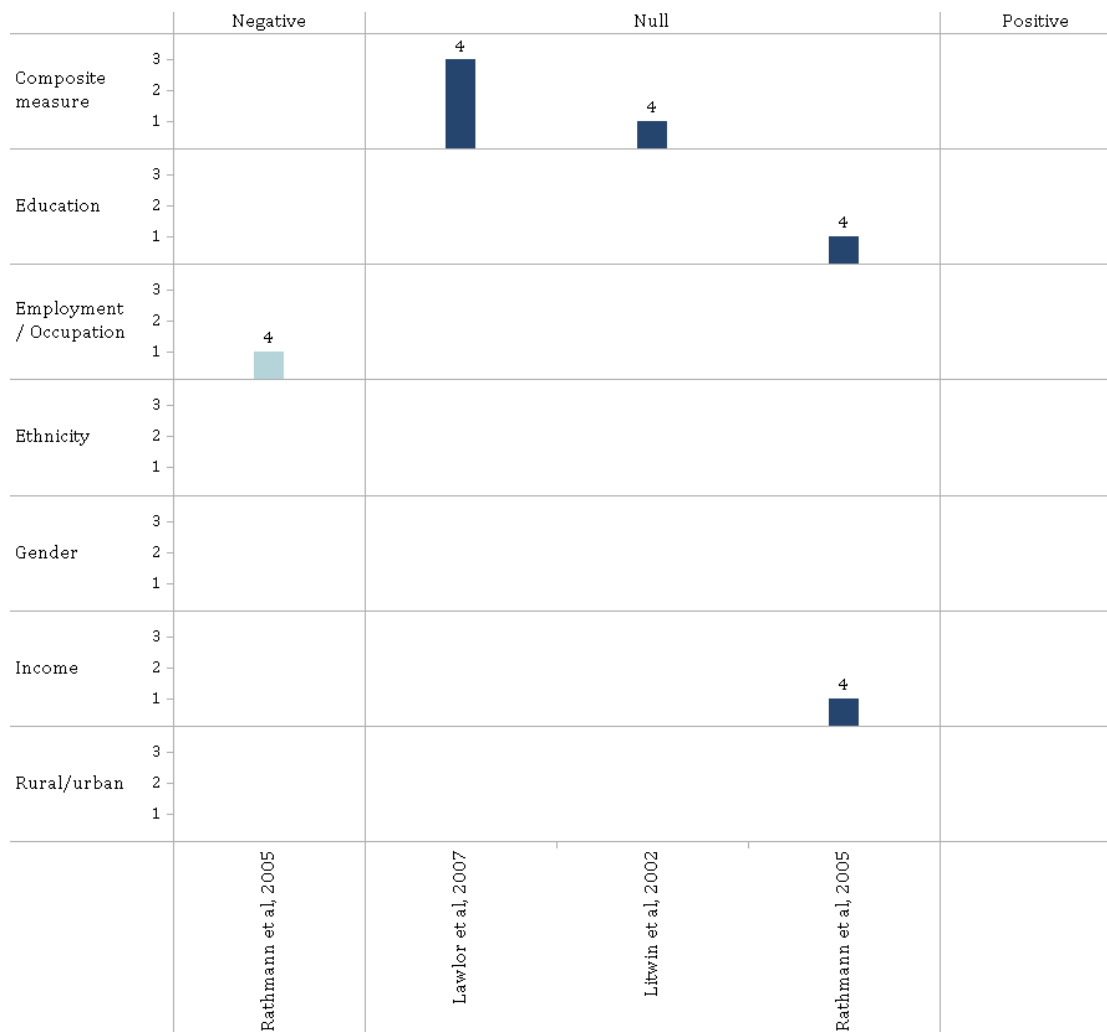


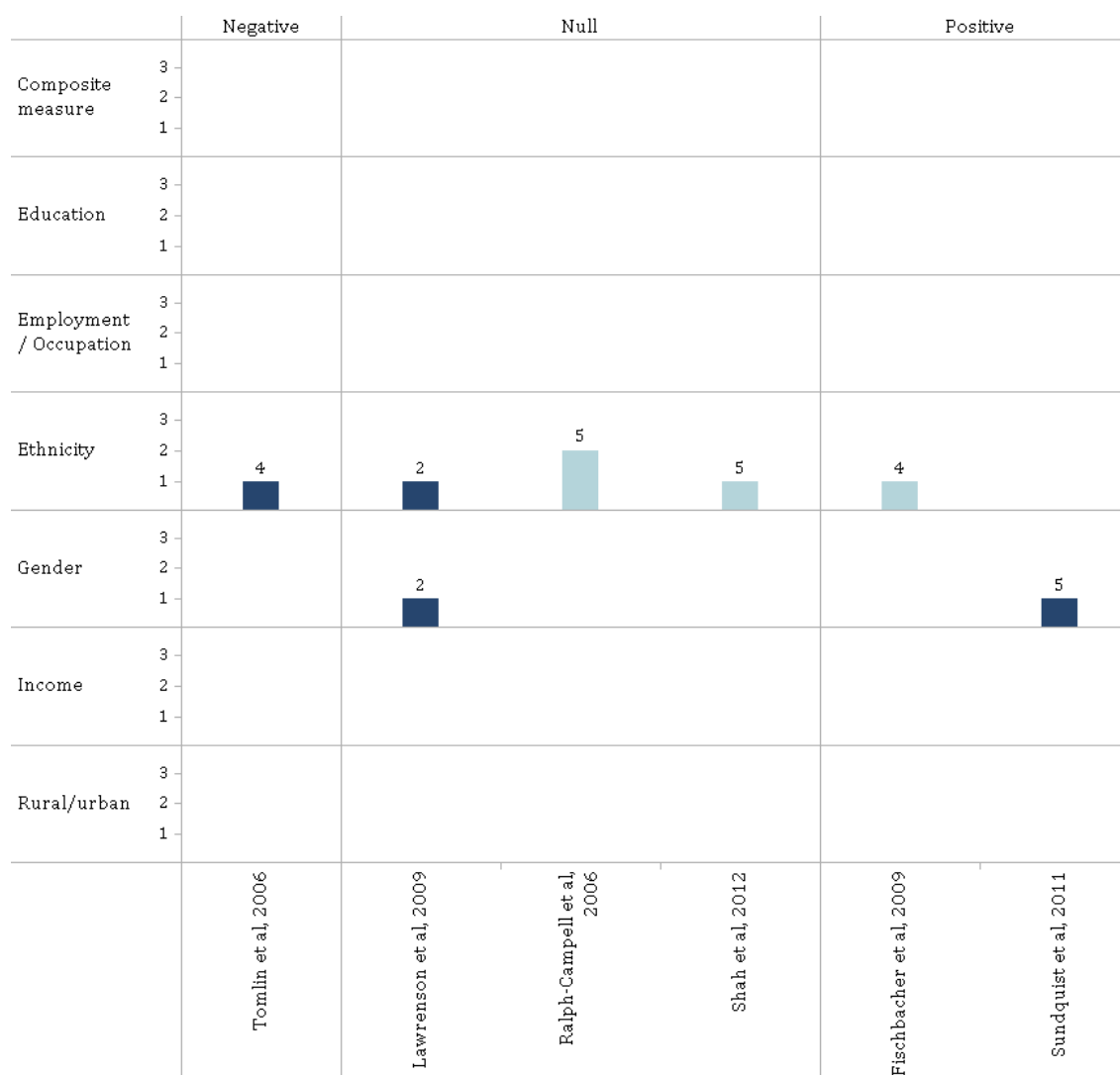
Figure 6: Evidence of inequalities in the diagnosis of type 2 diabetes patients

In the UK, analyses of the results of newly diagnosed patients' first retinal screening visit found no differences in the levels of deprivation of patients who had retinopathy compared to those who did not. Interestingly, an additional analysis of those who had a longer delay between diagnosis and first screening (24-72 months) found that those who had retinopathy were more affluent than those who did not; however, statistical significance levels were not reported [91]. This study only used univariate analyses and therefore did not control for multiple variables simultaneously.

A nationwide UK women-only study found no inequalities by childhood or adult SEP in having undiagnosed diabetes, even after controlling for lifestyle and anthropometric indicators. There was a longitudinal aspect of this study. However, it was examining hazard ratios for all-cause mortality and therefore did not meet the inclusion criteria. In addition, it only compared non-diabetic women with diabetic women and did not keep those undiagnosed at baseline as a separate group. As such it was not possible to assess the long-term implications of the potential delay in diagnosis [92]. After controlling for lifestyle, anthropometric and clinical characteristics a German study found no inequalities in being diagnosed or not by income or education for men and women. No inequalities were found by occupation status in being diagnosed for men, but there was a statistically significant association showing women with low occupational status were more likely to have undiagnosed diabetes than women with high occupational status [93]. The two surveys found conflicting evidence for inequalities by SEP in being diagnosed or not for women; however, the SEP measures used in these two studies were different.

The results presented here favoured the null hypothesis indicating that there were no inequalities associated with timely diagnosis of type 2 diabetes. However, there were only a small number of studies using cross-sectional study designs focusing on different population groups. As such, additional evidence is required to reinforce the current evidence.

### **Monitoring by health professionals**



**Figure 7: Evidence of inequalities in the monitoring of type 2 diabetes patients**

Six studies investigated inequalities associated with the monitoring of type 2 diabetes between 2006 and 2012, recruited from a variety of sources (Figure 3)[94-99]. Studies were undertaken in Britain [99], Sweden [95], Canada [96, 97] and New Zealand [94, 98].

Five studies examined inequalities in the monitoring of clinical characteristics by healthcare professionals by ethnicity [94, 96-99] and two by gender [95, 98]. Of these six, two had samples exceeding 10,000 participants. However, they had different results regarding ethnicity: the study based in Tayside, Scotland found a statistically significant association showing South Asians were more likely to have a structured review than non-South Asians patients. There were no statistically significant relationships between ethnicity and other checks except for BMI which was more likely to be recorded among South Asian women than non-South Asian women [99]. In contrast, the New Zealand study's statistically significant results showed that more New

Zealand Europeans than Maori and Pacific Islanders had both foot checks and retinal examinations. These analyses compared differences in proportions using chi-square tests without adjustment [94]. Another New Zealand study using Waikato Regional Diabetes Service data from three general practices found no statistically significant differences between both gender and ethnic groups for the odds of having had retinal screening over a two year period. Utilising marginal logistic regression, these analyses included ethnicity, gender and duration of diagnosis in the final model and also adjusted for the correlation between patients from the same practice. This adjustment suggests that patients' practice affects the odds of having retinal screening recorded. It would have been interesting to know if there were any statistically significant inequalities prior to this adjustment to explore this suggestion [98]. Only the two New Zealand based studies were comparable in terms of outcomes measures and ethnic groups [94, 98]. Ralph-Campbell *et al* examined differences in screening activities between aboriginal and non-aboriginals in Northern Alberta at baseline and 6 months later. Of the five activities Aboriginal patients were less likely to have their kidneys checked at baseline and eyes checked at six month follow up than non-Aboriginals [97]. Shah *et al* compared differences in process measures between Chinese, South Asian and the general population and found that Chinese patients were less likely to have their feet examined compared to the general population but no other significant differences [96]. Both these studies were of high methodological quality.

Two studies examined inequalities in monitoring by gender but had different results. Using adjusted odds ratios, the New Zealand based study found no significant differences in receiving retinal screening by gender [98]. In univariate analyses in Sweden, women had poorer recording levels of HbA1c and blood lipids compared to men [95].

Overall, the results suggest that there were no inequalities associated with the monitoring of patients health by ethnicity. In contrast, the most robust study suggests that there were inequalities associated with monitoring by gender. Most studies achieved a methodological quality score of four or five but only one study used repeated measurements. The harvest plot in Figure 7 highlights that the majority of these studies examined inequalities in monitoring by ethnicity with none analysing differences by measures of SEP or area type therefore research is required to fill these gaps in the evidence.

## Treatment

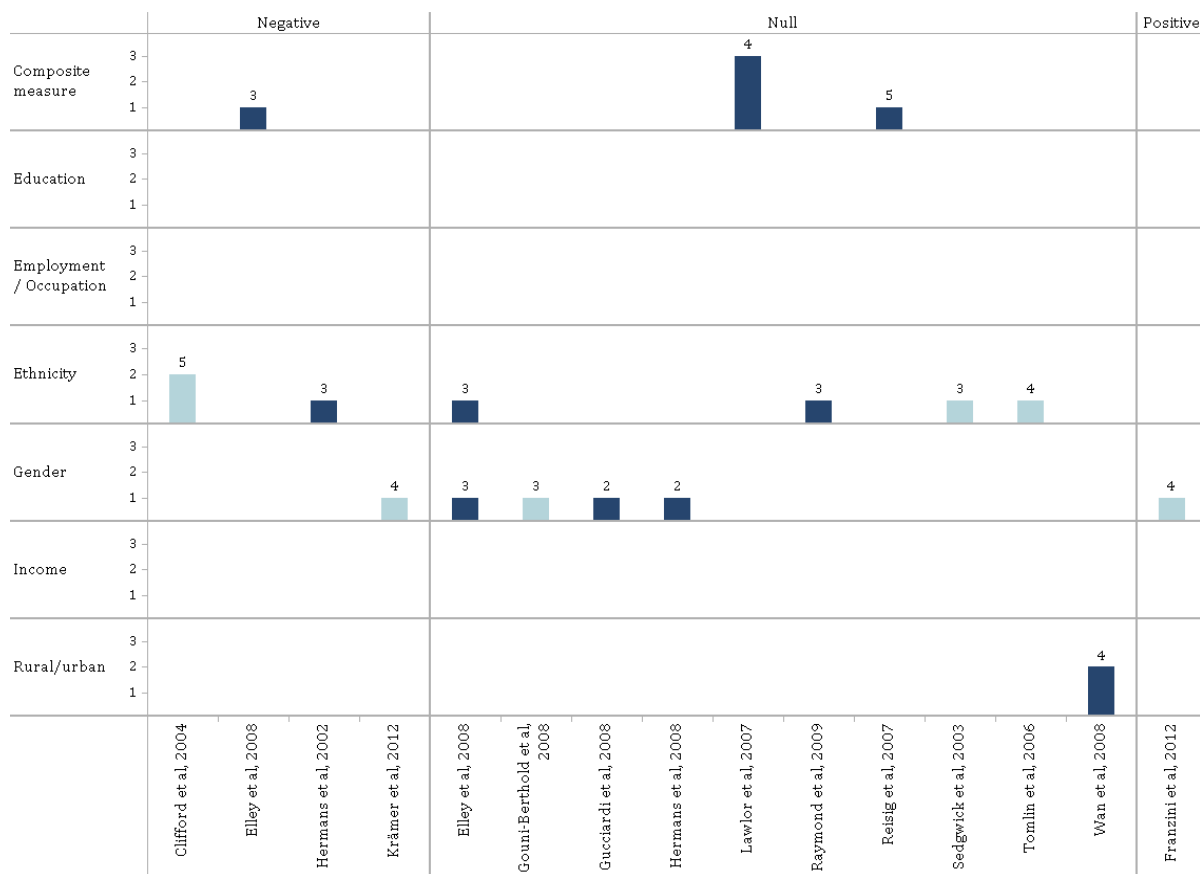


Figure 8: Evidence of inequalities in the treatment of type 2 diabetes patients

Between 2002 and 2012, fourteen studies examined inequalities associated with treatment for blood glucose control and/or associated diabetic health problems [92, 94, 100-111]. Seven of these studies had inequalities in treatments as the main focus of their study [92, 100-102, 106, 110, 111]. All participants were recruited through health services in seven different countries, with three studies based in the UK [92, 107, 109]. The majority of the evidence found no inequalities in the treatment of type 2 diabetes and its associated complications.

Of the seven studies which had treatments as the main focus of their study, three utilised repeat measurements [92, 100, 110]: An examination of medication involved in the CV risk management over time found no statistically significant inequalities between rural and urban participants in Australia [110]. Whilst this study adjusted for age and sex and used multilevel analyses to account for any potential clustering within practices and divisions, the authors did not take into account the clinical characteristics that determine the receipt of particular treatments. A UK prospective study looking at insulin use found a non-significant trend that southern European participants were more likely to be taking insulin after four years of follow-up. Yet found no differences by ethnic groups in time to requirement for insulin; nor the

progression to insulin following adjustment for demographic and health status in the Cox proportional hazards model [100]. Another UK prospective cohort study found no evidence of inequalities in being managed by diet alone by childhood or adult SEP [92].

The cross-sectional studies which had inequalities in treatment as part of the main focus of their study all examined differences by gender [101, 102, 106, 111] and with one by ethnicity and SES as well [101]. The New Zealand based study controlled for CV risk when investigating inequalities in treatment. Investigators found no evidence of inequalities by ethnicity and gender but patients from low status groups were less likely to receive treatment, with statistical significance as measured by 95% confidence intervals. However, this study had a relatively low methodological quality score [101]. A German study also controlled for CVD risk factors when examining inequalities in antihypertensive agents, lipid-lowering drugs and oral anti hyperglycaemic agents (OHAs) or insulin by gender and found in the main no significant differences. This study did find that women received significantly less lipid-lowering drugs amongst those with CVD, but not amongst those without CVD [102]. In Italy, analyses of 10 hospital-based outpatient clinics found that women were more intensively treated than men, after controlling for obesity and age [111]. In contrast, Kramer *et al* found that it was men who were more intensively treated when controlling for a more extensive set of covariates [106].

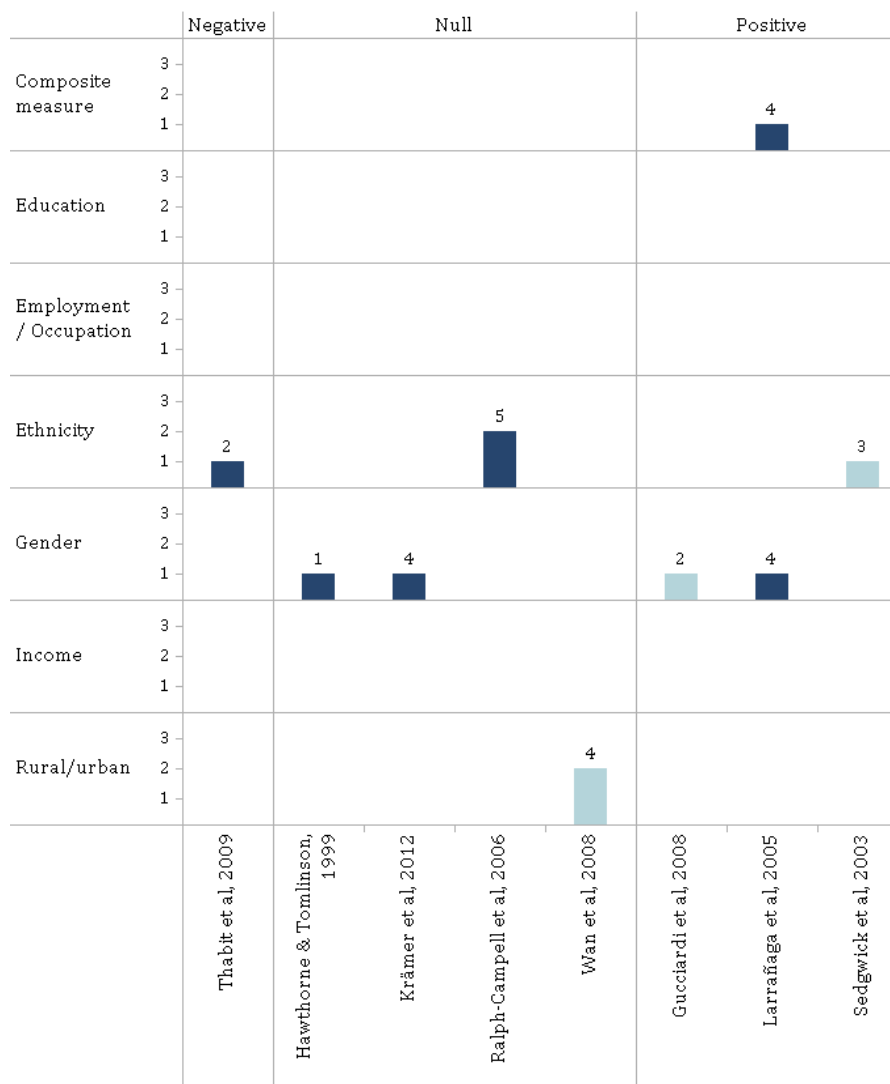
The other studies examined differences in treatment uses, either as part of an overall examination of diabetes care or in describing the socio-demographic and clinical characteristics of the studies' patients [94, 103-105, 107-109]. The overall standard was relatively low with the majority of these studies having a methodological quality of three or less and only one adjusted the analysis to consider the health status of the participants [109]. Sedgwick *et al* examined the differences in insulin use between ethnic groups in south London and found no statistically significant differences before or after adjustment for demographic, socio-economic and health status indicators [109]. Of the rest of the studies only one analysis found inequalities in treatment. However, it was not possible to establish whether the findings reflect differences in access to treatment or differences in clinical profiles [105].

The majority of the studies supported the null hypothesis i.e. that there are no inequalities associated with treatments for type 2 diabetes and its related complications by ethnicity [94, 101, 107, 109], gender [101-104], rural/urban areas [110] or other composite measures of SEP [92, 108]. However, three studies found evidence for inequalities in treatment by individual measures of SEP [101] and gender [106, 111]. In addition, the strongest study design, which examined inequalities by ethnicity, supported the negative hypothesis [100]. Overall, the

harvest plot shows that whilst there has been varied investigation into inequalities in treatment interventions, there are still areas of inequalities that have not been sufficiently explored.

## Services

Figure 9: Evidence of inequalities in uptake of and access to services for type 2 diabetes patients



Eight studies examined inequalities in the access to and uptake of diabetes related services available to diabetes patients between 2003 and 2012 [97, 103, 106, 109, 110, 112-114] (Figure 9).

One of the two studies using repeated measurements examined inequalities in referrals to ophthalmologist and optometrists and attendance at other allied professionals (diabetes educators and dieticians) between rural and urban patients in Australia general practices. However, following adjustment for age, sex and levels of care the only significant result was found in 2000, and not 2002, where urban patients were more likely to visit ophthalmologist and optometrists compared to rural patients [110].

Univariate analyses between Irish and immigrant patients attending the same diabetes clinic found a statistically significant result that the latter were more likely to have never attended a dietician [114]. A London based cross-sectional survey found patients from black African and black Caribbean ethnicities were significantly more likely to visit a dietician than white patients after adjustment for demographic, socio-economic and health status variables. This study also found statistically significant results showing that black Caribbean patients were more likely to have visited a diabetes nurse and black African patients more likely to have visited an ophthalmologist than white patients. These findings remained significant following the same adjustments [109].

Findings from two diabetes education centres in Canada of newly referred patients found that there were no gender inequalities in access to patient services and continual access to services, but a statistically significant result showed women were more likely to have a professional health care team support for diabetes than men [103]. Another Canadian, longitudinal study examined inequalities between Aboriginal and non-Aboriginal patients and found no inequalities at baseline and six month follow up in terms of physician visits, both in general and specifically for diabetes. However, the authors did find that Aboriginal patients had a higher average number of physician visits overall at baseline after adjusting for covariates [97]. Inequalities in physician visits was also examined in a German study and found no differences by gender [106]. In contrast, multivariate analyses of patients in the Basque country, Spain found statistically significant relationships for patients of lower SES to have had more primary care consultations than affluent patients and for women having more than men of the same socio-economic group. Duration of diabetes was adjusted for in the analyses. Whilst this adjustment was arguably an appropriate decision because diabetic patients are likely to develop more complications over time it does not account for those with more uncontrolled diabetes and for health problems which are not a result of their diabetes and require more contact with health services [113]. In the UK, a British sample of Pakistani Moslems in Manchester found that there were no gender differences in terms of place of care [112]. However, these studies lacked in depth analyses as they only used univariate techniques.

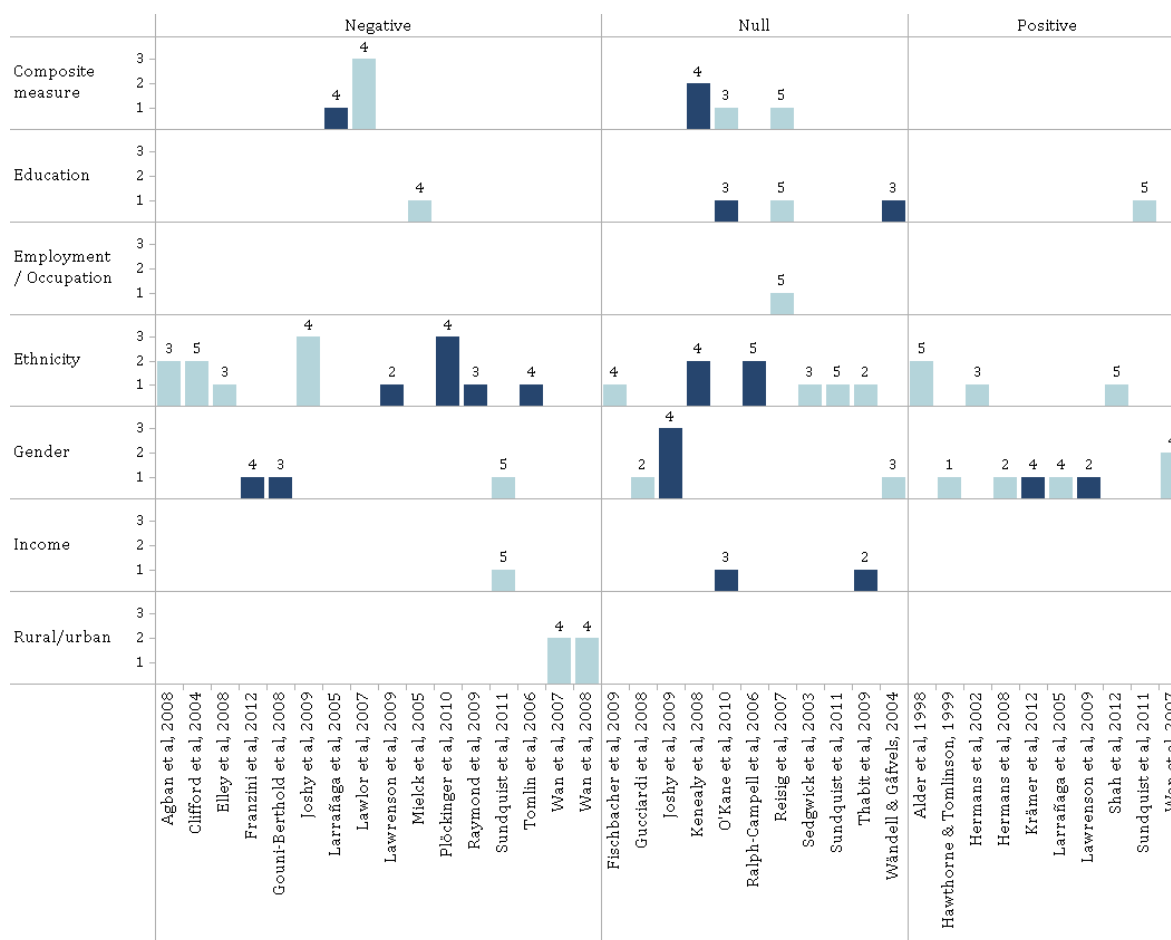
In terms of inequalities associated with services there was a fairly even spread of evidence for negative, positive and null hypotheses by a range of population groups. However, the studies overall were quite heterogeneous in terms of which services were under investigation. Therefore, more investigations into this area are needed to reinforce these findings.

## Control

Studies which examined patients intermediate and long-term complications were grouped into this category as patients' health was regarded as proxy measurements of the effectiveness of the management of type 2 diabetes.

Thirty-one out of the thirty three studies which met the inclusion criteria contained analyses of inequalities in patients' control over their condition as measured by intermediate clinical characteristics [92, 94-108, 110-122] and diabetes morbidities [99, 100, 103-109, 112, 113, 116, 121-123] dated from 1998 to 2012 (Figure 10).

Figure 10: Evidence of inequalities in control for type 2 diabetes patients



The majority of studies in this area examined inequalities by ethnic groups [94-101, 105, 107, 109, 114-116, 119, 122, 123] with the findings tending to support the negative hypothesis [94, 98, 100, 101, 107, 115, 119, 123]. The groupings of ethnicities made direct comparisons difficult; however, three New Zealand studies examined inequalities in control by similar ethnic groups: European, Maori, Pacific, Asian, Indian and Other. Kenealy *et al*, using Cox proportional hazards model, found that Maori patients had a greater chance and east Asian patients had a lesser chance of having a CV event compared to European and Other patients combined over a five year period controlling for other risk factors [116]. Compared to other studies, this had a relatively strong design and methodology with consistent results. Agban *et al* looked at various intermediate outcomes using two-tailed paired t-tests and McNemar Chi-Square and found no consistent results. Of the groups that had a statistically significant two year change in their mean HbA1c levels, European and Maori patients' health had declined whereas Pacific and Indian patients had improved. Pacific patients made greater improvement in sBP than European patients. Pacific patients also had greater improvements in total cholesterol compared to European and Asian patients. Both these findings reached statistical significance. No statistically

significant differences were found between the ethnic groups in the two year change of dBp or HDL-c [115]. A study examining inequalities in CV risk by ethnicity outlined the demographic and clinical characteristics of participants. However, no statistical tests were conducted to establish whether these characteristics were significantly different, therefore the results cannot be interpreted [101]. This was reflected in the study achieving a low score for its methodology.

Three other studies conducted in New Zealand had statistical significant evidence supporting the negative hypothesis using diverse statistical techniques. Univariate analyses found that Maori and Pacific patients had worse intermediate outcomes [94]; Cox proportional hazards model showed that Maori patients had higher chances of having dialysis or kidney transplantation [123]; and adjusted odds ratios Maori, Asian and 'Other' ethnic groups had increased odds of having HbA1c greater than 8% all in comparison to New Zealand European patients [98].

Three British studies examined intermediate outcomes and complications of South Asian patients in comparison to other ethnic groups had contrasting results: in multiple logistic regression analyses there was a statistically significant relationship between ethnicity and eye complications with South Asians more likely to have any retinopathy and maculopathy compared to white patients. There was no significant relationship between ethnicity and non-sight-threatening retinopathy [124]. The other study examined a range of outcomes and the statistically significant findings showed that South Asian patients were more likely to have retinopathy and less likely to have hypertension compared to non-South Asian patients. When examining genders separately South Asian patients had a higher HbA1c and lower sBP in both men and women. South Asian men had lower BMI than non-South Asian men [99]. This was categorised as supporting the null hypotheses, that is, there are no inequalities in diabetes control by ethnicity, with conflicting results to reflect the inconsistent findings. A national UK longitudinal study aimed to examine inequalities in incidence of myocardial infarction rates between white, South Asian and Afro-Caribbean type 2 diabetes patients and found that after adjusting for relevant covariates that Afro-Caribbean patients had a lower risk of MI than white patients whereas there was no significant difference between South Asian and white patients [122]. Overall, the statistical confidence of these results was likely to have been reduced due to the small sample of South Asian patients which also limited the possibility for further stratified analyses. Results reaching statistical significance from a London based analysis found that black Caribbean patients were more likely to have hypertension and less likely to have had a heart attack and other heart problems compared to white participants. Mostly, however, there were no inequalities between black African and white patients and, as such, this comparison supported the null hypothesis with conflicting findings [109].

The remaining seven studies examining inequalities between ethnic groups were mixed in terms of which ethnic groups were under investigation and what the results were [95-97, 100, 105, 114, 119]. However, two of these studies had consistent findings using longitudinal data, therefore had relatively stronger study designs than other studies, but examined different ethnic groupings. A study based in Germany found that immigrants had poorer HbA1c both at baseline and at 12 months follow up compared to natives [119]. The results from a Canadian study supported the null hypothesis and found no significant differences in intermediate outcomes both at baseline and 6 months between aboriginal and non-aboriginal patients following adjustments of other covariates [97].

Several of the studies also examined gender inequalities [95, 98, 102-104, 106, 111-113, 120, 121, 123]. The results were inconsistent but the majority supported the negative hypothesis [98, 104, 106, 112, 113, 120]. However, the overall quality of these studies was quite mixed and the majority had a cross-sectional study design. Statistical significant results from univariate analyses revealed that women were less likely to have retinopathy and heart disease [112] and lower predicted risk of non-fatal and fatal coronary heart disease, fatal coronary heart disease and non-fatal and fatal stroke [125]. However, one of these analyses found that women had higher absolute risk of coronary heart disease over time but there was variation in the statistical significance of other risk factors [120]; and the study found no gender inequalities in diabetes symptoms or related health conditions [126]. An univariate analyses of a cross-sectional survey in Italy found that women tended to have poorer CV risk factors than men [111]. In contrast univariate analyses of patients in Germany found that rates of CHD, intermittent claudication, stroke and nephropathy were statistically significantly higher in men than women [106]. The studies which utilised multivariate analyses to take into account potentially confounding variables, also found conflicting results. The findings which achieved statistical significance showed that women were less likely to have HbA1c greater than 8% [98], less likely to have macroangiopathy and nephropathy but not neuropathy and retinopathy [127], had worse glycaemic control, sBP and LDL-c cholesterol levels in patients with CV disease [102]. Another analysis found that non-South Asian women had higher sBP and BMI though most results from this study found no gender inequalities [99]. Another study found that overall women were significant less likely to achieve a range of treatment targets compared to men [95]. Three other studies found no gender inequalities in diabetes symptoms and related health conditions [126], renal events [123] and micro and macro vascular complications [121].

The included studies which examined inequalities in control by education, income and occupational status all used a cross-sectional design [95, 108, 114, 117, 118, 121] with only two of the five studies having a methodological score of four or five [95, 108]. Univariate analyses

found that no inequalities in control by income in an Irish study [114] and by educational level as a result of a diabetes education programme in Germany [128]. The other four studies used multivariate analysis techniques: another German study looked at a range of health outcomes by education level, occupation status and socioeconomic status. It found no evidence of inequalities apart from a few exceptions which reached statistical significance: lower education, SES and occupational status participants were more likely to fail to reach HbA1c target of < 6.5% than their respective comparison groups and only the prevalence of diabetic retinopathy showed a statistically significant inverse association to SES in one of the two data sources the authors used. No other statistically significant associations were found with other measures [129]. A study in Northern Ireland of patients at a hospital diabetes clinic examined health indicators by three different socio-economic groups. The authors found no evidence for differences in glycaemic control, BP or cholesterol by income, deprivation or education levels, except for higher levels of cholesterol tended to be associated with those living in more deprived areas [118]. In Sweden, there were no statistically significant differences in micro and macro vascular complications by education, as measured by 95% confidence intervals [121]. Interestingly a second Swedish study found evidence that patients with 10-12 years of education were more likely to achieve intermediate treatment targets compared to those with more than 12 years yet patients with lower incomes were less likely to achieve these targets compared to those in the highest income bracket [95].

In addition to those already mentioned above three further studies in the UK [92], Spain [113] and New Zealand [116] used composite measures of SEP. Statistically significant results from the Spanish analysis found that patients in more deprived areas had poorer glycaemic control and higher levels of cholesterol, and were more likely to suffer from macroangiopathy than patients from less deprived areas. No statistically significant differences were found in the odds of having neuropathy, retinopathy or nephropathy [113]. The findings from the New Zealand study showed only a weak association between participants' deprivation level and increased CV risk, while having a strong study design and large sample [116]. The UK study, following adjustment for smoking and exercise, found that both childhood and adult SEP were adversely related to fasting insulin, triglycerides, HDL-c-c and BMI, however, this data was not shown in the paper [92]. Only two longitudinal analyses looked at inequalities between rural and urban type 2 diabetic patients, both by the same investigating teams in Australia, and found that despite a number of initiatives rural patients health was inferior to their urban counterparts [110, 120].

The results from this area of investigation shows that there is more support for the negative hypothesis, with minority ethnic groups, men and those from rural areas tending to have poorer

control over their condition. These results also support the null hypothesis with no evidence of inequalities by other measures of SEP; however, these results are inconsistent.

## Discussion

### Principal findings

From 2,974 initial 'hits', 33 studies met the inclusion criteria and were included in the review. Five sets of harvest plots were produced for: diagnosis, monitoring, treatment, control and access and uptake of services. Many studies were included in more than one harvest plot as multiple interventions and/or markers of social and economic position were examined. The most common intervention was control of diabetes measured by clinical characteristics and complications.

The results displayed in the harvest plots showed that there was some evidence of inequalities associated with interventions to manage and treat Type 2 diabetes. Ethnic minorities, men, and those living in rural areas tended to have poorer control. The rest of the results examining inequalities in diabetes control by education, employment/occupation, income and by composite measures of SEP tended to support the null hypotheses. Studies that examined severity of symptoms at diagnosis found inequalities by SEP. Two studies looked at being diagnosed or not and found no differences by deprivation, education or income level but there were conflicting results regarding inequalities by occupation. There was evidence for inequalities associated with monitoring by ethnicity and only support for the null hypothesis by gender. No studies examined inequalities in monitoring by other population groups. The majority of the studies which examined inequalities associated with treatments supported the null hypothesis by ethnicity, gender, rural/urban and composite measures of SEP. Finally there was evidence for inequalities associated with diabetes services by education, ethnicity, gender and deprivation but not for between rural and urban patients.

The results here indicate that in most circumstances there was no evidence of inequalities associated with type 2 diabetes interventions. However, there were some notable exceptions, particularly associated with control, suggesting that despite the high economic development and the universal health care systems of the included countries these macro level circumstances

and the way diabetes care is planned and delivered are not enough prevent these inequalities occurring.

These results differ somewhat to the 2010 systematic review which found evidence of inequalities in treatment, control and service utilisation by ethnicity, socioeconomic inequalities in diagnosis and control, and no evidence of gender inequalities [74]. The discrepancies in the results examining socioeconomic inequalities in diagnosis and gender inequalities could be explained by the inclusion of more recent studies and the focus being on type 2 diabetes patients only.

### Secondary findings

The review also identified where evidence was lacking and if there were areas where the methodology of the available evidence base could potentially be improved. Firstly, few studies used repeat measurements. Overall only 9 out of 32 used repeat measurements data which is surprising as data regarding patients' health and care are routinely collected by healthcare providers worldwide [130]. Due to the nature of diabetes care and the progression of the disease it is difficult to attribute the cause of inequalities to particular interventions. However, more complex analyses of repeat measurements would be able to begin unpick what contribution, if any, inequalities in diagnosis, monitoring, treatment, services and control of intermediate outcomes have on the development of patients' diabetes and long-term complications by population groups.

There were a comparatively fewer studies that examined inequalities by population groups stratified by SEP in contrast to those looking at inequalities by ethnicity and gender. This is probably due to these studies relying on anonymised data collected by health care providers which in many cases do not routinely collect such information. However, many countries collect area based statistics which are often used as proxy measurements of individuals' SEP and could be used to fill this gap in the evidence base.

When focussing on the types of interventions examined from the harvest plots it was clear that there were gaps in the current body of literature. Over this twelve year period only three studies investigated whether there were any inequalities in the diagnosis of type 2 diabetes, with only one of these analyses focusing on the timeliness of that diagnosis. Being diagnosed early is critical for preventing and delaying the debilitating complications of the disease. None of

the studies which met the inclusion criteria investigated inequalities by demographic and socio-economic indicators other than ethnicity and gender in the monitoring of clinical indicators, again another key aspect of the effective management of the type 2 diabetes. There were more analyses of inequalities associated with the receipt of and access to various treatments and services as well as of a wide of range of indicators of control. There was no area which had a concentration of consistent findings. Consequently, this lack of consistent evidence makes it difficult to understand where and what action is needed, if any, to improve the equality of diabetes care.

Finally only a few of the studies in the final sample examined or controlled for healthcare provider in their analyses [110, 120, 128, 131] and only two of these used multilevel modelling techniques in order to control for any clustering effect. This is an important issue to consider as patients nested within the same providers are likely to have similar outcomes compared to those from other providers and as such could be a key cause of the inequalities noted here [132]. For instance, poorer quality of care has been shown to be associated with general practices in more deprived areas [133, 134].

### **Strengths and weaknesses of the review**

As discussed in the introduction to this literature review, this current work shared many characteristics with an existing systematic review. The major strength of the review was the methods utilised, particularly the use of harvest plots to synthesise the data. These graphical displays of the results replicate the immediacy of the 'Forest Plot', traditionally used in meta-analyses, whilst incorporating all the available evidence. The construction of the plots ensures that it is still clear where the strongest evidence lies but it also highlights where gaps or inconsistent evidence occurs. In addition to being more up to date and incorporating a graphical synthesis of the evidence, the quality assessment tool for this review was also arguably more appropriate as it is assessed the methodological rather than the reporting quality of each study [135].

Using all available evidence was also a drawback of this review as type 2 diabetes care incorporates many different interventions and can affect patients' health in many different ways. As such, comparison between studies is difficult and synthesis does not necessarily produce a more reliable set of evidence. However, this review does produce a greater insight

into the diabetes care pathway overall and may stimulate a series of more focused systematic reviews which could in turn provide greater insight into this complex area.

Whilst this review followed many of the methods of a Cochrane Systematic Review it differed in key ways in addition to the change of approach to data synthesis. The review did have a clearly defined aims and a sensitive search strategy. However, the existing data extraction and critical analyses forms were used but they were adapted in order to suit the types of studies which were included in the final sample and enabled the graphical synthesis of the results. Not all databases were searched because only primary studies examining the usual care of type 2 diabetes patients were included which made searches of some databases inappropriate. Finally, only one reviewer undertook the review which could potentially introduce bias in the selection of the studies and the synthesis and interpretation of the results [136].

### **Current implications**

Whilst the majority the findings indicated no evidence of inequalities across the five groups of interventions a notable proportion of the studies relied upon univariate analyses of cross-sectional data. There was also little investigation into inequalities associated with the timeliness of diagnosis and monitoring of type 2 diabetes by SEP, and the effect this may have on patients' health outcomes over time. To enable unpicking of the causes and to control confounding factors in the health outcomes associated with inequalities of patients continuing care more complex analyses using repeat measurements are required. The majority of the studies examining inequalities in diabetes and other treatments were unable to control for patients health status and as such were unable to determine whether prescriptions were appropriately administered or not. There were also few studies that investigated the organisational structure of the delivery of interventions designed to manage and treat type 2 diabetes. That is, the relationship between management of patients in primary care and/or secondary care services and patients' health outcomes in addition to the care they receive as an individual.

As a consequence of these findings, the following research questions were identified:

1. Are there socio-economic inequalities in intermediate outcomes and long-term complications associated with type 2 diabetes over time?
2. Are there socio-economic inequalities in interventions associated with type 2 diabetes over time?
3. Are there intervention-generated inequalities in type 2 diabetes care?
4. What impact do general practices have on inequalities by socio-economic status in diabetes care and health outcomes?

Having identified where new, substantive evidence could be added to the existing literature the next chapter discusses the methodological considerations and the outlines the methods chosen to conduct the secondary data analyses.

## Chapter 4: Methods

Following the systematic review, this thesis seeks to examine socio-economic inequalities in intermediate health outcomes and long-term health complications over time associated with the timeliness of diagnosis, the receipt of recommended care processes and treatment of blood glucose, BP and lipids. The structural arrangements of patients care and the quality of these organisations were also examined. These interventions were investigated utilising secondary datasets held by, and accessible to, the North East Public Health Observatory (NEPHO) as per part of the original proposal for this project [137].

This chapter discusses the key methodological issues and the chosen methods of the secondary data analyses. In particular, the chapter covers existing diabetes related datasets and other sources of relevant data discussing the strengths and limitations of each. The chapter moves onto discuss measurement issues relating to individuals' health outcomes, diabetes interventions and SES. This leads onto the next section which outlines some of the common methodological problems with secondary data analysis. At the end of each section the chosen methods are explained.

### Data sources

In epidemiology and public health research data is determined as primary or secondary depending on the relationship between why it was collected and why it was analysed. If the data was collected specifically for the research team then the data are considered primary. If the data was collected for a specific purpose then subsequently used for analysis for another purpose and/or research team then the data are considered secondary [138].

The main strengths of using secondary data for research are the availability and cost. The data have already been collected reducing the time and cost in sourcing such information. This has become increasingly important as research budgets are being squeezed; using secondary data can provide more cost-efficient use of resources. When using primary data, costs generally increase in relation to sample size. Using secondary data with large sample size and/or number of records can avoid this increasing cost, as it is usually incurred during the initial data collection process, as well as increasing the power of any subsequent analyses [138-140].

There are also a number of limitations. The main one being that because the data are already collected the research team cannot influence what data are collected and how. This can lead to gaps in the information required and to concerns regarding how variables are measured. Measurement issues, which are discussed further later in this chapter, as well as the political context in which they are produced, can reduce the reliability and variability of the final datasets [140].

In their overview of IGIs, White *et al* [1] highlighted how socioeconomic outcome inequalities can occur at multiple stages of the planning and delivery of interventions. An ideal dataset to investigate how and why these trends occur would therefore need to include variables which measure the provision, uptake and efficacy of an intervention as well as patient's long-term compliance and health outcomes [1]. In addition other variables which impact on patients' health, but are not directly addressed by the interventions being investigated, would be included to delineate the relative contribution each element makes on patients health. Figure 11 synthesises the broad categories which are often described as the social determinants of health [141]. Each of these categories refer to wide and varied number of issues. For example, in the Marmot Review [4] energy efficiency, tenure status, neighbourhoods, neighbours, quality, affordability, overcrowding, access to green space and insulation were all referred to in relation to housing being a social determinant of health. As such, capturing all social determinants of health in one dataset would be an extensive and potentially impossible task.

These analyses, therefore, take a pragmatic approach by initially sourcing data which can identify patients' who have type 2 diabetes, and routinely records their health and their diabetes care over multiple occasions. These are discussed in the next section 'Diabetes health datasets'. The next step was to find data which could supplement and/or validate this initial data to provide a more reliable and comprehensive dataset for the investigation of IGIs. This included 'Other health datasets' and 'Socio-economic status data' which are discussed separately.

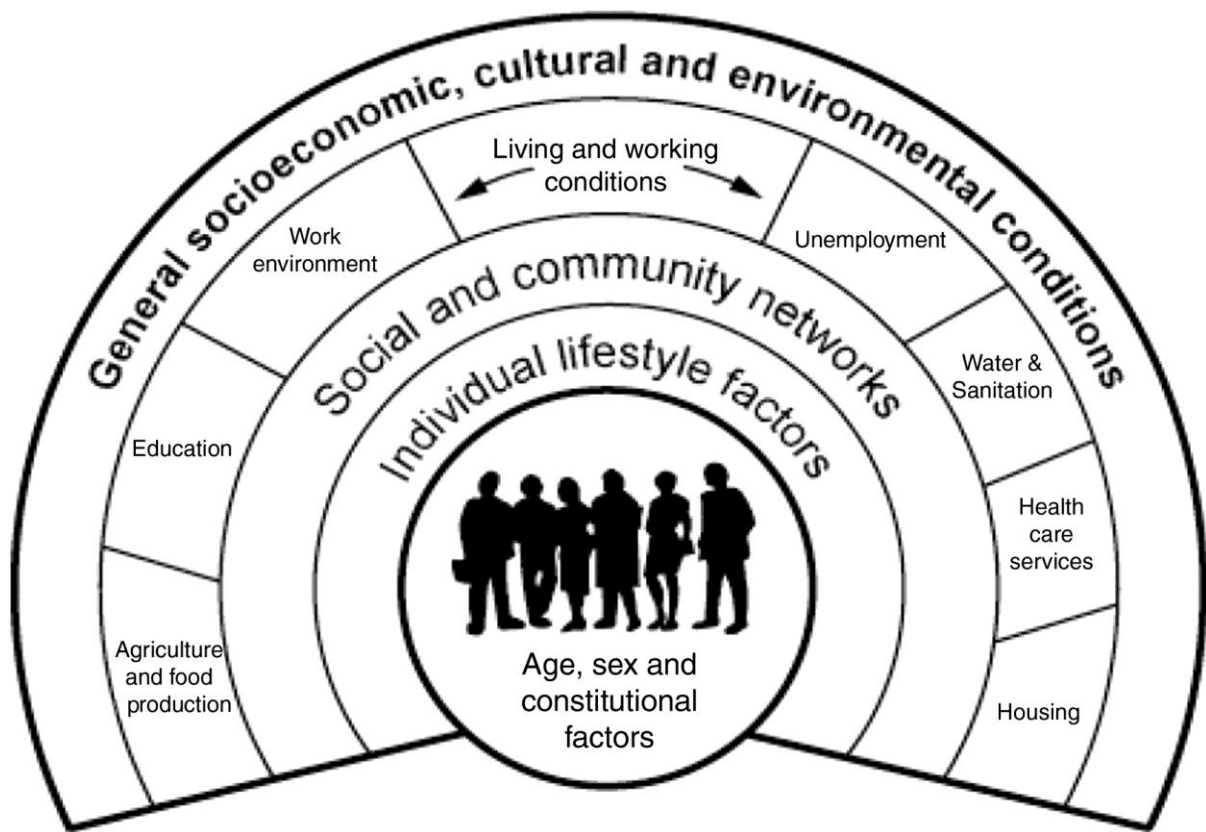


Figure 11: Social determinants of health [141]

## Diabetes health datasets

Diabetic patients, due to the nature of their condition, are normally engaged with a range of different services and treatments from primary and secondary healthcare that usually vary according to need, as well as for other reasons. As a result, data associated with diabetes care are collected and stored by several different organisations. This section discusses the major national diabetes related datasets in England and what data are available locally in the South Tees area.

### *National Diabetes Audit (NDA)*

First undertaken in 2003/04 and one of the world's largest clinical audits, the NDA is probably the most wide-ranging, longitudinal diabetes datasets currently collected. The data collected relates to the NSF for Diabetes and includes health outcomes, treatment targets and care processes from NHS organisations across England and Wales [51].

The dataset, which aims to collect information about all diagnosed diabetes patients in England and Wales, however, still has notable variation in participation rates. The first audit in England collected data during 2003/04 and had a participation rate of 20% from primary and secondary care organisations involving records of more than 253,000 patients [142]. In England in 2009/10, all 151 PCTs were represented with data on 1,929,985 patient records included. However, the participation rates within PCTs varied, for example in 24 PCTs less than 50% of the practices participated [51].

This is a valuable source of data as it creates a national picture of diabetes care using data from a range of sources. This information is normally stored by individual providers using a range of different data systems. Collating into one dataset annually enables analysis, benchmarking and feedback of clinical effectiveness across the NHS [51]. The range of organisations involved causes two main problems in terms of research. Firstly, the recording of data can vary between NHS organisations and as such extensive data cleaning would be required. Secondly, the terms of use and information governance issues are still being negotiated to allow for particular levels of analysis. This makes accessing the data and secondary data analyses currently very restrictive [143].

## *NHS Diabetic Eye Screening Programme*

The NHS Diabetic Eye Screening Programme is a nationally led programme delivered locally resulting in multiple standardised datasets across England [48]. It was set up to support health authorities to reach the NSF for Diabetes: Delivery Strategy [49] target that by 2006 a minimum of 80% of diabetic patients should be offered screening for diabetic retinopathy, rising to 100% in 2007. The achievement of this target is also supported by QOF indicator incentivising general practitioners to include their patients in a screening programme [47]. The programme aims to capture all diabetes patients aged 12 years and over as recommended by current guidelines [48, 144], in Middlesbrough PCT and Redcar & Cleveland PCT rates of over 80% have been achieved between 2006/07 and 2009/10 [51].

In terms of data quality, each screening programme is annually audited to ensure that each meets the national standards [48]. This ensures greater confidence in the dataset having consistent methods for recording the data. It is the most reliable source of retinal screening outcomes as it is the primary source of this information and therefore should be more complete than that held by organisations, such as general practices, who record the results subsequently.

The South Tees Hospitals NHS Trust ophthalmic department hosts the local screening programme and runs clinics in Middlesbrough, Redcar & Cleveland and Hambleton & Richmond locations in order to serve patients closer to home [145]. The Ophthalmic department has been using digital photography and grading images for approximately seven years. There have been a number of changes to the grading protocols since the NHS Diabetic Eye Screening Programme guidelines were introduced. The current database houses data from 2006 relating to the patient and their screening activity. Patient data includes demographic data such as NHS number, name, address, sex, date of birth, general practice and PCT codes, and non-demographic data including diabetes type, date of diagnosis and information relating to screening care [48].

The advantage of this dataset is the graded categories of patients' retinal screening outcomes. There are seven categories providing the potential for greater differentiation in patients' health. The major disadvantage of this dataset, in terms of utilising it for research, is that the data system which records the retinal screening results does not allow for multiple records to be extracted regarding specific criteria. For example, a dataset of solely type 2 diabetic patients graded outcome data cannot be generated. Also, very limited additional data are recorded limiting the possibilities for analysis when solely using this dataset [48].

Disease registers are generally regarded as databases with all known cases of a particular condition within a defined denominator population. The data from registers can be used to evaluate clinical care, services and technology and, along with the denominator population, be used in epidemiological research and needs assessment. The progressive nature of diabetes and severity of associated complications means that registers can provide effective support for health professionals to pro-actively and continuously manage disease. For example, registers can provide a list of patients due for their annual review [146].

Ideally registers should be active and continuous, in contrast to clinical audits which are collated at specific times. The UK has one of the most comprehensive cancer registration systems worldwide with every incidence of cancer being collated by one of 11 regional registries across the country [147]. Diabetes registries on the other hand have not been established in the same systematic way with the registries being hosted by many different types of organisations covering varying geographies. Since 2003 the QOF requires all general practices to be able to produce a register of all diabetes patients aged 17 and over and whether they have type 1 and type 2. However, it does not require them to produce a dataset relating to multiple aspects of patients diabetes care nor be standardised with other practices. These data are still collected as part of their usual care but without a register it is not necessarily easily accessed, analysed or recorded in a systematic way.

There are many hospital based diabetes registers in the UK including James Cook University Hospital, a hospital local to where this research was undertaken. James Cook University Hospital which is governed by South Tees Hospitals NHS Trust hosts a comprehensive diabetes register which covers the South Tees area spanning Middlesbrough LA and Redcar & Cleveland LA. Also, a specialist nurse annually collects data on patients who are managed in the South Tees area but do not attend the diabetes clinic in the hospital. As part of this process the setting of each patient visit is recorded, e.g. the Diabetes Care Centre or their general practice. This, therefore, provides an opportunity to explore the impact that receiving additional non primary care has on socioeconomic inequalities diabetes outcomes [60].

This diabetes register collects over 100 indicators relating to diabetes and has been in existence since 1987 [60]. As such it is a valuable and comprehensive longitudinal dataset ideally placed for investigating diabetes care and its long term impact. However, there are two main

drawbacks. Firstly, the register data are largely collected through paper proforma from the diabetes clinic, other hospital departments and general practices in the South Tees area. Therefore, the data quality can vary quite widely between original sources. Secondly, other health demands of patients can impact upon the quality of patients' care and outcomes which are not routinely recorded via the proforma, such as non-diabetes related co-morbid conditions, therefore limiting the scope of analysis if relying solely upon this dataset.

## Other health datasets

Whilst the datasets detailed in the previous section are specifically related to diabetes patients there are other health data which could be used. This section discusses some of the other data available in the UK that could be potentially used to measure diabetes interventions, particularly those that operate on the level of the individual patient but affect the context in which diabetes care is delivered. In addition, this section discusses datasets which could be drawn upon to capture other health conditions. Knowing about these co-morbidities is important as they can have an immense impact of a patients' quality of care and subsequent health outcomes [148].

### *Primary care data*

All NHS diabetes patients should be registered with a general practice and like certain other long term conditions, national policies aims to have the majority of these patients managed within primary care (for example: [47, 49]). Ideally, all information about all general practice registered patients' care and outcomes should be recorded within these organisations. Such data could be accessed through individual practices or from databases generated from general practice records [149].

There are a number of primary care databases covering a range of issues such as practice quality, disease incidence and prevalence, morbidity, consultation rates, health promotion and prescribing. Whilst these datasets provide opportunities to investigate specific health issues there are a number of drawbacks. Firstly, they are often reliant upon voluntary participation and as such they may not be representative of all primary care. Another source of bias is in the

quality and completeness of these databases which can vary quite widely. Also, information regarding patients' social circumstances and certain demographic data are often not recorded [149].

Data could be potentially extracted from practices individually, however, there are a number of problems with this. Firstly, it is a labour intensive process both in terms of gaining access (see section below for further discussion) and collating the data into a consistent format. Secondly, not all practices use the same data systems and the coding of data can vary widely between practices [150, 151]. Whilst there has been work at the level of PCTs to increase the consistency of recording using Read Codes to act as a central site for data extraction, the current climate of NHS reforms has had a major impact on the ability of staff to accommodate such a request [152].

## *Quality and Outcomes Framework*

Quality and Outcomes Framework (QOF) was introduced by the new General Medical Services contract and was one of the consequences of the government's aim to expand chronic disease management into primary care [1]. QOF is a voluntary annual reward and incentive programme established in general practices across the UK during 2004 with the aim to reward the provision of good quality care and improve standards [2, 3].

QOF was not designed to be a comprehensive data source about the quality of care in general practices, however, it does provide the opportunity to use the data in this way. It has the advantage over the datasets already discussed in that it is freely available online via The Health and Social Care Information Centre. The dataset has national coverage across England and Wales and uses a range of indicators of quality of care related to diabetes. The main drawback though is that it only contains practice-level data, therefore, it does not provide the opportunity to control for the individual patient clinical or socio-demographic characteristics. However, the dataset does provide good general information about general practices in terms of practice list size, patient experience and additional services. However, some of these fields published refer to the number of points achieved for a particular quality indicator in which there is often not a great deal of difference in achievement. Therefore, some of the fields may vary enough to warrant further analysis [47].

## *Hospital episodes statistics*

Established in 1987, Hospital Episode Statistics (HES) is a database which contains information about each episode of care provided by NHS hospitals and for NHS hospital patients treated by non-NHS providers. Data collected each financial year since 1989-90 are available but the procedures and structures of data collection have changed over time [153].

Whilst the quality of the data collated in emergency and outpatient departments is poor, HES could be a source of patients' diabetes complications and other health needs which result in inpatient hospital care. Obviously not all co-morbidities which impact on patients' health and diabetes related care result in being admitted to hospital yet using HES for this purpose has the advantage that it is a centrally accessed dataset with national and longitudinal coverage.

In 2011, the network of Public Health Observatories published The National General Practices Profiles accessed freely online via the network's website. They are designed to assist general practitioners and commissioning groups in providing healthcare services to meet the needs of their local population. The profiles bring together 2009/10 results from population data from the Attribution Dataset (ADS), GP Patient Survey data, QOF data and admission rates from NHS Comparators. The results are then displayed in various charts to enable comparison of GP level outcomes with PCT and national rates. The metadata which are used to generate each indicator is also available to download [154].

This is a valuable resource that can enable comprehensive and systematic comparisons of general practice quality and activity. Unfortunately profiles are only available for years 2010 and 2011 due to the type of data available which makes up the profiles, therefore, longitudinal analyses using this data is limited.

### *Survey data*

Survey data could provide greater insight into routine health data and supplement it as demonstrated by many of the studies in the literature review. However, they are usually designed around particular issues, cross-sectional in terms of the data collection, not readily available or widely publicised. The value of survey data could be increased by linking survey data with routine health data but this would pose particular ethical and information governance issues, which could potentially hinder time efficient research in this area.

### **Socio-economic status data**

When utilising secondary data for analysis the researcher is generally restricted in the choice of indicators of socio-economic status by what was initially recorded. Choice is restricted further if relying solely on routine health data. Demographic data, such as gender, age and ethnicity, are routinely collected by a range of services, however, measurements of SES, such as income,

education and occupation, are less common. None of the data sources described so far routinely record this type of information. A widely used solution to this is to link health data to census and administrative data measured at a particular geographical level via the patient addresses. This data can then be used as proxy indicators of individuals' social position [155, 156]. In this section the two primary sources in the UK are described. The specific indicators are discussed later.

### *Office for National Statistics Neighbourhood statistics*

In the UK the Office for National Statistics (ONS) collect, analyse and disseminate a vast amount of data at differing levels of geography including data about the social and physical environment, education, services and crime. The timeliness and range of indicators for each topic is dependent upon the source of the data which include a range of government departments. For example, information from the Department for Work and Pensions is made public on an annual basis. However, information which is gathered through the census can be only produced on a ten yearly basis. The major advantage of utilising data from this source is that is freely available online and, depending upon which indicators are used, measured at small geographic levels [157, 158].

### *Marketing data*

Geo-classification systems produced by marketing companies, such as ACORN and SuperProfiles, are an alternative for measuring individual SES at the area level. These systems are created to help their clients with advertising, marketing and targeting products to particular consumer groups. These systems are predominantly new methods of categorised Census data, however, SuperProfiles does include market research data and credit information too. Due to the purpose of these systems they generally incur a fee [157].

## Summary

There was an ideal opportunity in South Tees to construct a robust diabetes related dataset by linking the data from the South Tees Hospitals NHS Trust Diabetes register and NHS Diabetic Eye Screening Programme to compare changes in the rate of intermediate health outcomes and complications by patients' socio-economic status. The diabetes register was chosen as the core dataset as it contains a vast range of repeat measurements for a large population sample. Data dated from 1999 to 2007 inclusively could be relatively easily accessed following the appropriate permissions and contains consistent variables and identifiers to enable linkage with other datasets. It has also the added advantage of having staff with an in depth knowledge of diabetes, dataset and the local area which enables a greater insight into the data and understanding the analyses. This data were limited to the years 1999 to 2007 inclusively as it was the most up to date information at the time of extraction. Through this core dataset, information about patients' health, lifestyle and anthropometric status as well as about their care in terms of their general practice, monitoring and receipt of secondary services could be extracted. In addition, publically available data from ONS, QOF and Practice profiles could be linked to the core dataset to provide proxy measurement of their SES and further information about their general practices. This latter data was chosen due to its public availability and coverage.

## Study population

Following the identification of the most appropriate data, the study population was identified and defined. This was done through the core dataset, the South Tees Hospitals NHS Trust Diabetes Register. These inclusion criteria were designed to capture type 2 diabetes patients using established epidemiological methods and avoid recording error of type of diabetes in the register dataset. This identified study population sample should receive the same access and quality of care from the community services situated in the two PCTs. The Diabetes Care Centre and the local branch of the NHS Diabetic Eye Screening Programme both serve the South Tees area as a whole. Children and adolescents with diabetes were excluded as they are managed differently and therefore constitute a different care pathway which is not the focus of this research [52].

Patients' data were included in the final analyses if they met all the following criteria:

- Type 2 diabetes patients. Patients from the diabetes register were identified as having type 2 diabetes through the following established epidemiological definition [55, 159, 160]: if they were diagnosed with diabetes over the age of 35 or recorded as not using insulin.
- Living and registered with a general practice in the South Tees area. The South Tees area is defined as Middlesbrough LA and Redcar & Cleveland LA. Patients were considered living in South Tees if the LSOA of residence falls into this area as determined by ONS data [158] and GeoConvert [161]. Middlesbrough PCT and Redcar & Cleveland PCT are coterminous with the LA areas and practices which fall under their responsibility according to where patients live were defined as being in the South Tees area.
- Aged 16 years old or older

## Data extraction and construction of final dataset

As discussed previously, the South Tees Hospital NHS Trust Diabetes Register provides the core dataset for this project and to increase data coverage additional datasets were linked to this dataset. For this to be done effectively, both or more datasets had to share common identifiers to which the data refers.

In the UK, every NHS patients has a unique 10 digit number which is recorded during every visit with a NHS provider. They were introduced with the aim to improve safety and efficiency of healthcare [162] and have the benefit of enabling the straight forward linkage of patient level NHS data. Data linkage between patient level, non-NHS datasets would require multiple indicators of identifiable data and carries a higher risk that the linkage is inaccurate [163]. Using identifiable data for the purpose of data linkage, however, requires either patient consent or if this is not possible and/or a practical option then a section 251 approval has to be sought from the National Information Governance Board [164].

To avoid the timely process of acquiring patient consent and/or adhering to conditions associated with a section 251 approval the data linkage process was done by staff with prior access to the datasets. The following data were used and then removed from the final data extract by the data manager, Elaine Hall, before being pseudonymised for final external use:

NHS numbers, postcodes and dates of births. Details about how these fields were used are discussed throughout this chapter. A separate table containing the study patients' NHS number and a new unique study number was generated and retained by the data manager to allow direct linkage between the register and retinal screening programme datasets.

The final data extraction of the register data was sent by the data manager to NEPHO care of Professor John Wilkinson via their NHS secure email accounts. The data was sent in ten tables, one for each year of recorded data and one containing the demographic data of the patients which do not change: age at the end of 2007, year of diagnosis, sex and ethnic origin. All tables featured the study numbers unique to each patient to enable the linkage between the tables. These were then formatted in a long table format with each row containing one year of data for one patient. Prior to any data cleaning the initial extract contained 69,894 records for 13,687 patients.

Graded retinal screening outcome data for all patients from 2006 and 2007 was requested to increase the completeness and accuracy of the recording of this field. Where there were any discrepancies in the values between the two sources, the screening programme data was favoured. This data were sent from the South of Tees NHS Diabetic Eye Screening Programme to the diabetes register data manager, both situated within the South Tees Hospital NHS Trust. The data manager at the register then used the table containing the two sets of identifiers to assign the new unique study numbers to screening data. The NHS numbers and all the records of patients who were not identified through the diabetes register were then removed. This data was sent to NEPHO via the same method described above.

Information about patients' general practices was sourced from QOF and Public Health Observatories (PHO) of England General Practice Profiles. Quality and Outcomes Framework data were used to provide indicators of quality of care in general practices. These were generally from the diabetes domain of the framework and were extracted for all general practices in Middlesbrough PCT and Redcar & Cleveland PCT from the period 2004/05, the time period QOF was introduced, until 2007/08 from the Information Centre website [47]. The deprivation scores from the Practice Profiles were used to provide a proxy measure of the deprivation profile of South Tees general practice populations [154]. These data were linked to the register data via the general practice code for each patient for that year.

The national rank position data for each lower super output area (LSOA) using the 2004 Index of Multiple Deprivation (IMD) scores in England were downloaded from the Office for National Statistics (ONS) Neighbourhood Statistics website [158]. These were used as proxy measures for individual's SES. Whilst IMD data have been constructed during other time periods the

methodologies have changed over time [158], therefore, only one set of IMD data was used to keep a reliable indicator of SES over the study period. All LSOA located in Middlesbrough LA and Redcar & Cleveland LA were identified using the same downloaded data from the ONS and a table of these areas and the postcodes which fall into these areas was generated using GeoConvert [161]. Where postcodes covered more than one area the LSOA, the LSOA which the greatest proportion of the postcode area covered was used. This was identified through the percentage matched which is generated alongside the corresponding area by GeoConvert. This table was then emailed to the data manager at the diabetes register who linked data via patients' postcode for each year. The postcodes were then removed. This data linkage was done by the diabetes register data manager to protect the sensitive postcode data before releasing the data.

## Data access

Data access here refers to the ethical and information governance issues that needed to be addressed when undertaking university based research and more importantly when seeking to use patient level health data. Durham University postgraduate students are required to conform to their academic school's policies, in this case, the School of Medicine and Health, and the University's policies on ethics in research [165]. In addition, if using NHS data, undertaking research with NHS staff and/or researching within NHS environments ethical and information governance approval from the National Research Ethics Service [166] and the appropriate Research and Development are required. In turn, if patient identifiable data without prior consent is needed, an application to the National Information Governance Board for section 251 approvals is also required [164]. Gaining the appropriate approvals can potentially be a lengthy process, therefore, the efficiency of extracting the subsequent data is also an important consideration especially when there is a restricted time frame to undertaken the research.

Once the datasets were chosen, approval to access the data and undertake the research was sought and granted by the following organisations (Appendix B):

- School of Medicine and Health Ethics Committee, Durham University  
Ref: ESC2/2010/12
- County Durham & Tees Valley Research Ethics Committee, National Research Ethics Service  
Ref: 10/H0908/63

- Research & Development / Academic Division, South Tees Hospitals NHS Foundation Trust

## Dataset and variable construction

This section discusses the key methodological issues related to each variable. This is followed by descriptions of where the variable was sourced from, how it was measured and/or derived. This included what data cleaning procedures were administered to ensure the most reliable and valid data. A summary table of the source and formatting of each variable are provided in Appendix C. In addition, the level of missing data per variable over the study period are detailed in Appendix D.

## Type 2 diabetes interventions

### *Diagnosis*

There is evidence that early diagnosis of type 2 diabetes and the initiation of secondary prevention interventions can reduce and/or delay the presentation of diabetes related complications [167]. The studies from the literature review looked at two related issues regarding diagnosis: firstly timeliness of diagnosis, that is patients' health statuses at the point of diagnosis [168], and being diagnosed or not [92, 93]. The two studies which examined timeliness of diagnosis measured this as the level of retinopathy at diagnosis [168]. The former of these two studies utilised routine health data assessing patients' outcomes at their first retinal screening visit. However, this may occur months or years after patients' initial diagnosis but other health datasets have potential for examining other clinical indicators nearer to the time of intervention.

The main issue with using routine health data only means that the circumstances which led up to being diagnosed are unlikely to be routinely and systematically recorded. Survey data could potentially be a source of this data. However, no survey available through the UK Data Archive [169] and Data.Gov.UK [170] had looked at this issue. In addition, there are potential

information governance issues to address if patient identifiable information is required to link the data with other longitudinal health data in order to investigate the long-term consequence of a delayed diagnosis.

This thesis is concerned with patients from the point of diagnosis onwards. As such, the study population covers those who have previously been diagnosed. What could be investigated was the severity of patients' symptoms near the time of diagnosis, or what could be regarded as the timeliness of diagnosis. Here, indicators measured during the year the patient was diagnosed and analysed using only for patients diagnosed during the study period recorded in all their records.

Ideally, an indicator not prone to changes in an individual's temporal circumstances would have been chosen. Using the available data for this thesis, this would have been retinopathy. However the coverage of this indicator was poor and more so in the cohort who was diagnosed during the study. Consequently, due to its coverage and its importance as an indicator of diabetic control, the severity of patients' HbA1c at the time of diagnosis was used as a proxy indicator. This was based on the assumption that a lower HbA1c at diagnosis indicates that the diabetes was diagnosed earlier.

### *Monitoring*

Receiving recommended care processes could be regarded as an aspect of care quality. Few studies that met the inclusion criteria of the literature review examined inequalities in monitoring. This was surprising as it is an important part of diabetes care, forming part of many prominent guidelines for the management of type 2 diabetes [52, 69, 171]. In the UK, monitoring rates should be routinely captured in health datasets due to guidelines such as those by the NICE [46] and QOF [47]. In terms of monitoring, the NICE guidelines for type 2 diabetes state that patients should have nine anthropometric and clinical outcomes measured annually as part of their on-going care. These are recommended in order to achieve the best standard of care for the patient [69]. The NDA regularly reports the percentage of patients in a given area who have received all care processes [61].

The reason for the lack of investigation in this area may be due to one of the key weaknesses of using monitoring rates as a proxy measure of quality of care. That is, because using routine health data alone does not effectively capturing sufficient information to analyse the impact on

a patient's health. That is because in routine health data, the same information tends to capture the recording of the outcomes and the actual outcome. For example, if a patient has not had their eyes screened it is not known what level of retinopathy they have and therefore what impact non receipt of this care process has had. There are two solutions to this, firstly examining the overall number of care processes a patient receives during their routine visit and what impact this has on the health outcomes which are recorded. Whilst this is not ideal, by using a total 'score' this could act a proxy measurement for this particular aspect of patient care as it captures the level of compliance with national guidelines. A second solution is using the recorded data to impute, estimate or simulate the missing data. Imputation and simulation methods, which are discussed later in this chapter, have several advantages: firstly it avoids the bias which could be introduced if analysis is conducted upon complete case analysis alone [172]. For instance, missing data may be more prevalent in more deprived patients. Secondly, it allows the examination the lack of recording of care processes on the outcome that process monitors.

Another weakness is that the reliance upon routine health data and these approaches cannot account for those patients who do not engage with services at all as they are not represented in any of the recorded data. Also, it cannot distinguish between missing data as a result of not receiving that care process and data missing for other reasons. Data which may be missing may simply be due to the practitioner not recording the data even though the process was performed. It could also be the result of ineffective data sharing, especially when care is dispersed across many different services. Without effective data sharing, patients could be regarded as receiving sub-standard care when it is not actually the case.

In addition, there are other aspects of quality of care, such as those in the 2011 NICE quality standards programme for diabetes in adults [173] which are not routinely measured. Therefore, using monitoring rates as a proxy indicator of quality of care has the advantage of being routinely measured. In the statistical analyses in this thesis, quality of care refers to the number of NICE recommended care processes patients have received. Each year, patients should have the following data measured and recorded: BMI, HbA1c, BP, albumin, creatinine, cholesterol, smoking status and examination of their eyes and feet. For this study data on only eight processes of processes were extracted. The diabetes register team highlighted that the data relating to foot examinations are poorly recorded and recommended not to use this data [174]. Prior to the cleaning of these variables eight new variables were constructed to indicate whether the patients had received each care process described above; '1' indicated 'Yes', '0' for 'No'. The following variables were used to construct these new indicators: BMI, HbA1c, BP (either systolic or diastolic had to be recorded, not necessarily both), microalbuminuria,

creatinine, cholesterol (total, HDL-c, LDL-c or triglycerides), retinopathy and smoking status. A total score was then constructed which ranged from 0 to 8. As the majority of patients either received 4 to 8 of the care processes this variable was recoded into a categorical variable: 1 = less than 7, 2 = 7 and 3 = 8 care processes received. These are described in the results section as poor, medium or high quality of care.

### *Treatments*

Only a limited proportion of type 2 diabetes patients manage to control their glucose levels through lifestyle changes for more than a few months, therefore, the use of oral glucose-lowering drugs and/or insulin are likely to be prescribed. Type 2 diabetes patients also have a high risk of developing CVD, eye damage, kidney disease and microvascular damage which can be reduced through improved BP, cholesterol and blood lipid profile. Patients, therefore, with poor intermediate outcomes are likely to be prescribed therapies to improve their control [46].

Investigating inequalities associated with treatments is complex as the type, dosage, the starting of treatments and its duration are dependent upon multiple factors such as health status and patients willingness to engage with certain treatments. Secondary data analyses of these issues are unlikely to be able to capture all factors involved in the treatment of diabetes and its associated complications but it can begin to unpick whether there are systematic differences in treatments between different population groups.

The health status of patients is something which is likely to be routinely recorded in various health datasets. Of the seven studies from the literature review which primarily focused on inequalities in treatment [92, 100-102, 106, 110, 111], only four of these studies took this into account [100-102, 106]. Without this information it is not clear whether there are inequalities in treatment use or if these inequalities are acting a proxy indicator for inequalities in control as a result of other factors.

The diabetes register proforma had a section relating to which drug treatments patients were prescribed. A recording of '1' indicated if a patient was receiving that particular type of diabetes treatment and '0' if not. A major weakness of using these data is that they do not capture all the pertinent factors mentioned above relating to a patients treatment. However, a key strength is that they have the benefit of being extracted along with patients health status data. As such, it

can be established whether there are systematic differences in the treatments patients were being prescribed.

The following diabetes treatments were extracted: 'Diet Alone', Insulin, Sulphonylureas, Metformin, Acarbose, Glitazone. If a patient was recorded as having a diabetes treatment other than diet alone and diet alone was recorded as 1 this was recoded as 0. This was based on the assumption that the recording of a '1' for a particular diabetes treatment is more accurate. This data were recoded into one categorical variable due to the presence of collinearity between some of the binary variables and low use of acarbose and glitazone treatments. The categories were broadly based upon glucose lowering therapy algorithm in the NICE Type 2 diabetes guideline. The new variable was categorised as follows: 1 = Diet alone, 2 = Metformin or sulphonylureas only, 3 = Diabetes treatment combination excluding insulin, 4 = Insulin only and 5 = Diabetes treatment combination including insulin.

Blood pressure treatments – diuretics, beta blockers, alpha blockers, ACE inhibitors and calcium antagonists, lipid therapies and aspirin were also extracted. BP treatments were recoded into one categorical variable due to the low prescription rates of some of the treatments. The new variable was categorised broadly based upon BP treatments algorithm in the NICE Type 2 diabetes guideline [46]. The categories were as follows: 1 = No BP treatment, 2 = ACEIs only, 3 = ACEIs plus any combination of other BP treatments and 4 = other treatment(s). These categories is a simplification of the scheme for the management of BP for people with Type 2 diabetes found in the NICE type 2 diabetes guidelines. Most patients should move from category 1 through to 3 if their BP deteriorates with category 4 possible depending upon other factors such as ethnicity, pregnancy and having microalbuminuria [46]. Details of lipid therapy were recorded on the proforma as follows: 0 = None, 1 = Statin, 2 = Other, 3 = Multiple. However, the prevalence of the prescriptions of these therapies is relatively low for 'Other' and 'Multiple therapies', therefore, this was recoded into a binary variable with '1' indicating the patient was receiving any lipid therapy(s) and '0' for none.

### *Services*

Many of the studies in the literature review examined inequalities associated with what was broadly described as services. The use of 'services' here refers to types of services, as opposed to the process of care being undertaken, including places of care and services delivered dependent upon the qualification of staff involved. Due to the range of interventions that these

services encompass, and the various characteristics of each these services, there are potentially multiple ways inequalities could occur.

There are several issues when investigating inequalities associated with diabetes services. Firstly, there are services that all diabetes patients should be offered as recommended by national guidelines therefore ensuring that patients are offered and use these services. Receipt of these services could be regarded as an indicator of quality of providers who act as the gatekeeper to these services. Likewise quality, as measured by the rate providers adhere to recommended monitoring rates over the entire service population, which can vary between similar providers, therefore inequalities could occur due to being registered at one rather than another. Whilst these may not be considered indicators of quality there are other features of services like general practices which may influence equality of care such as the size of the practice, the level of staff and their skills, and the internal procedures. These issues were not considered in the final sample of the studies in literature review. Also, there are many sets of guidelines outlining what care diabetes patients should receive but there is little stating which professionals should deliver this care. The new 'Diabetes in adults quality standards' refer to an appropriately trained healthcare professional rather than a GP or dietician, for example [173]. It was not clear from the current literature if this has an impact on health inequalities or not.

A weakness of investigating the receipt of particular services using routine health data is that it is dependent upon information being systematically collected. Providers adhering to monitoring rates will be collected as these are part of their care, however, who delivered the care and the details about those services are less likely to be recorded. In addition, the problem of non-engagement with services cannot be evaluated using this data. Information about providers themselves is available from sources, such NHS websites and various NHS surveys (for example: [175, 176]), however, the historical nature of this data varies making analysis over time difficult. As such none of the available datasets enabled the investigation of these issues.

The strength of using the diabetes register as the core dataset for the statistical analyses it was possible to investigate several aspects of patients' services. Firstly, where the patient visit occurred was recorded each time the proforma was completed. This data was extracted and was recoded into a binary variable to indicate whether patients were being managed in primary care only or received additional specialist care within a particular year. Patients were coded as '1' for 'shared care' if the 'Source of Form', a field included in the original data extraction' was recorded as 'Diabetes Care Centre' or 'Community Intermediate Clinic', and '0' if this was recorded as 'General Practice' only. Secondly, a variable was constructed to indicate whether patients were being managed by Middlesbrough PCT or Redcar & Cleveland PCT within a given year. Patients

were coded as '1' for 'Middlesbrough PCT' if the practice they were registered with that year was in Middlesbrough. Patients were coded as '0' for 'Redcar & Cleveland PCT' if the practice they were registered with that year was in Redcar & Cleveland.

In addition, increased practice size, diabetes prevalence and more socio-economically deprived patients have been shown to be associated with poorer quality of care [177] therefore the following variables were collected to be included in the analyses. General practice deprivation scores were constructed using the Index of Multiple Deprivation (IMD) 2007 applied proportionally to the Attribution Dataset practice populations, 2010 for the production of the National Practice profiles by the network of Public Health Observatories [154]. Scores for all English practices were downloaded and divided into quartiles. The number of patients on practice and diabetes register and the practice diabetes prevalence were extracted from the QOF datasets on the Information Centre website. The diabetes register size is the number of patients with any diagnosed diabetes aged 17 and over registered at that practice. The prevalence is the percentage of the diabetes register over the practice list size for patients aged 17 and over.

Diabetes is one of the twenty clinical domains featured in QOF. This domain has featured 25 different indicators, some of which have varied between years [47]. The indicators which were consistently measured between 2004 and 2007 inclusively were included in the analyses providing a broad picture of diabetes care at a practice level. For each practice the percentage of patients for which each performance indicator was achieved was calculated using the size of practices' diabetes registers as the denominators. This is the method which is used in the Network of Public Health Observatories practice profiles as it retains all patients in the denominator, including those which have been excluded by the practice for calculation for payment on performance [154].

The following indicators are used and recalculated using the above method:

- Percentage of patients with diabetes whose notes record BMI in the previous 15 months
- Percentage of patients with diabetes in whom the last HbA1c is 10 or less (or equivalent test/reference range depending on local laboratory) in the previous 15 months
- Percentage of patients with diabetes with a record of the presence or absence of peripheral pulses in the previous 15 months
- Percentage of patients with diabetes with a record of neuropathy testing in the previous 15 months
- Percentage of patients with diabetes who have a record of the BP in the previous 15 months
- Percentage of patients with diabetes in whom the last BP is 145/85 or less

- Percentage of patients with diabetes who have a record of micro-albuminuria testing in the previous 15 months (exception reporting for patients with proteinuria)
- Percentage of patients with diabetes with a diagnosis of proteinuria or micro-albuminuria who are treated with ACEIs (or A2 antagonists)
- Percentage of patients with diabetes who have a record of total cholesterol in the previous 15 months
- Percentage of patients with diabetes whose last measured total cholesterol within the previous 15 months is 5mmol/l or less
- Percentage of patients with diabetes who have had influenza immunisation in the preceding 1 September to 31 March

With the exception of the practice list size which counts all registered patients, these indicators refer to patients with both type 1 and type 2 diabetes. QOF indicators are measured over a 15 month period. Due to the discrepancy in time periods between QOF and the diabetes register data the QOF data recorded for 2004/05 were regarded as a measure of 2004 performance, 2005/06 for 2005 and so on. These data were linked to patient-year records via the National Practice Code of the practice the patient was registered at for that year.

Following initial analyses Practice Deprivation and Practice list size were recoded into categorical variables in order to establish if there were any non-linear trends. All of the practice deprivation scores from the Practice Profiles were divided into quartiles. All the practices in the final dataset were then assigned to a quartile. No practice in South Tees fell into the least the least deprived quartile, as such, the three remaining categories are referred to as '1' high, '2' mid and '3' low practice deprivation. Practice list size was recoded '1' if there were less than 7,000, '2' if there between 7,000 and 9,999 inclusively, and '3' if there were 10,000 or more patients registered with the practice.

### Socio-economic status

Inequalities in health can occur across a range of population groups categorised by their socio-demographic and socio-economic status. For example, location, race, ethnicity, culture, occupation, gender, religion, age, education or income[1]. To investigate the extent of socio-economic inequalities associated with type 2 diabetes interventions stratified measurements of patients SES are required. However, SES can be conceptualised and measured in multiple different ways. The existence and extent of health inequalities are influenced by the choice of

indicators. For example, an analysis of the British Household Survey found that among initially healthy economically active respondents the strongest predictor of self-rated health was the National Statistics Socio-Economic Classification (NS-SEC), but for the initially healthy economically inactive was the respondents' Household Cambridge Scale Score. The NS-SEC is based upon respondents' most recent occupation. The Household Cambridge Scale Score is also based upon occupation but takes it account of lifestyles and resources too. Other measures analysed were personal income and household income [178].

As reflected in the multiple ways SES can be measured, it is widely recognised it is a multi-dimensional concept and there have been a number of indices which aim to capture this in a single measure or indices of deprivation. Many countries have constructed indices of deprivation utilising routinely collected area based statistics. In the UK the most well-known include the Townsend Index, Jarman, Carstairs and Morris Scottish Deprivation Score and Indexes of Multiple Deprivation [157].

The Townsend Index is measured using four variables taken from the Censuses incorporating both material and social deprivation. These are as follows: lack of access to good housing, lack of material possessions, lack of access to private transport and unemployment. Carstairs and Morris Scottish Deprivation Score, also known as Scotdep, is similar to the Townsend Index but replaces the housing variable with low social class. This was to reflect that within Scotland there is a higher proportion of social housing which reduced the sensitivity of the Townsend Index. The Indexes of Multiple Deprivation (IMD) also incorporates both measures of social and material deprivation but uses a greater range of indicators grouped into seven domains: income, employment, health and disability, education, skills and training, barriers to housing and services, living environment and crime. In addition to the greater range of variables used to produce the index, it is also assigned to smaller geographical areas than the previous two indexes [157]. These smaller geographical areas are lower super output areas (LSOA) which represent a minimum of 1,000 residents and 400 households [74].

The main problem with using any of these measures as a proxy for an individual's socio-economic status is that they inevitably under- or overestimate the personal circumstances of individuals in a given area as the deprivation score is an average of that areas population circumstances [179]. In addition, whilst IMD incorporates a greater range of indicators than the other indices mentioned and its methodology has been criticised for not being explicitly clear which could lead to a misinterpretation of the deprivation patterns [179]. Having said that it has been widely used and therefore it is invaluable in being able to compare results from other studies and could make meta-analyses more effective.

Despite its limitations, the 2004 IMD was chosen as it is the most comprehensive indicator of patients' socio-economic circumstances and measured at a small geographic area. The 2004 Index was chosen, rather than another year, as this was available during the study period and therefore most likely to reflect patients' circumstances that period. Using more than one index was not appropriate as the way the indices were measured changed between time periods therefore would not be consistent [180].

A number of steps had to be undertaken to assign each patient into a SES group. Firstly, all the English LSOAs were divided into five equal groups, quintiles, using the ranked position based on the IMD deprivation score. Quintile 1 indicated the most deprived fifth of all English LSOAs. The rank position and corresponding quintile were linked to the diabetes extract using patients' LSOA per year. Groups were used in the analysis instead of using the rank or score so that trends could be more easily identified [181]. However, it was clear from initial analyses that fewer groups were required to gain more robust results. Therefore, quintiles one and two remained the same, that is patients who live in the two most deprived fifths of areas in England. The remaining three quintiles were recoded into one category to represent the least deprived patients. This method was favoured over assigning patients into nationally created tertiles. This was explored, but the majority of patients in the South Tees area live in the most deprived third of English LSOAs therefore the above method was chosen as it produced three fairly evenly distributed groups whilst being related to a national scale.

### **Health status, socio-demographic, anthropometric and lifestyle data**

The following data discussed in this section measure a wide range of aspects of patients' health which were used as either outcome variables and/or controlled for in the statistical analyses. This was to establish what impact the difference in diabetes care patients receive has on their health and/or whether patients with the same health status receive different care. Indicators of patients' socio-demographic, anthropometric and lifestyle which can have an impact on patients' health outcomes were also extracted. These were chosen based on what was available from the chosen datasets and their relevance to the statistical analyses. As such, all data were extracted from the diabetes register as this dataset contained a large sample of patients who had multiple measurements taken over time.

Patients' sex and ethnicity as inequalities in health outcomes by these population groups have been well documented (for example: [182-186]) and specifically for type 2 diabetes as shown in the literature review for this study in chapter three. In the proforma, patients' ethnicity was coded as either: 'White', 'South Asian', 'Afro-Caribbean' or 'Other ethnicity'. To aid interpretation in the statistical analyses the ethnicity variable was recoded into three categories. White and South Asian categories remained the same and Afro-Caribbean ethnicity was recoded to be included in the 'Other ethnicity' due to the small numbers of both these categories. The 93 records, from 56 patients, with no ethnicity recorded were also removed. Seven records, from one patient, did not have a recording of sex. Therefore these were also removed.

Patients' ages were extracted as insulin deficiency increases over time. It is recognised that diabetes care should be delivered taking into account the age of the patients [46]. Therefore, this could be an important explanatory variable in the analyses. The age in whole years of all patients at the end of 2007 were extracted by the database manager using patients' dates of birth. The dates of birth were then removed before releasing the data in order to protect this patient identifiable data. This variable was 100% complete. Patients' age per year was calculated using this data plus the year of visit. Again, to enable identification of non-linear relationships this variable was recoded into a categorical variable where 1 = patients aged under 60 years old, 2 = 60 and over and less than 75 years old, and 3 = aged 75 and over.

Due to the progressive nature of the condition the duration since diagnosis is an important determinant of health outcomes. The years of patients' diagnosis, which are routinely recorded on the diabetes register proforma, was extracted. The duration of patients diabetes in whole years was calculated using this and the year of visit. Following cross tabulation, those who had the year of diagnosis following the year of visit had these values removed: 112 values from 85 patients. This variable was recoded into a categorical variables where 1 = 0-3, 2 = 4-9 years and 3 = 10 or more years since diagnosis.

Body mass index (BMI) and smoking status were also extracted as these both measure aspects of patients' lifestyle which can impact on many of the health outcome variables used for this analysis [46]. Body mass index ( $\text{kg}/\text{m}^2$ ) is calculated by the data input team from weight and height which are measured by the practitioner. Both patients' weight and BMI were extracted; from these values patients' heights were calculated. As recommended by the diabetes register team recordings of weight outside 0-220 kg and height below or equal to 0.8 and greater than or

equal to 2.1 metres were considered inaccurate and were removed from the dataset. When inspecting the BMI data by patient over time, both with the original BMI values and 'cleaned' weight and calculated height values, there were many instances of large variation. For instance, of those patients with more than one recording of BMI, approximately 20% of these values for BMI differed by over 5 kg/m<sup>2</sup>. An increase of over 5 kg/m<sup>2</sup> could mean a patient is categorised as underweight then overweight. However, due to the nature of diabetes, it is possible that patients experience sudden weight gain and/or loss over a short period of time. It is, therefore, not possible to judge with confidence whether these large variations in patients' BMI was an accurate reflection of their body mass due to changes in health and/or result of treatments or result of recording and/or measurement error. To minimise the risk of measurement error, the median height for patients with three or more values which were able to be calculated were used for a recalculation of patients' BMI at all time points. Median values were chosen as the mean is skewed more by extreme values. These median height values were also used to calculate the BMI for patients who did not have their height recorded for that year but had their weight recorded. BMI values greater than 100 were subsequently removed following this recalculation. Where less than three calculations of height per patient occurred the original BMI calculation was used.

Following initial modelling, the BMI variable was further formatted to establish whether there were non-linear trends and was recoded into a categorical variable where 1 = 'Under or normal weight' where BMI is less than 25 kg/m<sup>2</sup>, 2 = 'Overweight' where BMI is equal to or greater than 25 kg/m<sup>2</sup> and less than 30 kg/m<sup>2</sup>, and 3 = 'Obese' where BMI is equal to or greater than 30 kg/m<sup>2</sup>.

On the diabetes register proforma patients' smoking status is recorded as either yes, no or ex-smoker, noted as 0, 1, or 2 respectively. This information is based on self-report. From a visual inspection of the data it is clear that some patients have been categorised as non-smokers whilst having been recorded as a smoker in previous years, therefore, any recording of '0' following a recording of '1' in an earlier year was changed to a recording of '2' to reflect the previous and current smoking status per patient. This is based on the assumption that an ex-smoker is more likely to be inaccurately recorded as a non-smoker than an inaccurate recording of being a smoker previously.

*Intermediate outcomes*

As outlined in more detail in chapter two, type 2 diabetes patients have an increased risk of cardiovascular and micro vascular complications. Good control of blood glucose levels, BP and lipid profiles can reduce patients' risk of these complications. These risk factors are therefore recommended by NICE to be routinely monitored to enable effective decision making regarding appropriate treatments [46].

HbA1c is a measurement of a patients' blood glucose control over the previous three months. High levels of HbA1c increases patients' risk of complications and HbA1c should ideally be maintained at 6.5% or lower [46]. As recommended by the register staff, values were limited to those greater than or equal to 2.5 and less than or equal to 23. 198 values outside this range were removed, leaving 80 patients without a recording for HbA1c during the study period [187].

High BP also carries increased risk of complications and therefore it is recommended in care guidelines that patients should be maintained at 130/80 millimetre of mercury (mmHg) or lower [46]. Whilst there were no specific limits for expected values of systolic BP (sBP, the numerator value) or diastolic BP (dBP, the denominator value) were provided by the register team, if the corresponding sBP was equal to or less than its dBP value both were removed. However, following a visual inspection of the range of values for both measures, any value of sBP less than 60 and greater than 260 were removed. Likewise, values of dBP less than or equal to 0 were removed. These were done on the basis of the marked differences in values compared to the rest of the study population.

To enable more complex analyses only sBP was analysed as an outcome variable. This figure was chosen, rather than dBP or a binary hypertensive variable, because sBP deteriorates with age. In addition, dBP is more commonly evaluated in people aged less than 50 years old [65].

Cholesterol (mmHg) is one indicator which makes up an individuals' lipid profile and it is recommended by NICE to be monitored and targeted to ensure that patients' risk of cardiovascular disease (CVD) is reduced. As recommended by the register staff, values were limited to those greater than or equal to 1.5 mmHg and less than or equal to 40 mmHg [187]. Values outside this range were removed. LDL-c, HDL-c and triglycerides form the rest of the

lipid profile and were also extracted from the register dataset. Yet despite them being identified as key risk factors for CVD, the low level of the recording for these indicators over the study period (see appendix D) meant they were not as suitable for inclusion in the analysis of cholesterol.

National Institute for Health and Care Excellence recommends measuring creatinine and the estimated glomerular filtration rate (eGFR) using the abbreviated modification of diet in renal disease (MDRD) four-variable equation annually. Estimated glomerular filtration rate is discussed in the next section. Both are indicators of patients' kidney function, with higher rates indicating poorer function. However, both measures are problematic due to variation depending upon other factors, such as body muscle mass. Creatinine levels can vary quite dramatically. Following advice from the diabetes register staff, these values were limited to those greater than or equal to 20 and less than or equal to 1400. Values outside this range were removed. In addition, a new binary variable indicating whether patients had a creatinine level greater than 300  $\mu\text{mol/l}$  was created in order to control for this when analysing blood glucose outcomes. Having a high creatinine levels can affect the way blood glucose is treated.

### *Diabetes related complications*

Chapter two outlined in more detail the consequences of type 2 diabetes. The complications described here are those which were recorded in the final dataset.

Patients' vascular history is recorded as '1' for 'Yes (ever)' and '0' for 'No' for the following groups of conditions: 'Ischaemic Cardiac Disease' (ICD), this refers to angina, myocardial infarction and/or heart attack [188]; a stroke or transient ischemic attack (TIA); and/or peripheral vascular disease (PVD). However, the date of the first vascular event is not recorded. To retain consistency in the recording of these indicators any recording of '0' for 'No' following a recording of '1' for yes was recode to '1'. This was based on the assumption that the initial recording was more likely to be accurate.

Retinopathy, at the time these data were extracted, was recorded in the diabetes register as follows: 0 = None, 1 = Background, 2 = Pre-Proliferative, 3 = Proliferative. However, during the study period these categories have changed several times, as such, the database manager recoded the data prior to releasing the data. The new variables were categorised as follows: 0 = None, 1 = Background, 2 = Advanced; where advanced retinopathy is anything more serious

than background retinopathy. The data from the diabetic retinal screening programme for 2006 and 2007 were recorded as follows: R0M0 = No diabetic retinopathy, no maculopathy; R1M0 = Background diabetic retinopathy, no maculopathy; R1M1 = Background diabetic retinopathy, maculopathy; R2M0 = Pre-proliferative diabetic retinopathy, no maculopathy; R2M1 = Pre-proliferative diabetic retinopathy, maculopathy; R3M0 = Proliferative diabetic retinopathy, maculopathy; R3M1 = Proliferative diabetic retinopathy, maculopathy. Due to the prevalence of some of the grades being very low, particular at the severe end of the scale, all the retinopathy data were recoded into a binary variable with any retinopathy recorded 1 and 0 if not. In 180 cases the values between the sources conflicted. In these cases the values from the retinal screening programme were favoured as this is the primary source.

Estimated glomerular filtration rates were calculated per patient per year for those who had a recording of their age, ethnicity and creatinine level. The calculation was made using abbreviated Modification of Diet in Renal Disease equation as recommended by NICE, SIGN, and the Renal Association [66]:

$$\text{eGFRml/min/1.73m}^2 = 186 \times (\text{Creatinine} / 88.4)^{-1.154} \times (\text{Age})^{-0.203} \times (0.742 \text{ if female}) \times (1.210 \text{ if black})$$

This calculation estimates the severity of kidney damage. Normal kidney function is considered greater than 90mls/min/1.73m<sup>2</sup>. The lower the eGFR the greater the damage to kidney function [66]. It should be emphasised that this calculation is only an estimate and several factors are likely to affect the accuracy of the result including: extreme body types, for example amputees; some ethnic groups and if creatinine levels are unstable or near normal. For near normal levels of creatinine the calculation tends to underestimate eGFR [66].

Ideally eGFR would have been modelled as an outcome variable, however, the results were not robust enough. As a consequence, the binary variable indicating whether microalbuminuria was present or not was used. This variable was chosen as monitoring for microalbuminuria is one of the NICE recommended care processes.

## Missing data

Secondary datasets can have varying degrees of quality in terms of accuracy and consistency of recording. Data can be missing from a dataset for various reasons and can cause various problems for analysis with varying degrees of severity.

One of the biggest problems associated with missing data is unit non-response; cases where no data was collected. This is contrasted with item non response where partial data is available but for individual indicators data is missing. This missing data results in these patients not being represented within analysis. This becomes a particular problem when it is particular population groups who do not engage with services as bias can be introduced to the analysis. Unfortunately this problem cannot be overcome with particular statistical techniques and can only be overcome by improvements in the initial data collection [172, 189].

Item non-response is a greater or lesser problem depending upon the pattern of 'missingness'. The first reason and least problematic is missing completely at random (MCAR). That is, the measurement missing is not related to its value or any other measurements in the dataset. For example, this would be violated if not having BMI recorded was related to patients being heavier and/or more deprived than those who had their BMI recorded. Missing completely at random is the most unlikely pattern of missing data. A more plausible assumption is that data are missing at random (MAR). In this scenario missing body mass index data, for example, could be related to another indicator such as deprivation but not with the value of BMI itself. It is not possible to be completely certain whether data is MAR as the value of the missing data is not available [172, 189]. There are various statistical analysis techniques which can be used for tackling the problem of MCAR or MAR data, however, missing data which does not satisfy these assumptions are more problematic. Whilst there are techniques for handling other missing data these are a lot more complicated and the estimated data is very sensitive to the models used. In addition, a very good knowledge of the reasons for missingness is required [172].

There are several methods for how to deal with partially missing data, however, many are relatively naïve and can introduce bias and reduce the precision of the subsequent analyses. An extremely common solution to dealing with missing data would be to only use complete cases; also known as listwise deletion. It is a technique that can be used for any type of statistical analysis. If the data is MCAR then the remaining cases represent a subsample of the larger sample but if it is MAR then bias can be introduced, especially if patterns of association vary between different population groups. In multivariate analyses the regression coefficient will be biased if the chance of being a complete case is related to the outcome variable after controlling for the other variables in the model. Pairwise deletion or available case analysis are similar to complete-case analysis but retains the complete cases for the variables for particular analyses. These also suffers the same potential in reduce precision and introduction of bias [172, 189].

There are several methods that could be described as single imputation where missing values are replaced with an estimated value. These methods include using the mean, median or values

generated from regression analysis of the observed data which could be from complete data of the same dataset or using similar, external data. In longitudinal data, carrying the last observation forward can be used to replace the missing data. All these techniques tend to underestimate the variance and again can introduce potential bias in the parameter estimates [172, 189].

The general principle of multiple imputation (MI) is to use the existing correlations between the observed data to predict a range of other plausible values. The variability in the range of values allows this uncertainty to be included in the final analysis whilst maintaining statistical power, unlike the more naïve approaches described above. This is also in contrast to maximum likelihood (ML), which accounts for the missing data but does not predict what it may have been [190].

Modelling data using MI is computationally intensive and becomes more complex when data has underlying multilevel structure. A less demanding approach to overcoming uncertainty in point estimates in statistical analyses due to missing data is Markov Chain Monte Carlo (MCMC) estimation. The advantage of this technique is that it can be applied to a wide number of statistical models and can take into account data with multilevel structures, MCMC estimations take a large number of random samples from the known data to estimate the unknown parameters with the aim to produce more robust interval estimates. It is an iterative process using the previous results to produce the next set of estimations with the aim to produce values based upon the unknown parameters and produce more accurate interval estimates [191].

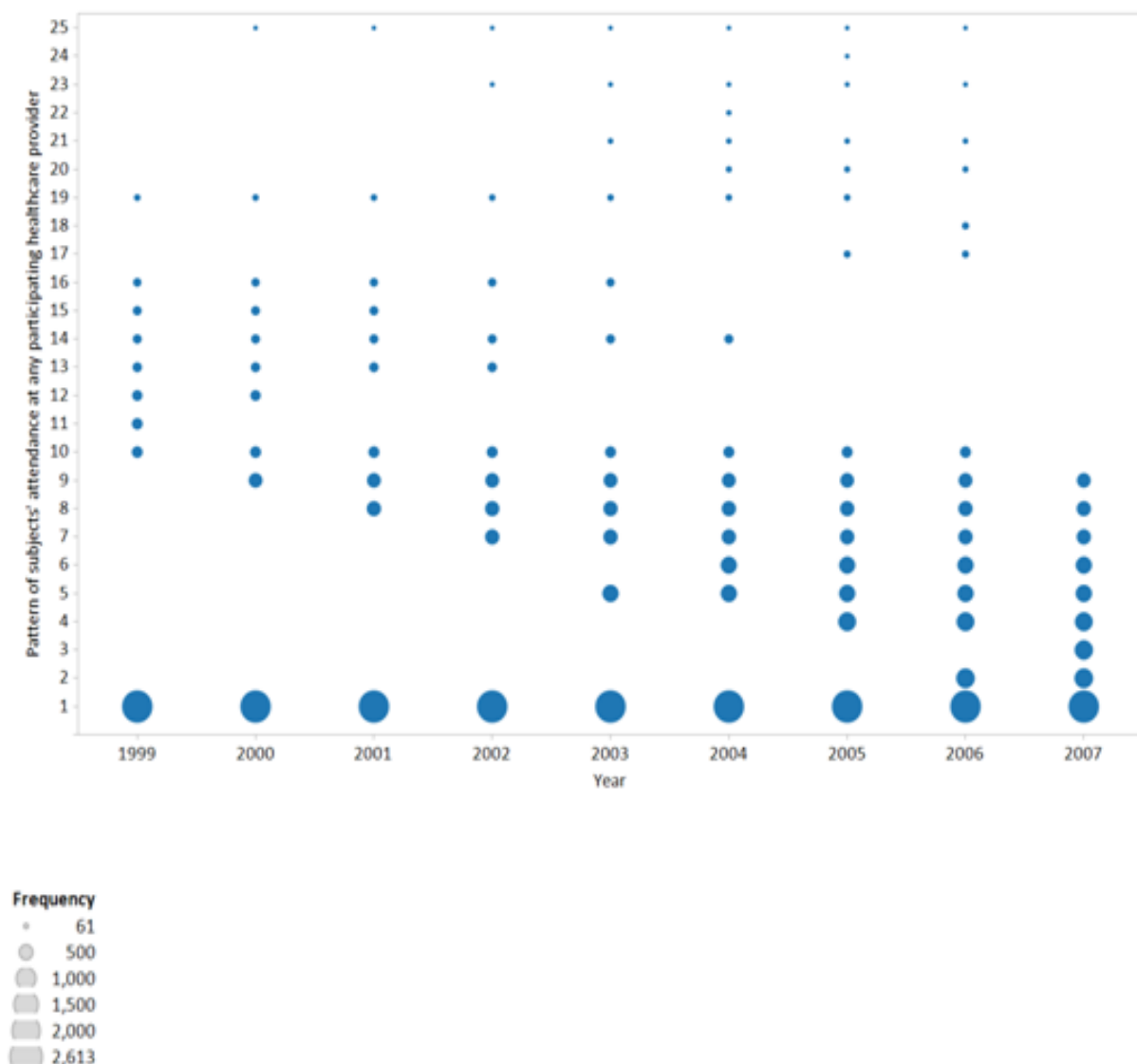
Prior to any statistical analyses, the prevalence and patterns of cases overall and per patient, the completeness of each variable and potential mechanism of missing data were explored. The purpose of examining these issues was to establish the appropriateness of study design, analysis methods and which variables to use. This was done in three steps.

Firstly, a table of patients' attendance at any participating healthcare provider per year was created using the final linked dataset. Then the missing patterns command in STATA [192] was used to examine the pattern of patient attendance over the study period. Figure 12 depicts the 25 most frequent patterns of patients' attendance, at any participating healthcare provider, representing just over 90% of the 13,597 subjects whose data is included in the final dataset. The most common pattern of attendance at any participating healthcare provider is every year between 1999 and 2007. However, this equates to only 21% of the study population. Markedly, the second most frequent pattern is subjects' having data recorded in 2006 and 2007, 8% of the study population. The overall pattern in Figure 12 shows that in general data capture improves over the study period. This is likely to reflect the increased prevalence of this period but may

also be a result of improved care and recall procedures; something which is explored further in these analyses.

In addition, Figure 12 shows that the final dataset contains unbalanced repeated measurements for over 10,000 patients. Unbalanced refers to the varying number of cases for each patient. Multilevel regression models are the most appropriate set of methods for this dataset as these methods can account for this clustering of measurements within patients and there is no assumption of equal numbers of cases for each patient [193].

Figure 12: Top 25 most frequent patterns of subjects' attendance at any participating healthcare provider



The second step with examining missing data was to the rate to which each variable was complete as a total of the number of cases for that year. The purpose of this was to establish which variables were appropriate for the inclusion in the final analyses. As mentioned previously, Appendix D contains tables showing the percentage that each variable considered for the analyses as a total of the numbers of cases per year. The tables were conditionally formatted with colour to ease interpretation: Bright yellow indicates 100% completed with red at the other end of the spectrum indicating no data for that variable that year.

The level of missing data per variable per year was discussed in more detail in the Appendix D. Two particular findings were of note: the level of missing data regarding HbA1c at the time of diagnosis and QOF data being recorded from 2004 onwards. These data were therefore analysed separately.

The final issue to explore regarding missing data was whether data was MCAR, MAR or not at random. As there is no way no knowing what the missing values were it is assumed that the data are either MCAR or MAR. To explore this issue the outcome variables were recoded as '1' for recorded and '0' for missing. Then using logistic regression analyses, the odds ratio of these new outcomes variables were calculated controlling for the following demographic variables: deprivation level, ethnicity, age and sex. The results in Appendix E show that the mechanism for missing data is not random and there are statistically significant relationships between the demographic of patients and missing data. However, these relationships were not uniform across all variables and patient characteristics.

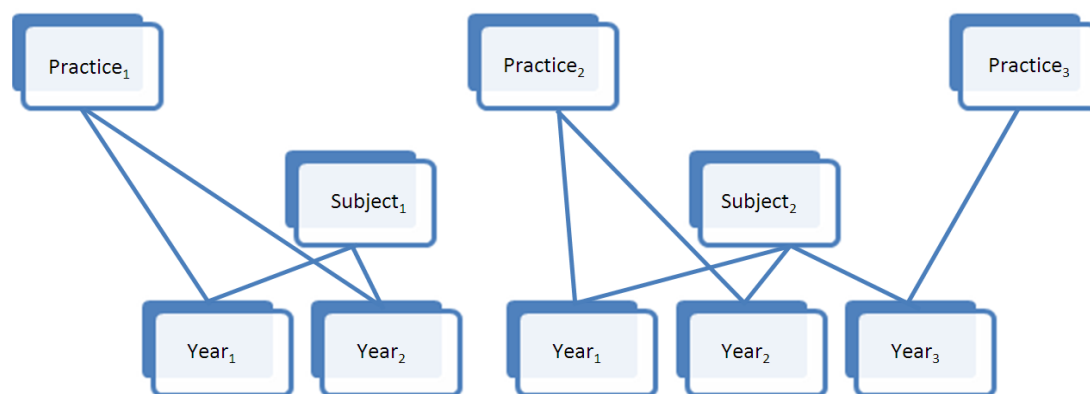
From these analyses of missing data, multilevel regression techniques were chosen using available case analyses. Using available cases instead of complete case retains more data in the model as patients with data for some years but not others can be retained. Multiple imputation of the dataset was considered to overcome the potential bias the missing data could introduce. However, due to the size of the dataset, the number of variables and the cross-classified, multilevel structure of the data producing robust imputations would be extremely computationally demanding. To overcome the uncertainty of the point estimates produced in analyses, the data were modelled with MLwiN in Stata using Markov Chain Monte Carlo (MCMC) estimation option [191]<sup>2</sup>. These methods take a large number of random samples from the known data to estimate the unknown parameters with the aim to produce more robust interval estimates [191].

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<sup>2</sup> The initial plan was to fit the models using the maximum likelihood (ML) method, IGLS (iterative generalized least squares), followed by the final models being fitted with MCMC. The rerun of the final models was going to act as a sensitivity analyses on IGLS fitted models which are computationally less demanding. However, many of the models using IGLS failed to convergence rendering the results unreliable therefore MCMC was used throughout.

The MCMC method has the added advantage that it can handle cross-classified data [191], which is data which is not strictly hierarchical. That is whilst repeated measurements are nested within patients who in turn are nested within general practices; about 7% of the patients in the study population changed practices at least once during the study period. Not accounting for these changes in the nesting of the data may bias the results. The diagram below illustrates the structure of the data.

**Figure 13: Diagram of the cross-classified data structure**



## Statistical analyses

The literature review highlighted the lack of complex analyses over time. This was mainly due to most of the data used in the final sample either being cross-sectional or only having two or three different measurements over time. This limited the type of analysis that could be conducted.

Two papers in the final sample made use of multilevel analyses [110, 120]; statistical methods which are becoming increasingly popular in health research. However, whilst the authors had repeat measurements these were not treated as such, with measurement occasions (years) being nested within subjects and analysed using multilevel regression models. Instead the authors only accounted for the clustering within place of care. One of the advantages of multilevel analyses is that it does not assume equal number of measurement occasions and allows for all observations to be retained in the model. This is in contrast to analyses such as MANOVA where, if there is a missing observation of an outcome variable, the entire case is

removed from the analyses [193]. Here that would mean entire cases would be removed potentially leading to bias in the results. In contrast, multilevel analyses retains each occasion where the outcome variable is recorded rather than deleting the entire case. However, this only applies to outcome variables. That is, if any explanatory variable was missing that case data was deleted.

Multilevel analysis, and Generalized Estimating Equations (GEE), are both able to take into account the potential hierarchical nature of health datasets. For example, patients are clustered within general practices which can influence their health outcomes. Using these techniques allows for the exploration of these factors, as well as controlling for them [193]. However, multilevel analysis is generally favoured as it is more flexible in terms of comparing relationships between groups, i.e. random coefficients, as well more complex data structure such as cross-classified and multiple memberships. That is, not all data are strictly hierarchical, as patients may change practices during a study period (cross-classified) or belong to two services at the same time (multi-membership).

Multilevel modelling techniques, such as time-lag models and autoregressive, are also able to take into account the relationships between repeated measurements using random coefficient regression. These techniques have the advantage that they are able to measure change over time and can measure the relationship between explanatory and outcome variables being modelled. However, assumptions, particularly with observational data, have to be taken about the directional of the relationships between the variables and what constitutes change in the outcome variables. Measuring change is particularly problematic when there are floor and ceiling effects in continuous variables, for example HbA1c [194].

Ideally random coefficient modelling would have been used for the statistical analyses. However, it was not possible using this data as the models failed to convergence. As a consequence, interaction effects between visit year and SES were used as an alternative to exploring longitudinal change by SES. This is a common, practical alternative to measuring differences in outcome variables over time by different groups (for example: [32, 195, 196]).

The remainder of this section describes each stage of analysis beginning with the general aspects moving through to specific analyses used to answer each question. This was an iterative process with earlier stages informing the remaining analyses.

## Stage one: Descriptive univariate analyses

The purpose of this stage was to describe the final dataset. These summary statistics are useful for establishing if there were any initial patterns of inequalities but they do not take into account any explanatory variables.

Univariate analyses were conducted calculating the mean, median or proportion of each variable by SES, as measured by IMD grouped into quintiles reflecting the national distribution. Ninety-five percent confidence intervals were used here and in subsequent analyses, where appropriate, to identify if any inequalities reached statistical significance. Variables were considered to make a statistically significant contribution to a model if the 95% confidence intervals did not cross 0. When examining comparative results, findings are considered statistically significant from each other if the confidence intervals did not overlap [197].

The median rate for each general practice and QOF variable was calculated, along with the interquartile range, by PCT. These outcomes were particularly skewed; therefore the mean was an inappropriate statistic. This data were compared by PCT as calculating the distribution by patient would have been inappropriate due to practices with a high proportion of type 2 diabetes patients would have been overrepresented in the analyses. These descriptive analyses provide an overview of the variation in these variables whilst comparing the quality of general practices by PCT.

Finally, intermediate outcomes, complications and interventions variables, which were used as dependent variables in the multilevel models, were graphically analysed by SES over time to examine whether they varied. Mean or percentage rates were used with 95% confidence intervals calculated to identify if any inequalities reached statistical significance. Due to the way patients' history of vascular disease were recorded the results from year 2000 onwards reflect only new incidences of these complications for that year. As such, once a patient had been recorded as having ICD, stroke or TIA or PVD their records were not included in subsequent years. This was done to avoid double counting. The same approach was used in the multilevel modelling, where data from 1999 was also excluded to ensure the results were not biased by patients with existing complications prior to 1999.

## Stage two: Random intercept multilevel modelling

Prior to fitting any multilevel models, a series of analyses were estimated to establish whether multilevel models were statistically necessary. These models were also estimated to see if having random intercept and/or random coefficients of SES were appropriate and at which level. These initially showed that due to the lack of variance of SES at the patient level that random coefficient models with SES at this level was not appropriate. A random intercept was, therefore, used at each level of the linear and practice and patient levels only for logistic multilevel regression analyses. Except where stated otherwise, all models were fitted with repeated measurements nested within patients who were cross-classified with general practices.

To ensure that the models had converged effectively each modelled was fitted using MCMC estimation for 100,000 iterations after a 10,000 burn-in, the number of iterations conducted before the iterations for the final MCMC estimation. This was chosen as a compromise between accuracy and timeliness as while a longer burn in length may have produced more reliable results an increase would have required more time and be more computationally demanding.

The Deviance Information Criterion (DIC) is often used as a measure of how a model fits the data, using information about both the fit and complexity of a particular model [191]. Here the Bayesian DIC statistics were compared to see if the more complex models fitted the data better than previous, simpler models. A smaller result indicates that there is less unexplained variance in the model and therefore it has a better fit. The intraclass correlation coefficients (ICC) of the null were calculated to establish how much variation was explained at the level of general practice level.

$$\rho = \frac{\sigma_{U_0}^2}{\sigma_{V_0}^2 + \sigma_{U_0}^2 + \sigma_e^2}$$

### Stage three: Analyses of research question one and two

1. Are there socio-economic inequalities in intermediate outcomes and complications associated with type 2 diabetes over time?
2. Are there socio-economic inequalities in interventions associated with type 2 diabetes over time?

A series of models with each intermediate outcome, long-term complication and intervention as the dependent variable were estimated to see if there were inequalities by patients' SES. These models were fitted with an interaction effect between SES and visit year to examine if inequalities occurred throughout the study period.

A stepwise approach to the analyses was used to compare how much the variation was explained by the introduction of sets of variables. In the first step, a null model was fitted to establish how much variance of the dependent variable occurred at each level. Secondly, the interaction effect between SES and visit year was added to see if there were statistically significant associations with the outcome variable occurred prior to other data being added to the model. Next, relevant socio-demographic, anthropometric, lifestyle and health covariates were added to see if any significant inequalities were explained by controlling for this data. In all models these data were as follows: socio-demographic – age, duration of diabetes, ethnicity, gender; lifestyle – smoking status and BMI. The included health data varied depending upon the outcome variable as not all were directly relevant. Having a creatinine level greater than 300 was only included when analysing patients' HbA1c. Being hypertensive was included in all models except when blood pressure treatments were under investigation where the separate continuous variables sBP and dBP were used instead. Cholesterol was included in all models except when analysing HbA1c and cholesterol. eGFR was included when analysing long-term complications and intervention outcomes. History of ICD, stroke or TIA and PVD were included in the analyses of intermediate health outcomes and intervention variables. Retinopathy and microalbuminuria were not included as their low recording rates would have reduced the number of available cases and therefore the robustness of the results. In the final step intervention data were added. In general, all other intervention data were considered important factors in the outcomes and, therefore, included in the models with a few exceptions. Firstly, it was not included when it was the dependent variable. General practice data were only included in sub-analyses for question four, due to only being recorded in the latter half of the study

period. Diabetes treatments were not included in cholesterol model, lipid therapies and aspirin were not included in HbA1c model, and no other treatment data were included when inequalities by SES in prescription of treatments were being modelled. This was because these relationships would have been difficult to interpret.

The health outcomes HbA1c and cholesterol, and the variables indicating the quality of care a patient receives and the timeliness of diagnosis were modelled as continuous outcome variables using linear mixed effect models:

$$Y_{ijk} \sim N(XB, \Omega)$$

$$Y_{ijk} = \beta_{0ijk} \text{cons}$$

$$\beta_{0ijk} = \beta_0 + v_{0k} + u_{0jk} + e_{0ijk}$$

$$\begin{bmatrix} v_{0k} \end{bmatrix} \sim N(0, \Omega_v) : \Omega_v = \begin{bmatrix} \sigma_v^2 & 0 \\ 0 & 0 \end{bmatrix}$$

$$\begin{bmatrix} u_{0jk} \end{bmatrix} \sim N(0, \Omega_u) : \Omega_u = \begin{bmatrix} \sigma_u^2 & 0 \\ 0 & 0 \end{bmatrix}$$

$$\begin{bmatrix} e_{0ijk} \end{bmatrix} \sim N(0, \Omega_e) : \Omega_e = \begin{bmatrix} \sigma_e^2 & 0 \\ 0 & 0 \end{bmatrix}$$

All long-term complications and variables indicating patients' receipt of diabetes treatments, BP treatments, aspirin, lipid therapies and shared care were modelled as binary variables using logistic random-intercept models.

$$Y_{ijk} \sim \text{Binomial}(\text{cons}_{ijk}, \pi_{ijk})$$

$$\text{logit}(\pi_{ijk}) = \beta_{0jk} \text{cons}$$

$$\beta_{0jk} = \beta_0 + v_{0k} + u_{0jk}$$

$$\begin{bmatrix} v_{0k} \end{bmatrix} \sim N(0, \Omega_v) : \Omega_v = \begin{bmatrix} \sigma_v^2 & 0 \\ 0 & 0 \end{bmatrix}$$

$$\begin{bmatrix} u_{0jk} \end{bmatrix} \sim N(0, \Omega_u) : \Omega_u = \begin{bmatrix} \sigma_u^2 & 0 \\ 0 & 0 \end{bmatrix}$$

$$\text{var}(Y_{ijk} | \pi_{ijk}) = \pi_{ijk}(1 - \pi_{ijk}) / \text{cons}_{ijk}$$

### Stage four: Analyses of research question three

3. Are there intervention-generated inequalities in type 2 diabetes care?

This question was answered by modelling health outcomes with interaction effects between SES and interventions. Significant results in the interaction effects were interpreted as the intervention differed in its association with the health outcome according to the patients SES and therefore could indicate the presence of intervention generated inequalities. That is, that interventions were differentially effective according to SES.

A stepwise approach to the analyses was used to compare how much the variation was explained by the introduction of sets of variables. Firstly, the null model to establish how much variance of the dependent variable occurred at each level. Secondly, an SES only to see if there were statistically significant associations with the outcome variable occurred prior to other data being added to the model. Next relevant socio-demographic, anthropometric, lifestyle and health covariates, the same which were used for the models estimates for question one and visit year, were added to see if any significant inequalities were explained by controlling for this data. Next relevant intervention data were added, the same as question one, and finally the interaction effect between the intervention of interest and SES.

### Stage five: Analyses of research question four

4. What impact do general practices have on inequalities by socio-economic status in diabetes care and health outcomes?

This question was addressed in two ways. Firstly, two series of models with HbA1c and cholesterol as dependent variables were fitted using the same forward stepwise approach as question three, excluding the interaction term. Here general practice data, including QOF indicators, were added as a final step of these analyses. This was to establish whether any of these indicators had an impact on the relationship between SES and intermediate outcomes.

Secondly, the ICC of all the multilevel analyses was reviewed to establish what impact accounting for clustering at general practice had on the overall analyses. A larger coefficient at this level indicates a greater variation in health outcomes and uptake of interventions by general practices suggesting care was not consistent across all practices.

## Presentation of results

In the following five chapters, the results are displayed in a series of tables and described, with a chapter for the initial descriptive analyses and one for each of the four research questions. The results of the fully saturated models are displayed, with the preceding stepwise models displayed in Appendices F-I. Due to the relationship between each set of analyses these chapters should be viewed together as cross references between them are made throughout.

The results for the research questions in the subsequent chapters were followed by a summary of the principle findings. The main discussion in chapter eleven explores the strength and limitations of the data and methods, the possible mechanisms and implications for clinicians and policymakers and finally the unanswered questions and future research.

Due to the volume of tables and variables included in each model the variables which reached statistical significance were coloured coded to ease the interpretation of the results. In the descriptive results, the SES group with the highest results were coloured red and the lowest was coded green, if the results were significantly different were each other. In the multilevel analyses, the variables were coloured red or green if they were positively or negatively associated with the outcome variable, respectively.

## Chapter 5: Descriptive Results

This chapter provides an overview of the final dataset using univariate analyses to illustrate the distribution of the available variables for the analysis across three socio-economic status groups. In addition, the outcome variables used in the multilevel models were also graphically analysed to examine whether there were socio-economic inequalities over time. The results were summarised and referred to in the subsequent results chapters.

### Socio-demographic, anthropometric and lifestyle data

Table 1 contains patients' socio-demographic, anthropometric and lifestyle data by socio-economic status. The results show that there were a higher proportion of men than women across each SES group in the study population. Over 90% of the patients were White and there was a statistically significant higher proportion of non-White patients in low SES patients compared to the high SES patients. Low SES patients were significantly younger at the end of 2007 and at the time of diagnosis than the rest of the study population. Using all patient records, the mean BMI and weight by SES indicated negative social gradients, with low SES patients having a significantly greater BMI and weight than the rest of the study population. Interestingly, high SES patients were both significantly more likely to be 'under or normal weight' or overweight but significantly less likely to be obese than low SES patients. There were significant social gradients in rates of current and non-smokers in patient records overall. With greater proportions of smokers in low SES patients. There are no differences in the duration of diabetes in whole years.

### Health status data

Using all available patients' records, Table 2 contains the intermediate health outcomes statistics for study population by SES. The results show that there were statistically significant differences by SES in mean sBP, with low SES patients having lower sBP than the rest of the

study population. In contrast, there were no significant differences in mean dBp or when these variables were measured as a binary variable indicating hypertension. Low SES patients had a significantly higher mean HbA1c compared to the rest of the study population. There were no differences in mean levels of cholesterol. Interestingly, mean creatinine levels were significantly lower in low SES patients compared to the high SES patients.

Table 3 displays the results for long-term complications statistics for the study population by SES. Long-term complications were compared by patients, and not patient records, with the 'worst' outcomes compared. There were clear negative social gradients in rates of PVD and retinopathy, with higher rates seen in low SES patients compared to high SES patients. Higher rates of ICD and stroke or TIA tended to be associated with low SES patients compared to high SES patients, but were not statistically significant. Rates of eGFR by SES reflect the findings for creatinine levels, with low SES patients having higher rates than high SES patients. However, there were no significant differences in microalbuminuria by SES.

Table 1: Socio-demographic, anthropometric and lifestyle data by socio-economic status

		Statistic	Low SES	Mid SES	High SES	Total	No. of Obs
Number		Number	6,319	3,262	4,016		
		(%)	(46.5)	(24.0)	(29.5)		
Male		%	52.6	53.2	55.8	53.7	13,597
		(95% CI)	(51.3, 53.8)	(51.5, 54.3)	(54.3, 57.3)	(52.8, 54.5)	
Ethnicity	White	%	91.6	96.8	96.0	94.2	
		(95% CI)	(90.9, 92.3)	(96.2, 97.4)	(95.4, 96.6)	(93.8, 94.6)	
	South Asian	%	7.1	2.7	3.4	4.9	
		(95% CI)	(6.4, 7.7)	(2.2, 3.3)	(2.8, 4.0)	(4.6, 5.3)	
	Other	%	1.3	0.5	0.5	0.9	
		(95% CI)	(1.0, 1.6)	(0.2, 0.7)	(0.3, 0.8)	(0.7, 1.0)	
Age at the of end of 2007 (years)		Mean	66.6	69.3	69.7	68.2	
		(95% CI)	(66.3, 67.0)	(68.9, 69.8)	(69.2, 70.4)	(67.9, 68.4)	
Age at diagnosis (years)		Mean	58.0	60.6	61.0	59.5	
		(95% CI)	(57.6, 58.3)	(60.1, 61.1)	(60.6, 61.4)	(59.3, 59.7)	
Duration of diabetes (years)		Median	7	7	7	7	69,226
		(IQR)	(7, 7)	(7, 7)	(7, 7)	(7, 7)	
BMI (kg/m <sup>2</sup> )		Mean	31.4	30.8	30.1	30.9	53,342
		(95% CI)	(31.4, 31.5)	(30.7, 30.9)	(30.0, 30.2)	(30.8, 30.9)	
BMI categories	Under or normal weight	%	14.2	14.6	16.3	14.9	
		(95% CI)	(13.8, 14.6)	(14.0, 15.2)	(15.7, 16.8)	(14.6, 15.2)	
	Overweight	%	30.8	35.5	39.1	34.4	
		(95% CI)	(30.2, 31.4)	(34.6, 36.3)	(38.4, 39.9)	(34.0, 34.8)	
	Obese	%	55.0	49.9	44.6	50.7	
		(95% CI)	(54.4, 55.6)	(49.0, 50.8)	(43.8, 45.4)	(50.3, 51.2)	
Weight (kg)		Mean	86.5	85.9	84.9	85.9	56,275
		(95% CI)	(86.3, 86.8)	(85.5, 86.2)	(84.6, 85.2)	(85.7, 86.1)	
Smoking Status	No	%	34.5	39.6	42.3	38.0	58,974
		(95% CI)	(34.0, 35.1)	(38.8, 40.4)	(41.6, 42.9)	(37.6, 38.4)	
	Yes	%	22.6	15.5	10.5	17.5	
		(95% CI)	(22.1, 23.1)	(14.9, 16.1)	(10.0, 11.0)	(17.1, 17.8)	
	Ex	%	42.8	44.9	46.4	44.6	
		(95% CI)	(42.3, 43.4)	(44.1, 45.7)	(45.4, 47.5)	(44.2, 45.0)	

**Table 2: Intermediate health outcomes statistics for study population by socio-economic status**

	Statistic	Low SES	Mid SES	High SES	Total	No. of Obs
sBP (mmHg)	Mean	139.4	140.8	140.6	140.1	62,195
	(95% CI)	(139.2, 139.6)	(140.5, 141.1)	(140.3, 140.9)	(139.9, 140.2)	
dBP (mmHg)	Mean	77.9	77.6	77.6	77.7	60,573
	(95% CI)	(77.7, 78.0)	(77.4, 77.7)	(77.4, 77.7)	(77.6, 77.8)	
Hypertensive (mmHg)	%	36.8	36.7	35.9	36.5	60,545
	(95% CI)	(36.2, 37.3)	(35.9, 37.5)	(35.2, 36.6)	(36.1, 36.9)	
HbA1c (%)	Mean	7.8	7.6	7.6	7.7	61,368
	(95% CI)	(7.8, 7.8)	(7.6, 7.6)	(7.5, 7.6)	(7.7, 7.7)	
Cholesterol (mmol/l)	Mean	4.8	4.8	4.7	4.8	60,437
	(95% CI)	(4.7, 4.8)	(4.8, 4.8)	(4.7, 4.7)	(4.7, 4.8)	
Creatinine (µmol/l)	Mean	101.0	101.9	102.6	101.7	60,886
	(95% CI)	(100.6, 101.5)	(101.2, 102.5)	(102.0, 103.2)	(101.4, 102.0)	

**Table 3: Long-term health outcomes statistics recorded during the study period by socio-economic status**

	Statistic	Low SES	Mid SES	5 = High SES	Total	No. of Obs
eGFR	Mean	65.6	64.1	63.9	64.7	57,880
	(95% CI)	(65.4, 65.9)	(63.8, 64.4)	(63.6, 64.1)	(64.6, 64.9)	
ICD	%	34.0	34.5	31.7	33.6	13,178
	(95% CI)	(32.9, 35.2)	(32.8, 36.2)	(30.2, 33.1)	(32.8, 34.4)	
Stroke or TIA	%	14.0	15.3	13.6	14.2	13,168
	(95% CI)	(13.1, 14.9)	(14.0, 16.6)	(12.5, 14.7)	(13.6, 14.8)	
PVD	%	11.0	10.5	8.8	10.2	13,139
	(95% CI)	(10.2, 11.8)	(9.5, 11.6)	(7.9, 9.7)	(9.7, 10.8)	
Any retinopathy	%	28.1	27.7	21.4	27.5	10,713
	(95% CI)	(26.9, 29.4)	(25.9, 29.4)	(17.0, 25.7)	(26.7, 28.4)	
Microalbuminuria	%	54.6	48.9	53.7	52.1	10,701
	(95% CI)	(53.3, 56.0)	(47.0, 50.8)	(48.2, 59.3)	(51.1, 53.0)	

Table 4: Mean HbA1c (%) at diagnosis for patients diagnosed during the study period by socio-economic status

	Statistic	Low SES	Mid SES	High SES	Total	No. of Obs.
HbA1c at diagnosis	Mean (95% CI)	7.9 (7.9, 8.0)	7.8 (7.7, 7.9)	7.6 (7.5, 7.7)	7.8 (7.7, 7.8)	5,687

Table 5: Prevalence of prescriptions for treatments for study population by socio-economic status

	Statistic	Low SES	Mid SES	High SES	Total	No. of Obs.	
Diabetes treatments	Diet alone	% (95% CI)	21.5 (21.0, 21.9)	26.0 (25.3, 26.7)	27.3 (26.7, 28.0)	24.2 (23.9, 24.5)	67,063
	Metformin or sulphonylureas only	% (95% CI)	39.1 (38.6, 39.7)	36.6 (35.9, 37.4)	36.0 (35.3, 36.7)	37.6 (37.3, 38.0)	
	Combination excluding insulin	% (95% CI)	19.6 (19.1, 20.0)	19.0 (18.4, 19.6)	17.7 (17.1, 18.2)	18.9 (18.6, 19.2)	
	Insulin only	% (95% CI)	12.3 (12.0, 12.7)	11.3 (10.8, 11.8)	13.2 (12.7, 13.7)	12.3 (12.1, 12.6)	
	Combination including insulin	% (95% CI)	7.5 (7.2, 7.8)	7.1 (6.7, 7.5)	5.8 (5.5, 6.2)	6.9 (6.7, 7.1)	
	BP treatments	No BP treatments	% (95% CI)	28.4 (27.9, 28.9)	24.8 (24.1, 25.5)	26.9 (26.2, 27.5)	
	ACEIs only	% (95% CI)	11.0 (10.7, 11.9)	11.4 (10.9, 11.9)	12.5 (12.0, 13.0)	11.5 (11.3, 11.8)	
	ACEIs plus other BP treatment	% (95% CI)	30.7 (30.2, 31.2)	32.7 (31.9, 33.4)	30.5 (29.8, 31.2)	31.1 (30.7, 31.5)	
	Other BP treatment combination	% (95% CI)	29.9 (29.3, 29.3)	31.1 (29.3, 30.4)	30.1 (29.4, 30.6)	30.2 (29.9, 30.6)	
Aspirin	% (95% CI)	42.8 (42.3, 43.4)	43.1 (42.3, 43.9)	41.6 (40.8, 42.4)	42.5 (42.1, 42.9)	64,016	
Lipid therapies	% (95% CI)	57.3 (56.7, 57.9)	56.3 (55.4, 57.1)	57.1 (56.4, 57.8)	57.0 (56.6, 57.4)	60,952	

Table 6: Proportion of care processes recorded for study population by socio-economic status

	Statistic	Low SES	Mid SES	High SES	Total	No. of Obs
Number of care processes recorded	Mean (95% CI)	6.0 (6.0, 6.0)	6.2 (6.1, 6.2)	6.2 (6.1, 6.2)	6.1 (6.1, 6.1)	67,967

Table 7: Proportion of patients by place of care for study population by socio-economic status

	Statistic	Low SES	Mid SES	High SES	Total	No. of Obs
Shared care	% (95% CI)	29.0 (28.5, 29.5)	23.0 (22.3, 23.6)	25.6 (25.0, 26.2)	26.6 (26.3, 26.9)	67,947
Middlesbrough PCT	% (95% CI)	62.9 (62.4, 63.4)	25.7 (25.1, 26.4)	47.4 (46.8, 48.2)	49.7 (49.4, 50.1)	67,947

## Intervention data

Table 4 shows the mean HbA1c (%) at diagnosis for patients diagnosed during the study period by SES.; the proxy measurement used as timeliness of diagnosis. The results show that there was an inverse trend between timeliness of diagnosis and SES, with the low SES patients having significantly higher HbA1c at diagnosis compared to high SES patients. **Table 5** shows the percentage of patients with a prescription for treatments by SES.

The results show that there was a social gradient in the proportion of patients being treated by diet alone. Low SES patients were less likely to be treated this way compared to the rest of the population. This trend was reversed for all the other diabetes treatments regimens except for being prescribed insulin only. Mid SES patients were significantly less likely to be prescribed insulin only compared to high SES patients.

Low SES patients were significantly more likely to be prescribed no BP treatments and less likely to be prescribed ACEI only compared to high SES patients. Mid SES patients were significantly more likely to be prescribed ACEI in combination with other BP treatments compared to both high and low SES patients. There were no statistically significant differences in prescriptions for other combinations of BP treatments, aspirin and lipid therapies.

Table 6 shows the mean number of care processes for the study population by SES. The results shows that low SES patients significantly having a lower number of care processes recorded compared to high SES patients. Finally, Table 7 shows that the percentage of patients managed in shared care and Middlesbrough PCT. Low SES patients were significantly more likely to receive shared care and Middlesbrough as their PCT of responsibility than high SES patients.

## Practice level data

Table 7 below summarizes the practice level data both overall and by PCT. The results show that practices in Middlesbrough PCT serve more deprived populations. Across each process and outcome indicators, practices in Middlesbrough PCT perform worse than those in Redcar & Cleveland PCT. The varying number of observations reflects the introduction and suspension of

particular QOF indicators over the study period. As a result, only those which appear consistently since the introduction of QOF were used in the subsequent analysis for this thesis

Table 8: Median rates of practice level variables by PCT

	Stat.	Middlesbrough PCT	Redcar & Cleveland PCT	Total	No. of Obs.
Practice deprivation score	Median (IQR)	42.0 (33.0, 46.0)	29.0 (23.0, 34.0)	34.0 (29.0, 46.0)	197
Practice list size (N)	Median (IQR)	7474 (5372, 9598)	6305 (5191, 9006)	7080 (5278, 8683)	155
Diabetes register size (N)	Median (IQR)	256 (177, 332)	260 (197, 315)	259 (178, 321)	155
QOF DM prevalence (%)	Median (IQR)	3.4 (2.8, 3.7)	3.8 (3.5, 4.2)	3.6 (3.2, 4.0)	155
BMI recording level (%)	Median (IQR)	92.0 (89.8, 94.7)	93.7 (92.1, 95.9)	92.8 (90.7, 95.6)	155
Smoking recording level (%)	Median (IQR)	97.8 (96.1, 99.0)	97.9 (97.0, 99.0)	97.9 (96.9, 99.0)	69
HbA1c recording level (%)	Median (IQR)	96.4 (94.1, 97.9)	97.0 (95.6, 97.9)	96.7 (94.9, 97.9)	155
Patients achieving HbA1c ≤ 7.4% (%)	Median (IQR)	46.3 (42.9, 49.9)	52.9 (46.1, 57.6)	48.4 (44.2, 55.9)	69
Patients achieving HbA1c ≤ 10% (%)	Median (IQR)	87.7 (84.3, 89.6)	90.6 (88.1, 92.8)	86.4 (89.1, 91.5)	155
Retinal screening level (1) (%)	Median (IQR)	84.8 (78.6, 88.5)	87.8 (82.0, 91.1)	86.5 (80.9, 89.7)	69
Peripheral pulses recording level (%)	Median (IQR)	86.6 (79.7, 89.2)	89.6 (86.2, 92.9)	88.1 (83.3, 91.5)	155
Neuropathy test recording level (%)	Median (IQR)	85.9 (80.0, 89.2)	90.0 (86.0, 92.1)	88.0 (83.3, 91.5)	155
BP recording level (%)	Median (IQR)	97.4 (96.0, 98.4)	98.4 (97.3, 99.0)	97.8 (96.7, 98.8)	155
Patients achieving BP ≤ 145/85 (mmHg) level (%)	Median (IQR)	71.2 (67.3, 76.4)	74.2 (66.7, 80.3)	72.6 (67.1, 79.0)	155
Microalbuminuria recording level (%)	Median (IQR)	81.4 (73.5, 87.3)	83.8 (77.1, 87.2)	82.6 (75.6, 87.2)	155
Serum creatinine recording level (%)	Median (IQR)	97.4 (96.1, 98.2)	97.9 (96.2, 98.8)	97.6 (96.2, 98.5)	69
Proteinuria/microalbuminuria treated w. ACE inhibitors level (%)	Median (IQR)	3.0 (1.3, 7.8)	10.6 (5.6, 17.6)	6.8 (1.5, 13.1)	155
Total cholesterol recording level (%)	Median (IQR)	96.5 (94.2, 98.0)	97.8 (96.4, 98.7)	95.7 (97.4, 98.3)	155
Patients achieving total cholesterol ≤ 5mmol/l (%)	Median (IQR)	74.2 (68.1, 77.3)	75.9 (71.6, 79.1)	75.0 (69.8, 78.1)	155
Influenza immunisation level (%)	Median (IQR)	77.2 (73.4, 81.8)	81.0 (78.2, 83.1)	79.4 (74.6, 82.7)	155
Patients achieving HbA1c ≤ 7.5% (%)	Median (IQR)	60.3 (54.2, 63.5)	66.2 (62.8, 71.3)	62.9 (57.8, 67.3)	86
Retinal screening level (2) (%)	Median (IQR)	82.9 (79.4, 85.4)	86.3 (84.0, 90.6)	84.9 (80.2, 87.5)	86
eGFR or serum creatinine recording level (%)	Median (IQR)	96.1 (94.6, 97.8)	97.2 (97.9, 99.1)	97.5 (95.4, 98.4)	86

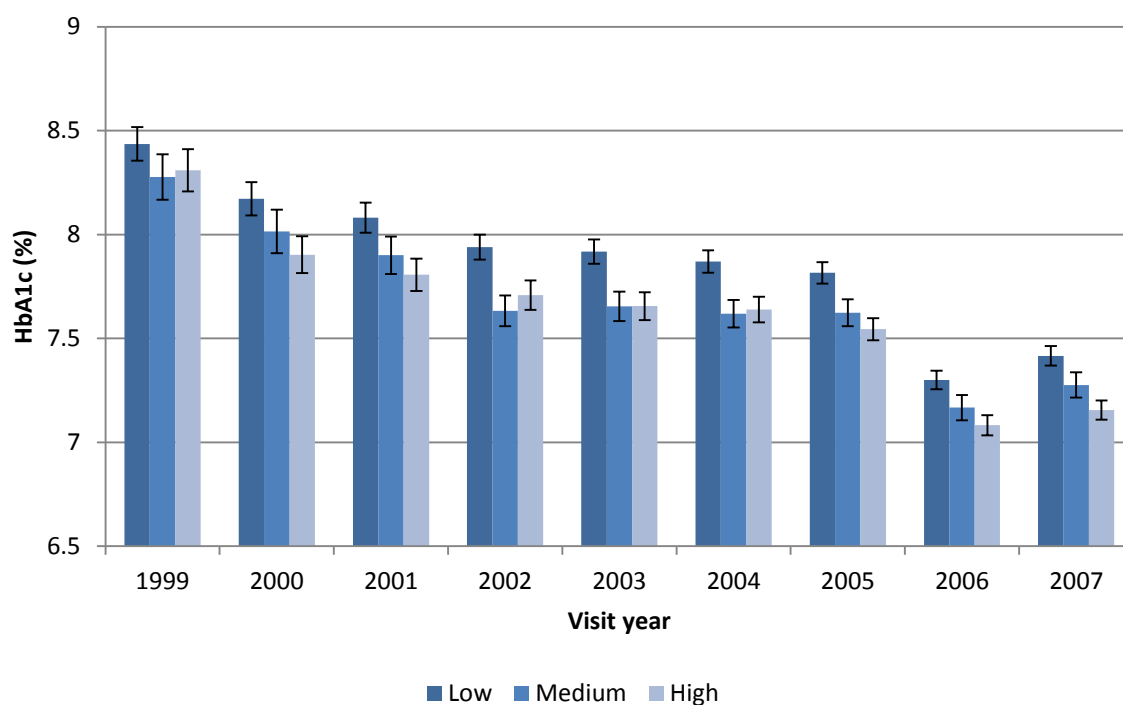
## Graphical analyses

This section examines the mean levels or proportion of the outcome variables used in the multilevel models in the subsequent chapters. The results were displayed in a series of bar charts with their 95% confidence intervals to establish the statistical significance of the results.

### Health outcomes

The first two figures indicate that there has been dramatic improvements in these two intermediate outcomes over the study period. These results are likely to reflect the changes in particular interventions in the diabetes care pathway over time, which were shown in the analyses in the next section. However, these time trends may also reflect the introduction of macro level policy initiatives which cannot be adequately accounted for, such as the NSF for Diabetes which outlined a number of standards and targets to be achieved [52]. The remaining figures in this section examine the levels of long-term complications by SES over the study period.

**Figure 14: HbA1c (%) by SES from 1999 to 2007 (N = 61,368)**



**Figure 15: Mean cholesterol level (mmol/l) by SES from 1999 to 2007 (N = 60,437)**

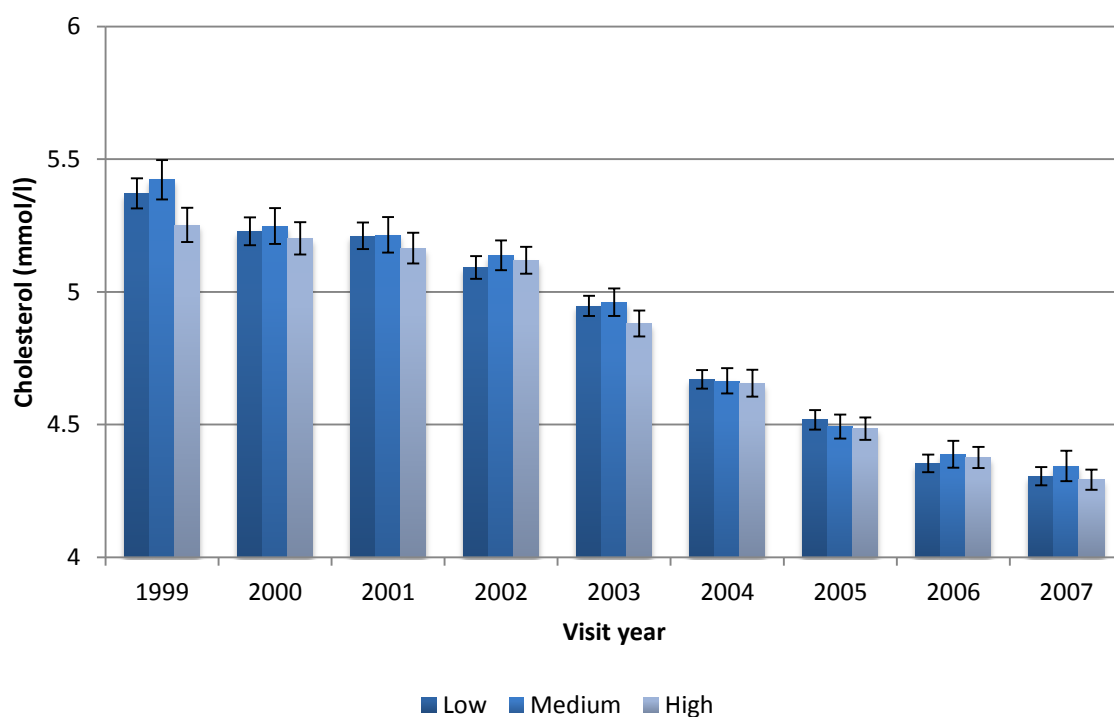


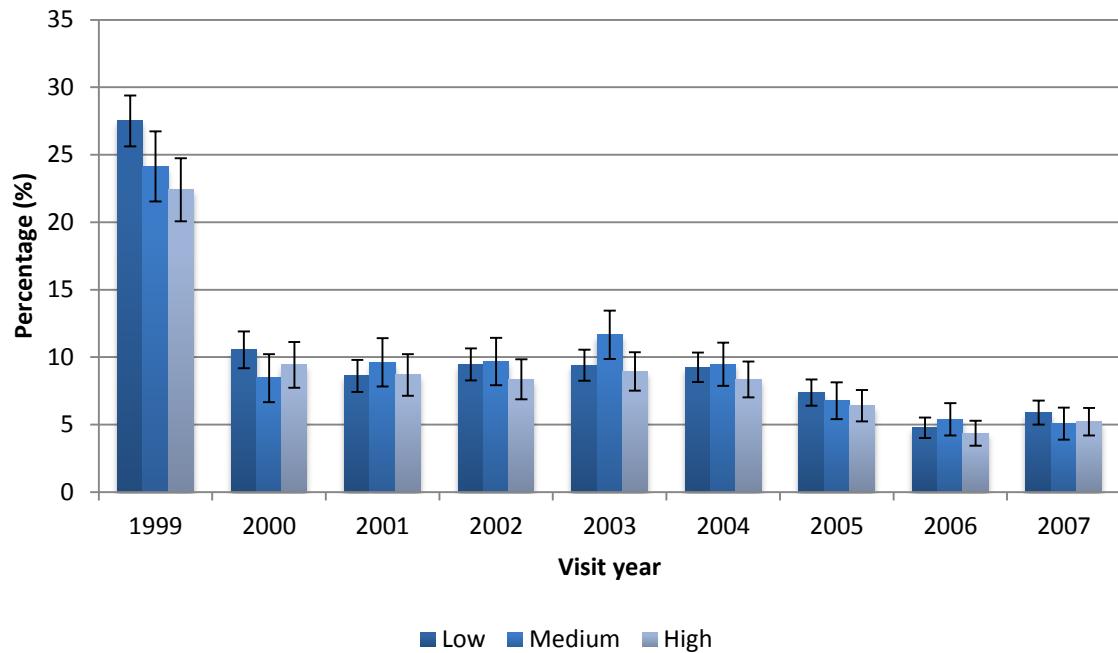
Figure 14 shows that there were statistically significant differences in HbA1c and that these differences have occurred over time. From 2000 patients with lowest SES consistently have significantly higher blood glucose control compared to with the highest SES patients; with a clear social gradient occurring in most years. In contrast, Figure 15 shows that statistically significant differences in cholesterol levels by SES only occurred in 1999 patients with the highest SES having the lower mean level of cholesterol compared to those with mid SES.

Figure 16, Figure 17 and Figure 18 display the levels of ICD, stroke or TIA and PVD by SES per visit year. The higher levels in 1999 reflect the proportion of patients who have a recording of these complications at the beginning of the study period. Subsequent results indicate a new recording for patients who with a 0 or no data recorded previously.

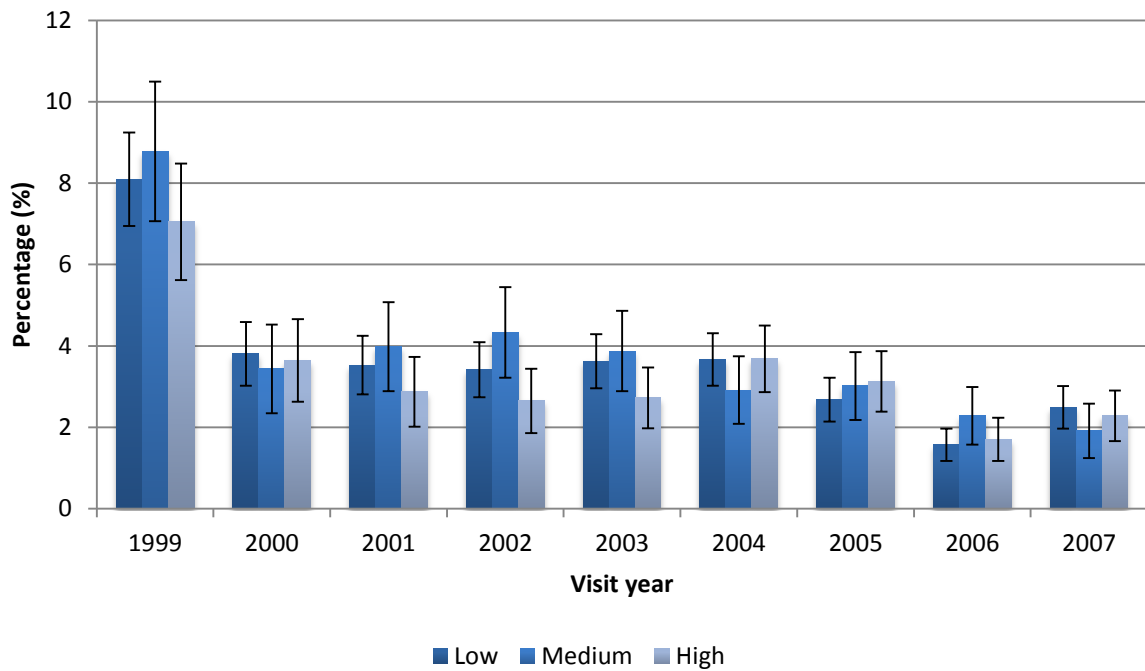
Figure 16 shows that there were statistically significant differences in the proportions of patients with existing ICD in 1999 by SES, with the lowest SES patients having significant higher levels than highest SES patients. There were no significant differences in the levels of ICD by SES in the other years but there was been a significant reduction in levels overall in years 2006 and 2007 compared to levels between 2000 and 2004.

Figure 17 and Figure 18 show that there were lower levels of stroke or TIA and PVD over the study period compared to levels of ICD yet the pattern of the results were similar. There were no significant differences in any year for levels of stroke or TIA and PVD but there were statistically significant improvements in the levels in 2006 and 2007 compared to earlier years.

**Figure 16: Percentage levels of ischaemic cardiac disease by SES from 1999 to 2007(N = 46,531)**



**Figure 17: Percentage of levels of stroke or TIA by SES from 1999 to 2007 (N = 55,400)**



**Figure 18: Percentage of levels of PVD by SES from 1999 to 2007 (N = 56,214)**

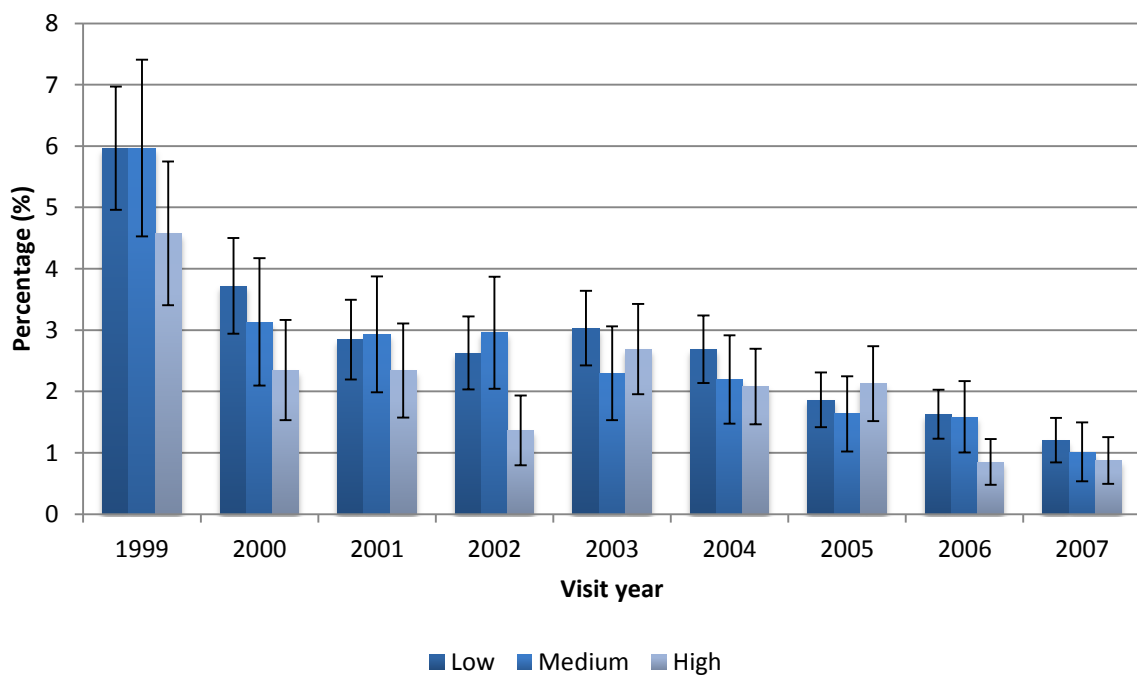


Figure 19 shows that in 2007, there were a statistically significant greater proportion of low SES patients who had any level retinopathy compared to high SES patients. No other year showed any statistically significant differences in retinopathy by SES. Unlike the figures depicting the percentage of patients with other vascular disease, there were no indications of any reductions in levels over time. In addition, there was a marked difference in the results for 2006 than the rest of the study period.

Figure 20 shows the percentage of patients with microalbuminuria during the study period. Between 2004 and 2006 there was a significant increase in the proportion of patients recorded as having microalbuminuria with low SES patients having statistically significant higher levels than the other patients. The levels of the outcome falls again in 2007, however, the wider confidence intervals were likely to reflect the poorer recording levels of this variable during this time (see Appendix D).

Figure 19: Percentage of patients with any retinopathy by SES from 1999 to 2007 (N = 30,980)

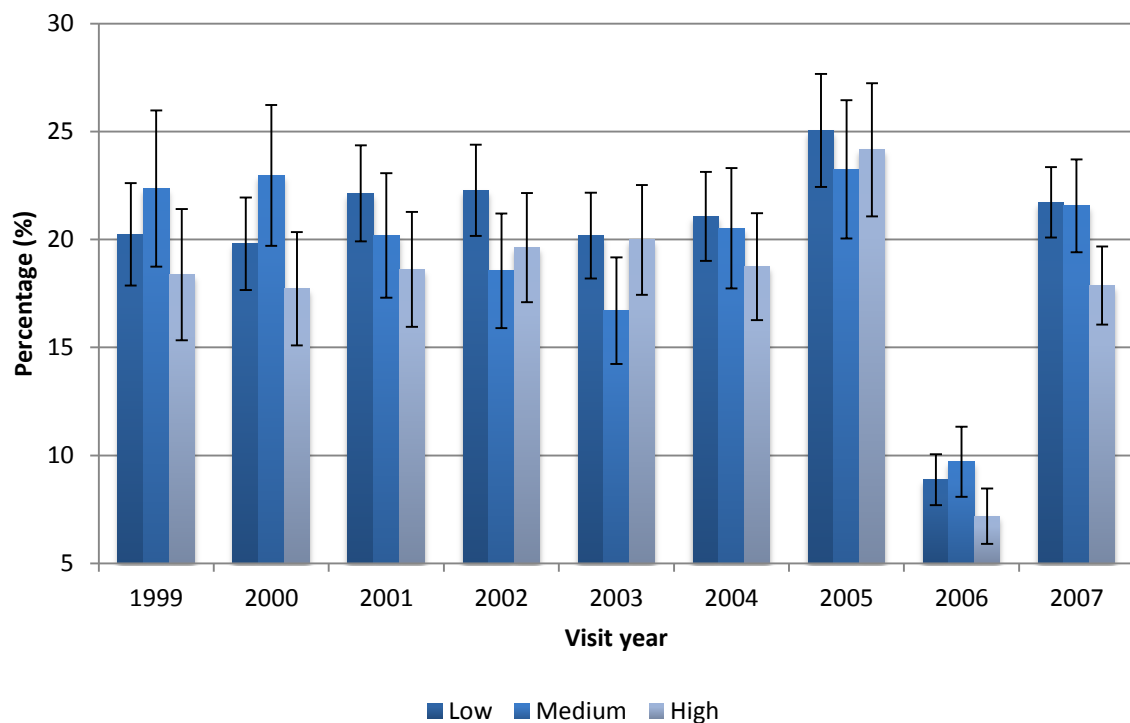
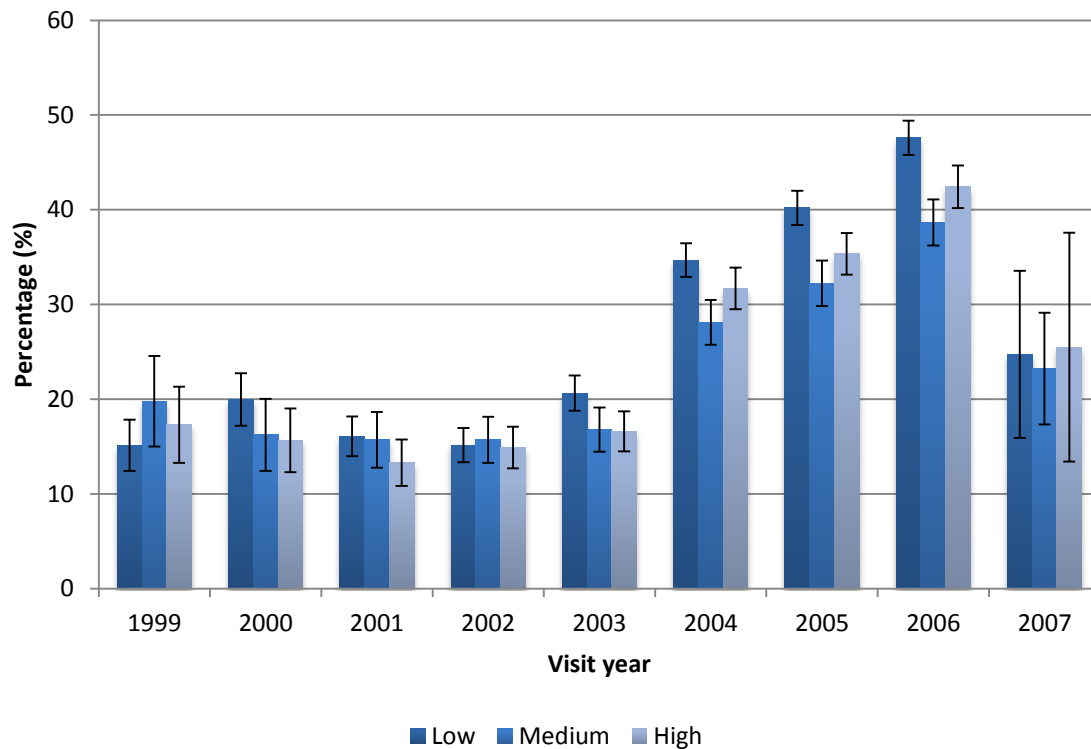


Figure 20: Percentage of patients with microalbuminuria by SES from 1999 to 2007 (N = 31,391)



## Intervention data

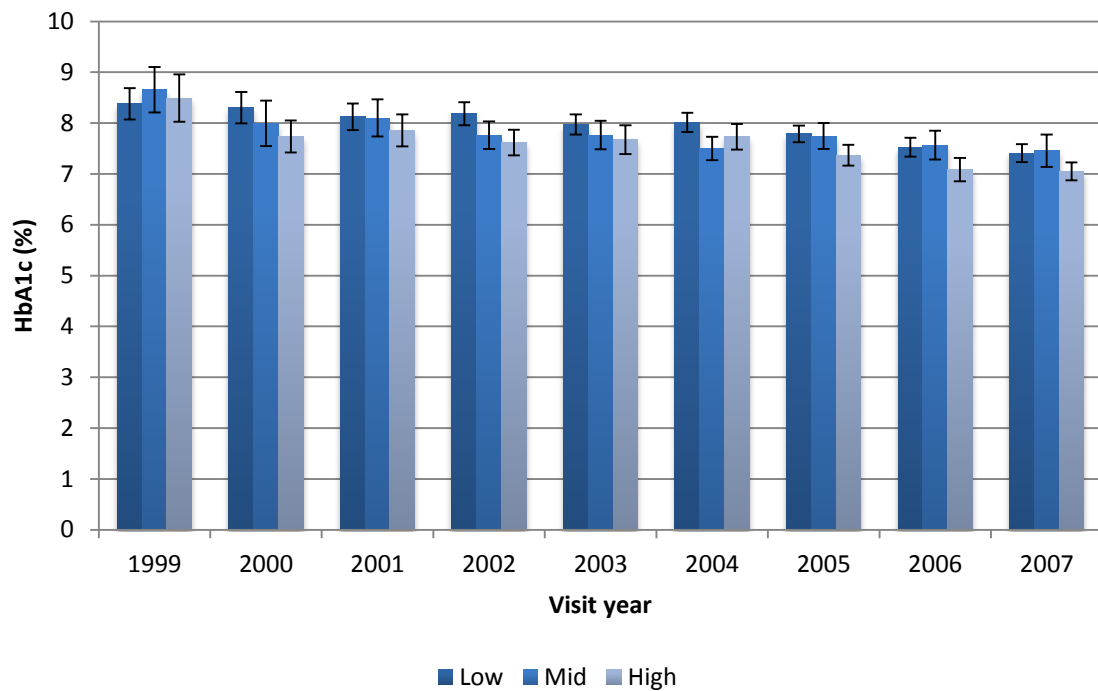
This section examines the timeliness of diagnosis and quality of care by SES from 1999 to 2007 as measured by patients mean level of HbA1c at diagnosis and the mean number of care processes received respectively. The percentage levels of diabetes, BP, aspirin and lipid treatments and being managed in shared care by SES over the study period were also examined over.

### *Timeliness of diagnosis*

Figure 21 uses the recording of patients HbA1c during the year the patient was diagnosed. 5,687 patients were diagnosed during the study period and captured in the dataset. The results show some evidence of statistically significant differences in timeliness of diagnosis by SES; with low

SES patients more likely to have a higher mean HbA1c to other status groups in 2002 and from 2004 to 2007.

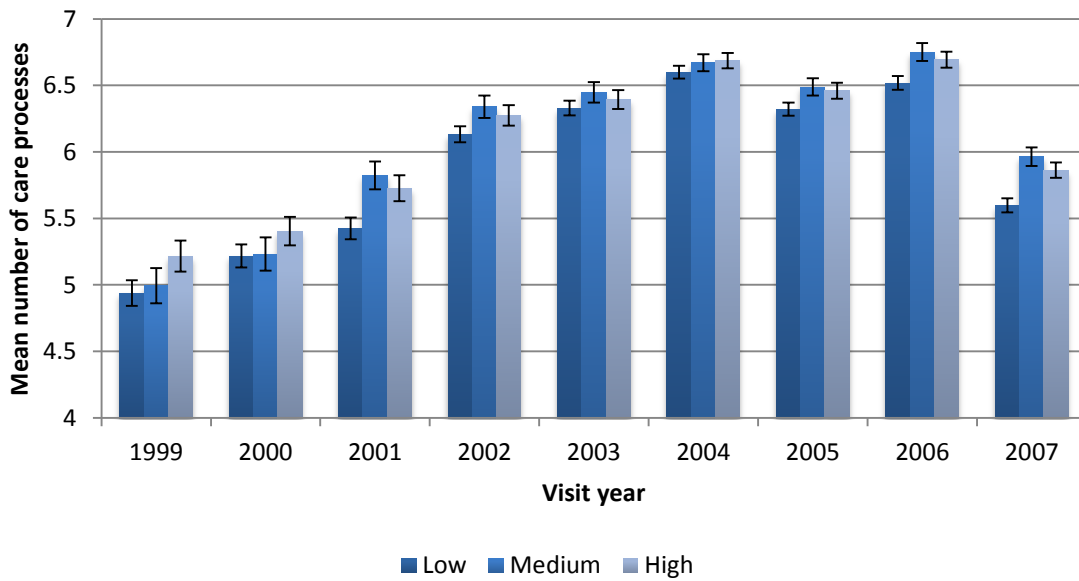
Figure 21: Mean HbA1c at diagnosis by socio-economic status over study period (N= 5,687)



### Quality of care

Figure 22 shows a steady improvement in the mean number of care processes that patients received by SES up to and including 2006. Low SES patients had significantly lower mean number of care processes compared to other status groups in most years.

Figure 22: The mean number of care processes by patients socio-economic status over the study period (N= 67,967)



*Glucose control treatments*

Table 22 displays marked statistically significant differences with patients from the lowest status group who were consistently less likely to receive no diabetes treatments. Between 1999 and 2001 high status patients were more likely to be treated this way compared to low status patients and from 2002 onwards both high and mid status groups. Interestingly, the levels of patients receiving no diabetes treatments appear to have fallen for low SES patients over time, but not for other SES groups.

Figure 24 shows an increasing prevalence of patients' diabetes being treated by metformin or sulphonylureas only over the study period. From 1999 through to 2004 there appears to be a negative social gradient in being treated with this regimen, with statistically significant differences in the first five years between high and low SES groups. In contrast, the prescriptions of more than one diabetes treatments with no insulin (Figure 25) and insulin only (Figure 26) have decreased over the study period with no significant differences by SES. The percentage of patients being prescribed insulin in combination with one or more diabetes treatment including insulin significantly increased between 1999 and 2005. From 2002 patients from low status groups were significantly more likely to be prescribed this treatment combination compared high status patients (Figure 27).

Figure 23: Percentage of patients prescribed no diabetes treatments by SES over the study period ( $N = 67,822$ )

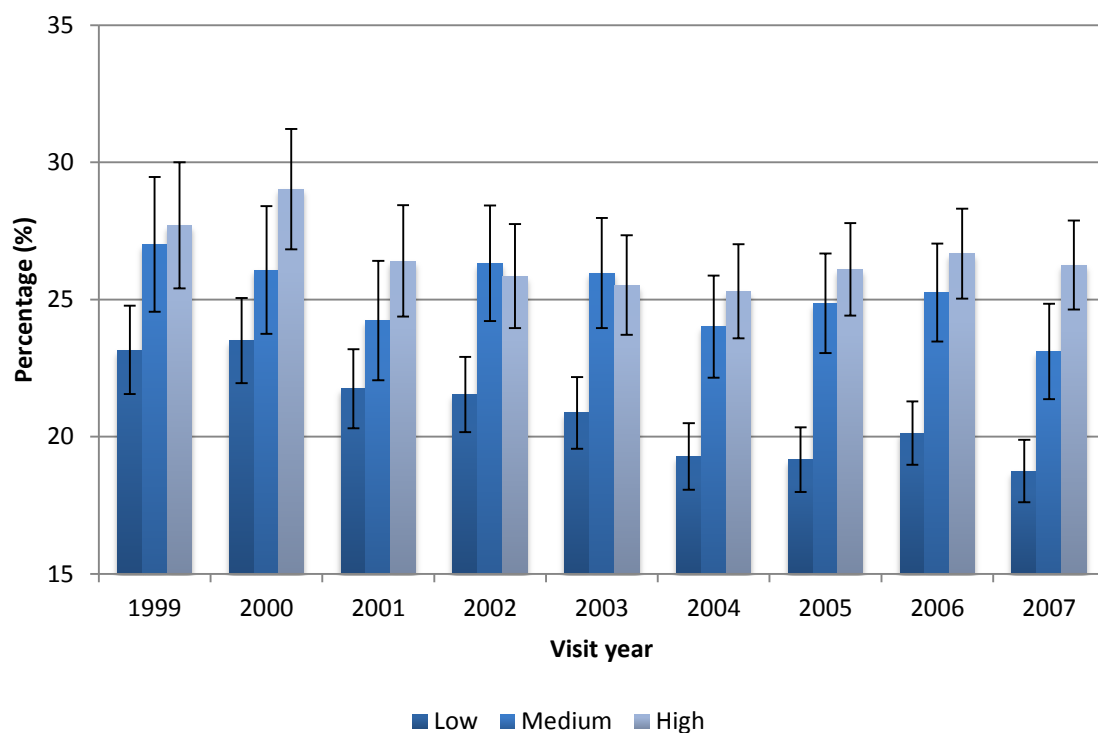


Figure 24: Percentage of patients prescribed metformin or sulphonylureas only by socio-economic status over the study period ( $N = 67,822$ )

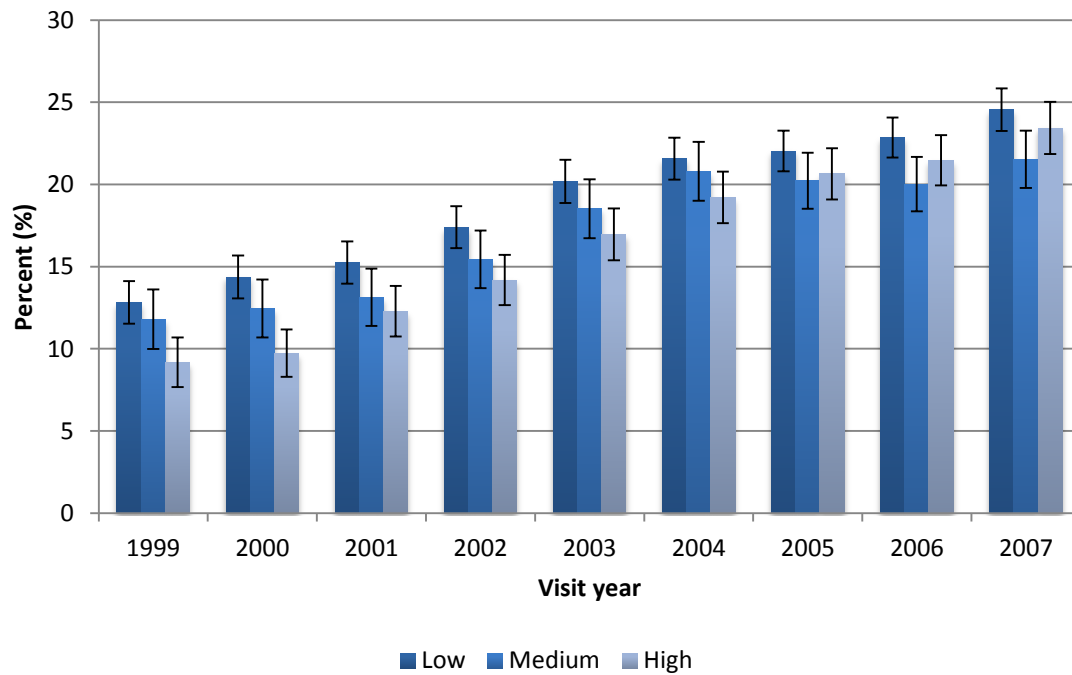


Figure 25: Percentage of patients prescribed combination of diabetes treatments with no insulin by socio-economic status over the study period ( $N = 67,822$ )

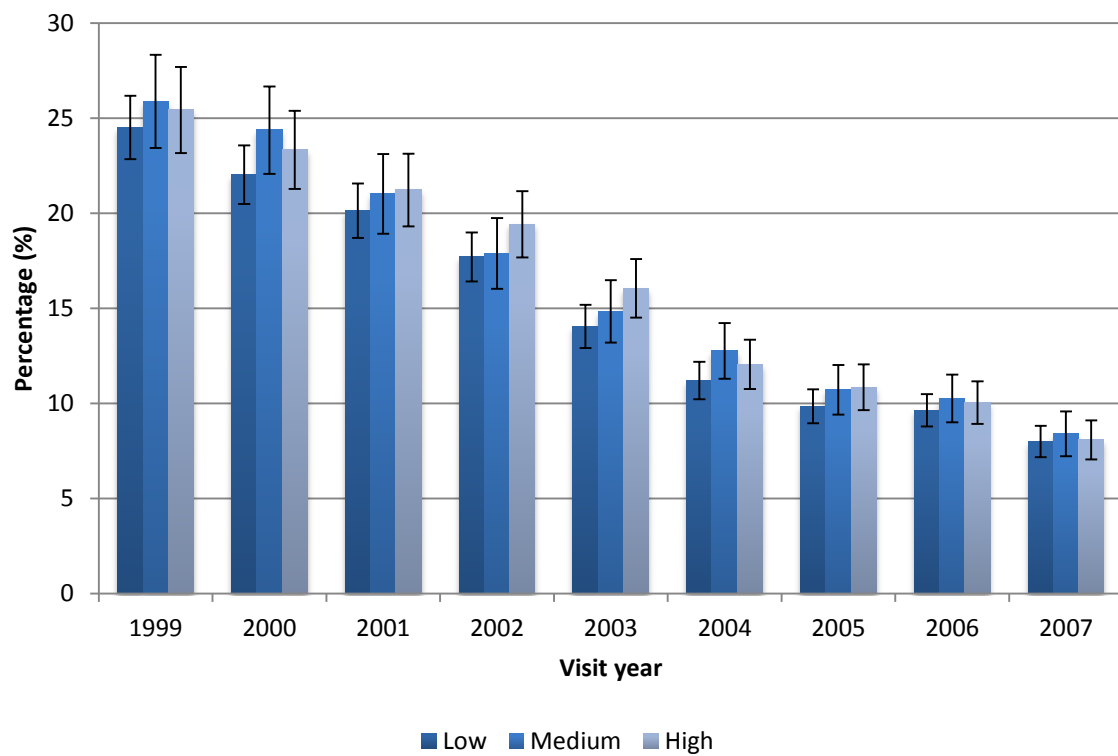


Figure 26: Percentage of patients prescribed insulin only by socio-economic status over the study period (N = 67,822)

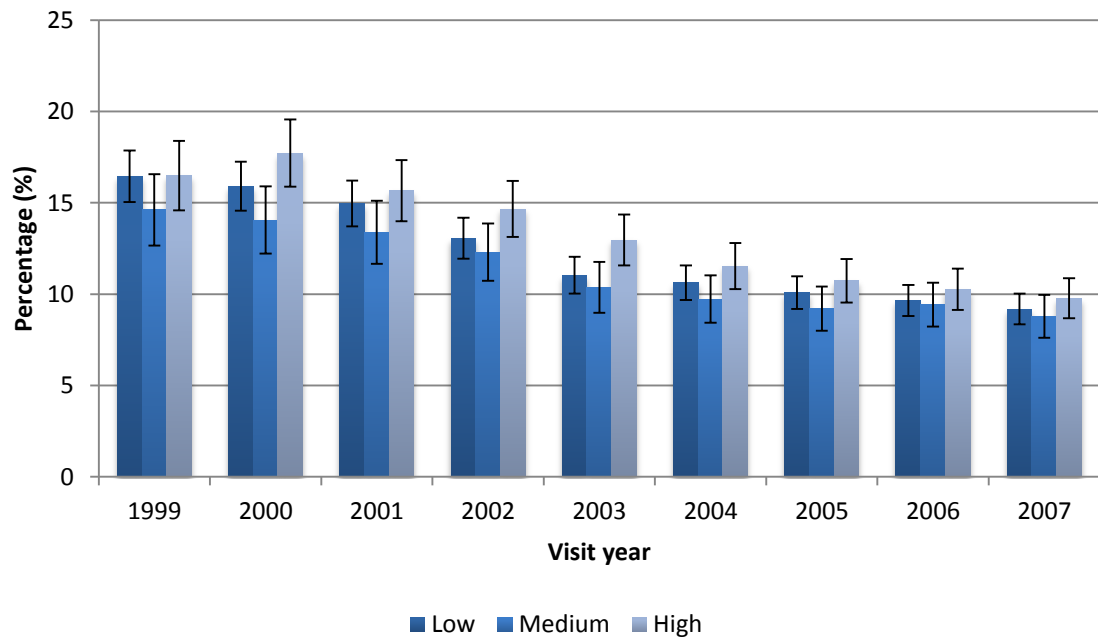
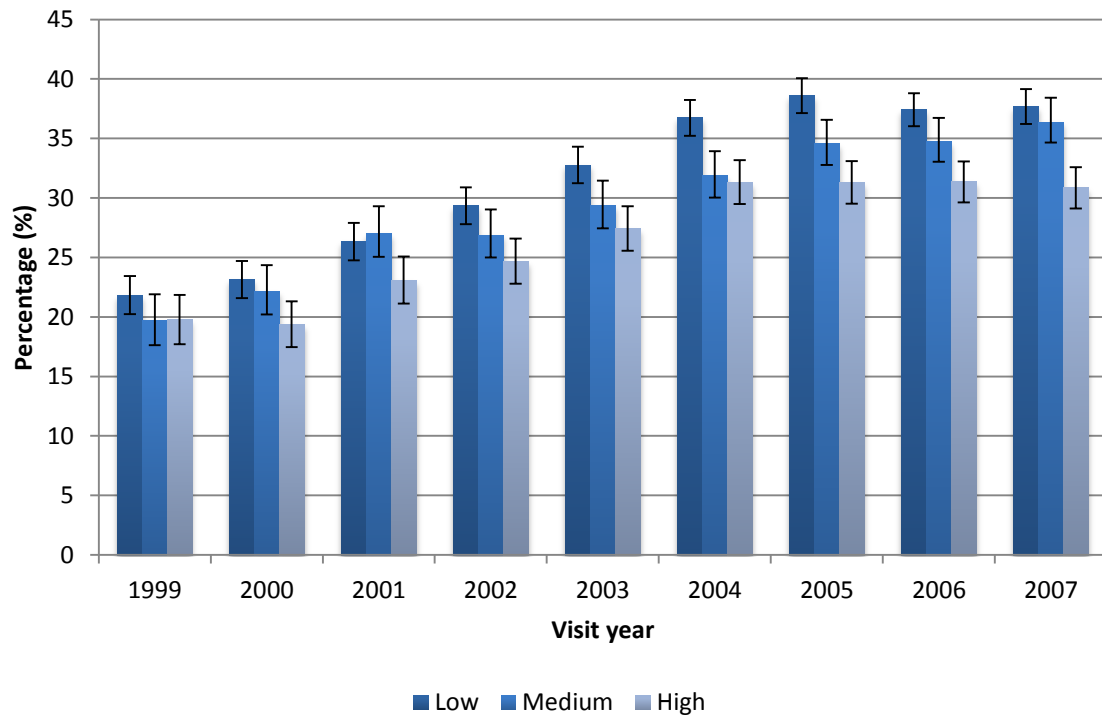


Figure 27: Percentage of patients prescribed other diabetes treatments and/or combinations by socio-economic status over the study period (N = 67,822)



### Blood pressure treatments

The results displayed in Figure 28 show an increase in blood pressure treatments being prescribed over the period with significant differences by SES occurring in a number of years. In particular, there were statistically significant higher proportions of low SES patients receiving no BP treatments compared to mid SES patients in 2006 and 2007.

Figure 29 and Figure 30 indicate that there were steady increases in the percentage prescribed ACEI, both alone and in combination with other treatments, in the earlier stages of study period which does not occur in the later years. Whilst there appears to have been a fairly consistent negative social gradient in the receipt of ACEI only, there were no statistically significant differences. There were no differences in use of ACEI in combination with other treatments (Figure 30).

Figure 31 shows that there was a steady decline in the use of other BP treatments in the first half of the study period and that there were no differences by SES.

Figure 28: Percentage of patients prescribed no blood pressure treatments by socio-economic status over the study period ( $N = 61,329$ )

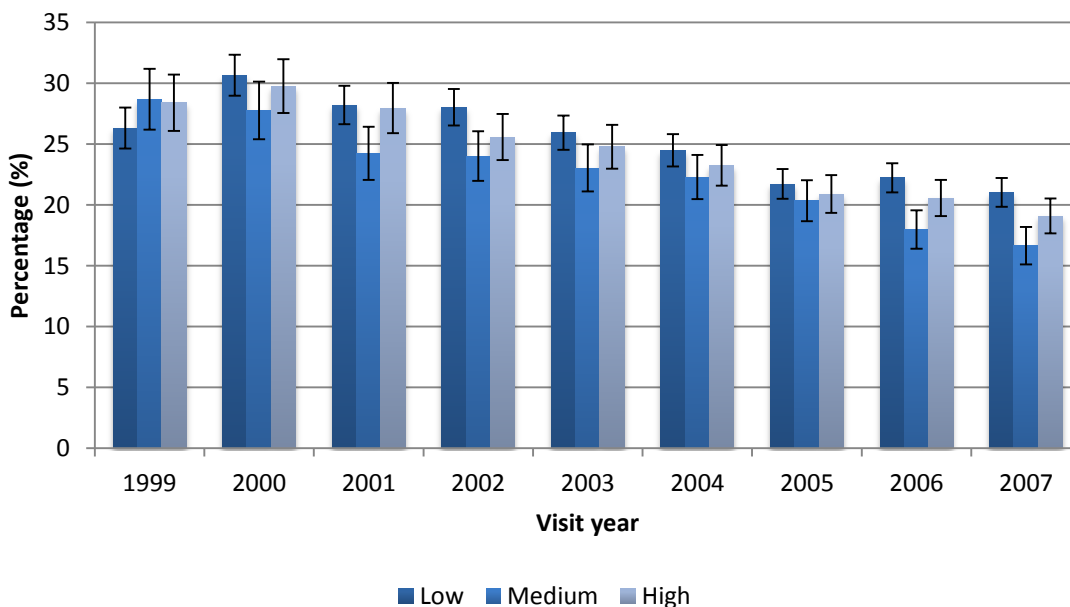


Figure 29: Percentage of patients prescribed ACE inhibitors only by socio-economic status over the study period (N = 61,329)

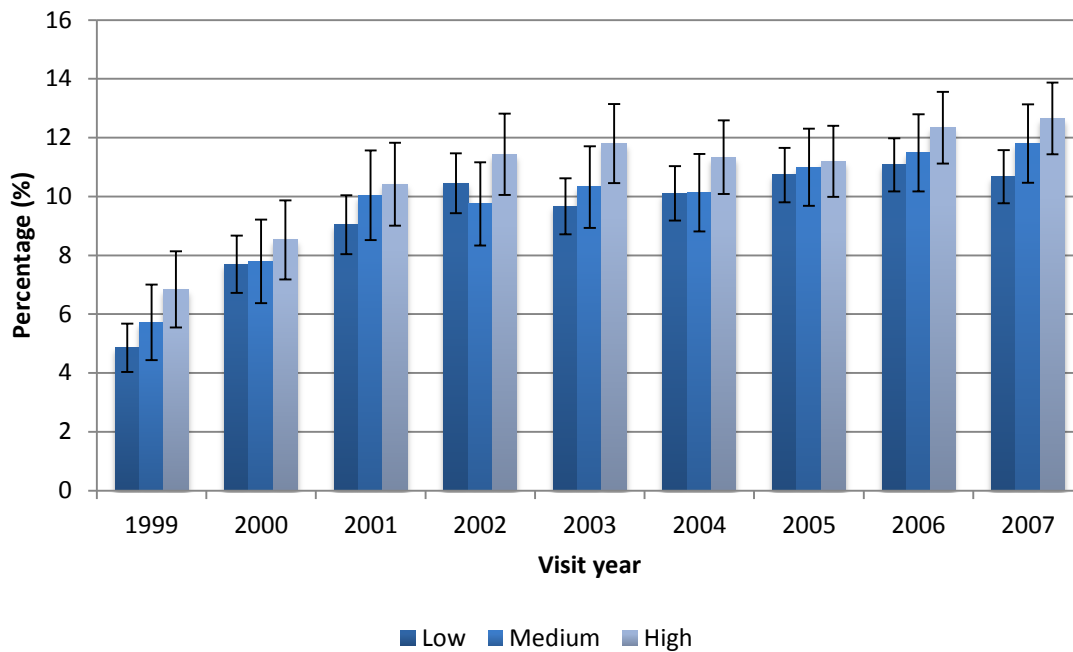


Figure 30: Percentage of patients prescribed ACE inhibitors plus other blood pressure treatment(s) by socio-economic status over the study period (N= 61,329)

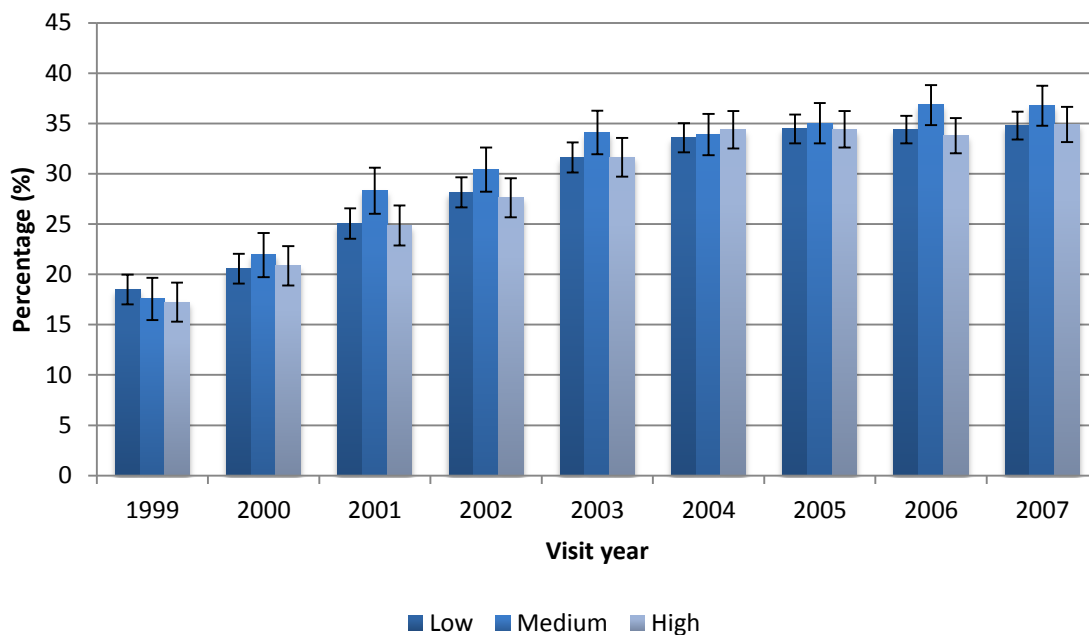
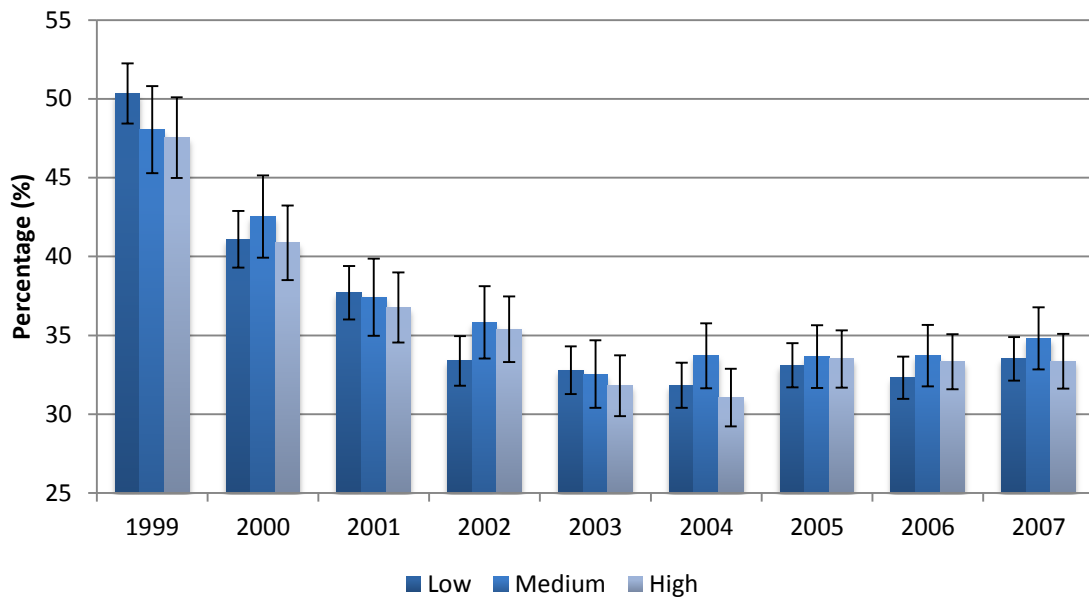


Figure 31: Percentage of patients prescribed other blood pressure treatment(s), excluding ACE inhibitors, by socio-economic status over the study period (N= 61,329)



### *Antithrombotic and lipid therapies*

Figure 32 and Figure 33 display the proportion of patients prescribed aspirin and lipid therapies by SES over time, respectively. The use of both types of therapies has increased over the study period. There were statistically significant differences between mid SES patients and high SES in the use of aspirin during 2002. Also, in 1999, there were also statistically significant differences between low SES patients and mid SES patients in the prescription of lipid therapies. Otherwise, there were no significant differences in the prescriptions of these treatments by SES over time.

Figure 32: Percentage of patients prescribed aspirin by socio-economic status over the study period (N=64,016)

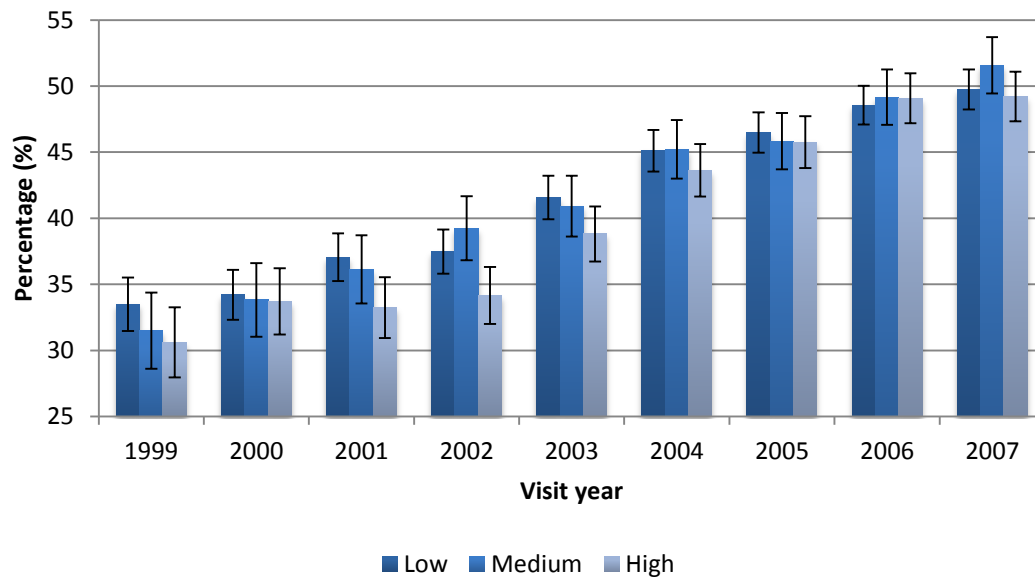
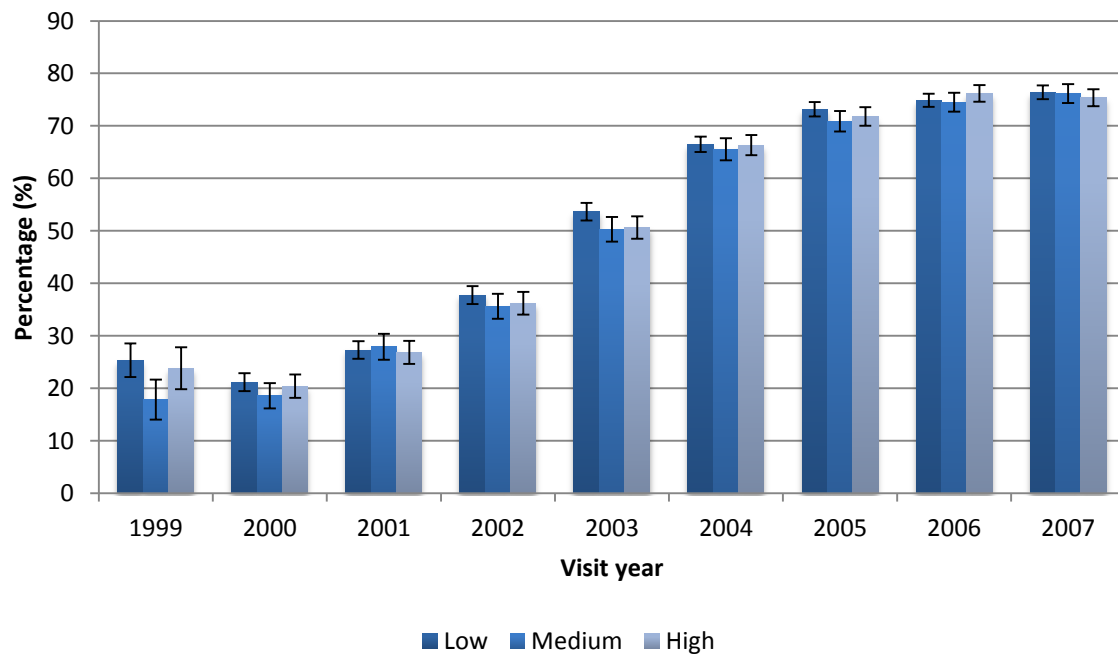


Figure 33: Percentage of patients prescribed lipid therapy(s) by socio-economic status over the study period (N = 60,952)



## Shared care

Figure 34: Percentage of patients receiving shared care by socio-economic status over the study period (N= 69,647)

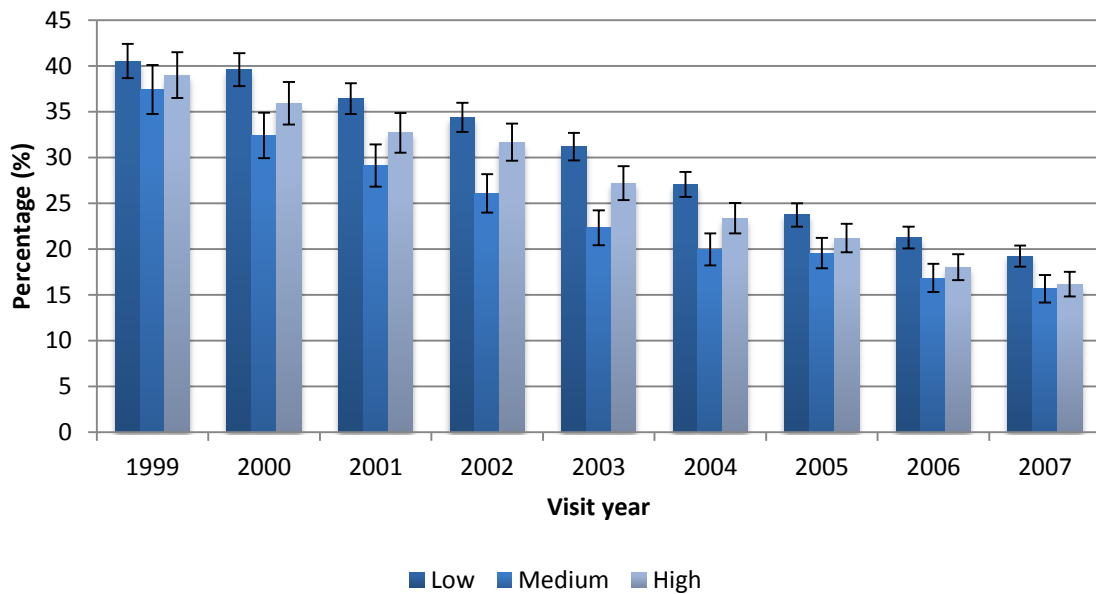


Figure 34 shows statistically significant differences in the receipt of shared care from 2000 to 2007. The chart shows a higher percentage of patients from low SES backgrounds compared to other status groups. Interestingly, it seems it was patients at the extreme ends of SES groups that were most likely to receive shared care, with this pattern reaching statistical significance in 2002 and 2003.

## Principle findings

This chapter describes the extensive dataset used for the secondary data analyses and highlights where the results indicate inequalities by SES in terms of both health outcomes and interventions. Low SES patients were found to be more likely to be younger overall and at diagnosis. There were negative social gradients in BMI and current smoking levels which reflected trends found elsewhere (for example:[198, 199]). In terms of health outcomes low SES patients were more likely to be hypertensive, have poorer HbA1c, and long-term complications but have lower levels of kidney problems. There were statistically significant differences in timeliness of diagnosis as measured patients' HbA1c, prescriptions for treatments and indicators of quality and place of care.

The graphical analyses indicated improvements over the study period for HbA1c, cholesterol, ICD, stroke or TIA and PVD. Levels of retinopathy were similar across the study period with a marked reduction in incidences in 2006. Levels of microalbuminuria increased over the study period. The charts also showed that there were improvements in timeliness of diagnosis and the level of care patients received. There were marked changes in prescriptions of treatments over time and a steady reduction in the proportions of patients receiving shared care. Generally, low SES patients had higher HbA1c overall and at time of diagnosis over time. There were significant differences in microalbuminuria in the later study years. There were marked significant differences in being treated by diet alone, receiving combination of diabetes treatments, and receiving shared care.

The next four chapters address each of the research questions, drawing and building upon these initial analyses.

## Chapter 6: Are there socio-economic inequalities in intermediate outcomes and complications associated with type 2 diabetes over time?

This chapter describes the multilevel models, which were fitted to examine whether there were socio-economic inequalities in intermediate outcomes and complications associated with type 2 diabetes over time, once other explanatory variables and structure of the data had been taken into account.

### Intermediate health outcomes

Table 9 shows the results for the saturated linear regression multilevel model for the comparison of HbA1c and cholesterol by SES. There were 38,413 available cases for the model comparing HbA1c by SES. The findings show that there were no significant differences in HbA1c by SES (Table 9); this finding was the same for each step of the modelling process. However, there were some statistically significant interactions effects between SES and time with high SES patients more likely to have greater HbA1c in 2000 and 2003 than low SES patients in 1999. Overall, visit year was statistically significant in all models supporting the graphical analyses that there have been reductions in HbA1c levels for type 2 diabetes patients in the South Tees area over time.

When examining the stepwise models (Table 56, Appendix F), the interaction effect between SES and time was partially explained by the introduction of socio-demographic, anthropometric, lifestyle and health data into the model. Increasing age, being male and having a creatinine level less than 300 were significantly associated with lower HbA1c. In contrast, increasing duration, being from a minority ethnic background, being a current or ex-smoker, overweight or obese, hypertensive and a having history of ICD and PVD were associated with higher levels of HbA1c.

The significant interactions effects were explained further when intervention data were added to the model. Increasing quality of care and time were significantly associated with lower HbA1c. All diabetes treatment regimens were significantly associated with higher HbA1c compared to those being treated by diet alone; similarly, shared care was significantly

associated with higher HbA1c. There were no differences in HbA1c across PCTs. Following the introduction of intervention data into the final model, being overweight and history of ICD were no longer significant and interestingly having a history of stroke or TIA and PVD became significantly positively related to HbA1c.

Table 9: Saturated linear regression multilevel models examining HbA1c and cholesterol by SES from 1999 to 2007 with interaction effect between SES and visit year, conditional on relevant explanatory variables

	HbA1c	Cholesterol
<b>Social-economic status &amp; visit year</b>		
Social-economic status, reference group: Low SES		
Mid SES	-0.09 (-0.26, 0.06)	0.06 (-0.13, 0.26)
High SES	0.04 (-0.10, 0.19)	-0.22 (-0.40, -0.05)
Visit year, reference group: 1999		
2000	-0.24 (-0.37, -0.12)	-0.25 (-0.38, -0.12)
2001	-0.47 (-0.59, -0.35)	-0.28 (-0.41, -0.16)
2002	-0.49 (-0.60, -0.38)	-0.29 (-0.41, -0.16)
2003	-0.53 (-0.64, -0.43)	-0.43 (-0.55, -0.31)
2004	-0.61 (-0.71, -0.50)	-0.64 (-0.76, -0.52)
2005	-0.68 (-0.79, -0.58)	-0.79 (-0.91, -0.67)
2006	-1.16 (-1.27, -1.06)	-0.92 (-1.04, -0.80)
2007	-1.11 (-1.22, -1.00)	-0.99 (-1.11, -0.87)
SES*Visit year, reference group: Low SES*1999		
Mid SES*2000	0.01 (-0.20, 0.23)	-0.01 (-0.24, 0.22)
Mid SES*2001	0.14 (-0.07, 0.35)	-0.02 (-0.24, 0.20)
Mid SES*2002	-0.04 (-0.23, 0.15)	-0.02 (-0.23, 0.20)
Mid SES*2003	0.01 (-0.18, 0.20)	-0.04 (-0.25, 0.17)
Mid SES*2004	0.03 (-0.15, 0.22)	-0.06 (-0.27, 0.15)
Mid SES*2005	0.05 (-0.13, 0.24)	-0.08 (-0.29, 0.13)
Mid SES*2006	0.08 (-0.11, 0.26)	0.02 (-0.18, 0.23)
Mid SES*2007	0.11 (-0.07, 0.30)	-0.03 (-0.24, 0.18)
High SES*2000	-0.26 (-0.46, -0.06)	0.16 (-0.05, 0.37)
High SES*2001	-0.09 (-0.28, 0.10)	0.26 (0.06, 0.46)
High SES*2002	-0.16 (-0.34, 0.02)	0.22 (0.02, 0.41)
High SES*2003	-0.20 (-0.37, -0.02)	0.20 (0.01, 0.39)
High SES*2004	-0.11 (-0.28, 0.06)	0.22 (0.04, 0.41)
High SES*2005	-0.14 (-0.31, 0.03)	0.22 (0.04, 0.41)
High SES*2006	-0.11 (-0.28, 0.05)	0.27 (0.08, 0.45)
High SES*2007	-0.13 (-0.30, 0.04)	0.21 (0.02, 0.39)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	-0.33 (-0.36, -0.29)	-0.20 (-0.23, -0.17)
Age: 75+ years	-0.41 (-0.46, -0.37)	-0.26 (-0.30, -0.23)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	0.06 (0.02, 0.09)	-0.09 (-0.11, -0.06)
Duration 10+ years	0.05 (0.01, 0.10)	-0.13 (-0.16, -0.10)
Ethnicity, reference group: White		
South Asian	0.46 (0.39, 0.54)	-0.08 (-0.14, -0.02)
Other Ethnicity	0.47 (0.31, 0.63)	0.08 (-0.05, 0.20)
Male	-0.06 (-0.09, -0.03)	-0.34 (-0.36, -0.32)
Smoking status, reference group: Non smoker		
Smoker	0.23 (0.19, 0.27)	0.08 (0.05, 0.11)
Ex-smoker	0.05 (0.02, 0.08)	0.00 (-0.03, 0.02)

BMI status, reference group: Low & normal weight		
Overweight	0.02 (-0.02, 0.07)	0.03 (0.00, 0.07)
Obese	0.08 (0.04, 0.13)	0.03 (0.00, 0.07)
Creatinine > 300	-0.81 (-1.06, -0.56)	
Hypertensive	0.10 (0.07, 0.13)	0.14 (0.12, 0.16)
Ischaemic Cardiac	0.00 (-0.04, 0.03)	-0.13 (-0.15, -0.10)
Stroke or TIA	-0.06 (-0.10, -0.01)	-0.02 (-0.06, 0.01)
PVD	-0.06 (-0.12, -0.01)	0.01 (-0.03, 0.05)
<b>Interventions</b>		
Quality of Care level, reference group: Low quality		
Mid quality	-0.13 (-0.16, -0.09)	-0.10 (-0.13, -0.07)
High quality	-0.15 (-0.19, -0.11)	-0.15 (-0.18, -0.11)
Diabetes treatment, reference group diet alone		
Metformin or sulphonylureas only	0.81 (0.77, 0.85)	
Combo. no insulin	1.25 (1.20, 1.29)	
Insulin only	1.67 (1.61, 1.73)	
Combo. with insulin	1.75 (1.69, 1.82)	
Aspirin		-0.09 (-0.11, -0.06)
Lipid therapy(s)		-0.28 (-0.31, -0.26)
Shared care	0.17 (0.13, 0.21)	-0.07 (-0.10, -0.04)
Middlesbrough PCT	0.10 (0.00, 0.20)	-0.03 (-0.09, 0.03)
<b>Cons</b>	7.56 (7.42, 7.70)	5.98 (5.85, 6.12)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.02 (0.01, 0.04)	0.01 (0.01, 0.01)
<b>Patient level</b>	0.00 (0.00, 0.02)	0.00 (0.00, 0.01)
<b>Visit year</b>	1.91 (1.88, 1.94)	1.14 (1.13, 1.16)
<b>Bayesian DIC</b>	133988.98	110350.13
<b>Available cases (N)</b>	38,413	37,085

There were 37,085 available cases for the model that compared cholesterol by SES. The results in Table 9 shows that there were statistically significant differences in cholesterol by SES, with high SES patients having more favourable cholesterol levels compared to the lowest status patients. In contrast, there were statistically significant interactions between SES and visit year with highest status patients having higher cholesterol levels compared to low SES patients in 1999. Visit year was consistently significant in all steps, which again supported the graphical analyses that there have been reductions in cholesterol levels for South Tees type 2 diabetes patients over time.

The introduction of explanatory variables did not explain the significant differences in cholesterol levels by SES and actually increased the number of years where the interaction effect between SES and visit year was statistically significant. This suggests that there were possible further interaction effects not controlled for in the model.

Following the introduction of socio-demographic, anthropometric and lifestyle covariates into the model, increasing age, duration, being South Asian, male and having a history of ICD and stroke or TIA were significantly associated with lower cholesterol. Being a smoker and hypertensive were significantly associated with higher cholesterol. All remained significant,

except having a history of stroke or TIA, following the introduction of intervention data into the model. Increasing quality of care, being treated with aspirin and lipid therapies and receiving shared care were all significantly associated with healthier cholesterol levels.

The Bayesian DIC statistics indicates that in both sets of stepwise models in Table 56 and Table 57 in Appendix F that more variance were explained as each set of variables were added to the model, improving the model fit. In the null model of both sets, the ICC for practice and patient level were less than 2%, suggesting that there was very little clustering of the data at these levels for intermediate health outcomes.

### Long-term complications

Table 10 and Table 11 show the results for the saturated logistic regression multilevel models examining long-term complications by SES.

There were 24,004 available cases for the comparison between incidences of ICD by SES. The results in Table 10 show that mid SES patients had statistically significant lower incidences of ICD over the study period compared to low SES patients. In addition, there was one statistically significant interaction result indicating the incidences were significantly higher for mid SES patients in 2003 than low SES patients in 2000. This finding was significant in all steps of analyses (**Table 58**, Appendix F) and was not explained by the introduction of other covariates. In contrast, there were no significant differences in the incidences of stroke or TIA (29,800 available cases), PVD (30,053 available cases), microalbuminuria (23,304 available cases) or retinopathy (18,665 available cases); nor any significant interactions between SES and visit year. These findings were consistent over each step of analyses for both outcomes.

Table 10: Saturated logistic regression multilevel models examining incidences of vascular disease by SES 2000 to 2007 with interaction effect between SES and visit year, conditional on relevant explanatory variables

	ICD	Stroke or TIA	PVD
<b>Social-economic status &amp; Visit year</b>			
<b>Social-economic status, reference group: Low</b>			
Mid	-0.73 (-1.31, -0.15)	0.20 (-0.50, 0.93)	0.06 (-0.85, 0.98)
High	-0.30 (-0.83, 0.23)	0.05 (-0.69, 0.80)	-0.99 (-2.04, 0.01)
<b>Visit year, reference group: 1999 or 2000</b>			
2000			
2001	-0.55 (-0.97, -0.10)	0.32 (-0.25, 0.92)	-0.38 (-1.00, 0.23)
2002	-0.36 (-0.73, 0.03)	0.21 (-0.32, 0.78)	-0.17 (-0.71, 0.37)
2003	-0.68 (-1.04, -0.30)	0.31 (-0.19, 0.85)	0.11 (-0.39, 0.63)
2004	-0.84 (-1.20, -0.45)	0.09 (-0.41, 0.64)	0.16 (-0.34, 0.67)
2005	-1.34 (-1.72, -0.94)	-0.20 (-0.71, 0.39)	-0.40 (-0.94, 0.14)
2006	-1.89 (-2.27, -1.46)	-0.87 (-1.45, -0.24)	-0.62 (-1.18, -0.06)
2007	-1.95 (-2.35, -1.52)	-0.53 (-1.11, 0.09)	-1.09 (-1.76, -0.45)
<b>SES*Visit year, reference group: Low SES*1999 or Low SES*2000</b>			
Mid SES*2000			
Mid SES*2001	0.54 (-0.26, 1.31)	-0.25 (-1.20, 0.67)	0.47 (-0.52, 1.49)
Mid SES*2002	0.41 (-0.28, 1.09)	-0.24 (-1.12, 0.62)	0.39 (-0.49, 1.32)
Mid SES*2003	0.91 (0.25, 1.58)	-0.05 (-0.89, 0.77)	-0.12 (-1.04, 0.80)
Mid SES*2004	0.60 (-0.09, 1.24)	-0.41 (-1.25, 0.41)	-0.18 (-1.04, 0.73)
Mid SES*2005	0.41 (-0.28, 1.08)	-0.20 (-1.07, 0.65)	-0.05 (-0.99, 0.93)
Mid SES*2006	0.69 (-0.02, 1.37)	0.16 (-0.75, 1.04)	-0.23 (-1.21, 0.75)
Mid SES*2007	0.37 (-0.37, 1.08)	-0.70 (-1.67, 0.26)	-0.02 (-1.10, 1.10)
High SES*2000			
High SES*2001	0.49 (-0.23, 1.21)	-0.13 (-1.09, 0.82)	0.13 (-0.87, 1.13)
High SES*2002	-0.11 (-0.76, 0.53)	-0.40 (-1.32, 0.50)	-0.81 (-1.81, 0.19)
High SES*2003	0.30 (-0.32, 0.92)	-0.56 (-1.44, 0.29)	-0.17 (-0.98, 0.68)
High SES*2004	0.13 (-0.48, 0.73)	0.16 (-0.67, 0.98)	-0.42 (-1.25, 0.44)
High SES*2005	0.02 (-0.60, 0.63)	0.17 (-0.71, 1.02)	0.35 (-0.48, 1.22)
High SES*2006	0.20 (-0.45, 0.84)	0.06 (-0.88, 0.99)	-0.46 (-1.40, 0.49)
High SES*2007	0.18 (-0.47, 0.83)	-0.07 (-1.00, 0.83)	-0.14 (-1.21, 0.91)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>			
Age, reference group: <60 years			
Age: 60-74 years	0.33 (0.18, 0.48)	0.74 (0.50, 0.99)	0.63 (0.38, 0.89)
Age: 75+ years	0.60 (0.41, 0.78)	1.10 (0.82, 1.38)	0.83 (0.52, 1.14)
Duration of diabetes, reference group: 0-3 years			
Duration: 4-9	-0.59 (-0.74, -0.45)	-0.31 (-0.49, -0.12)	0.13 (-0.10, 0.35)
Duration 10+	-0.63 (-0.80, -0.47)	-0.18 (-0.39, 0.03)	0.38 (0.14, 0.62)
Ethnicity, reference group: White			
South Asian	0.08 (-0.25, 0.40)	0.11 (-0.34, 0.53)	-0.79 (-1.52, -0.16)
Other Ethnicity	-0.68 (-1.55, 0.09)	-1.17 (-3.07, 0.17)	-0.04 (-1.10, 0.84)
Male	0.33 (0.21, 0.45)	-0.11 (-0.28, 0.06)	0.39 (0.20, 0.58)
Smoking status, reference group: non smoker			
Smoker	0.15 (-0.02, 0.32)	0.28 (0.05, 0.51)	0.94 (0.68, 1.19)
Ex-smoker	0.31 (0.18, 0.44)	0.14 (-0.04, 0.31)	0.38 (0.17, 0.59)
BMI status, reference group: under & normal weight			
Overweight	-0.02 (-0.20, 0.15)	-0.08 (-0.29, 0.13)	-0.20 (-0.46, 0.05)
Obese	0.12 (-0.06, 0.29)	-0.19 (-0.41, 0.03)	-0.25 (-0.50, 0.00)
HbA1c	0.07 (0.03, 0.11)	0.02 (-0.03, 0.07)	0.01 (-0.05, 0.07)
Hypertensive	-0.28 (-0.40, -0.16)	0.14 (-0.01, 0.30)	0.13 (-0.05, 0.31)
Cholesterol	-0.23 (-0.29, -0.17)	-0.09 (-0.17, -0.02)	-0.05 (-0.13, 0.03)
eGFR	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)
<b>Interventions</b>			
Quality of care level, reference group: Low quality OR Mid quality			
Mid quality	-0.31 (-0.44, -0.17)	-0.17 (-0.36, 0.03)	-0.18 (-0.42, 0.06)

High quality	-0.40 (-0.57, -0.24)	-0.03 (-0.24, 0.19)	0.19 (-0.06, 0.43)
Diabetes treatment, reference group diet alone			
Sulphonylureas/metformin only	-0.28 (-0.42, -0.13)	-0.18 (-0.38, 0.02)	-0.05 (-0.31, 0.21)
Combination, no insulin	-0.40 (-0.60, -0.20)	-0.30 (-0.56, -0.04)	-0.12 (-0.43, 0.20)
Insulin only	0.12 (-0.12, 0.37)	-0.08 (-0.39, 0.23)	0.33 (0.00, 0.67)
Combination, with insulin	-0.29 (-0.58, -0.01)	-0.29 (-0.68, 0.08)	0.33 (-0.05, 0.71)
Blood pressure treatment, reference group: No treatments			
ACE inhibitors only	0.26 (0.00, 0.52)	0.31 (0.03, 0.61)	0.50 (0.17, 0.83)
ACE & other(s)	1.50 (1.31, 1.70)	0.16 (-0.08, 0.40)	0.43 (0.15, 0.72)
Other combination	1.25 (1.05, 1.44)	0.18 (-0.05, 0.43)	0.38 (0.10, 0.67)
Aspirin	1.38 (1.26, 1.50)	1.06 (0.89, 1.23)	0.57 (0.40, 0.76)
Lipid therapy	0.61 (0.48, 0.74)	0.08 (-0.09, 0.26)	0.10 (-0.09, 0.30)
Middlesbrough PCT	-0.20 (-0.39, -0.01)	-0.12 (-0.39, 0.14)	-0.17 (-0.54, 0.21)
Shared care	0.27 (0.11, 0.43)	0.45 (0.24, 0.65)	0.85 (0.63, 1.07)
<b>Cons</b>	-3.50 (-4.69, -2.18)	-5.14 (-6.50, -4.01)	-6.09 (-7.60, -4.69)
<b>Variance estimate at:</b>			
<b>Practice level</b>	0.05 (0.02, 0.11)	0.11 (0.04, 0.22)	0.28 (0.14, 0.48)
<b>Patient level</b>	2.15 (0.53, 7.50)	1.36 (0.33, 4.57)	1.43 (0.34, 4.92)
<b>Bayesian DIC</b>	9208.63	6143.66	5033.64
<b>Available cases (N)</b>	24,004	29,800	30,053

Table 11: Saturated logistic regression multilevel models examining incidences of vascular disease by SES 2000 to 2007 with interaction effect between SES and visit year, conditional on relevant explanatory variables

	Microalbuminuria	Retinopathy
<b>Social-economic status &amp; Visit year</b>		
<b>Social-economic status, reference group: Low</b>		
Mid	0.52 (-0.20, 1.29)	-0.38 (-1.07, 0.26)
High	0.36 (-0.27, 1.07)	-0.45 (-1.00, 0.12)
<b>Visit year, reference group: 1999 or 2000</b>		
2000	-0.11 (-0.60, 0.41)	-0.17 (-0.60, 0.26)
2001	-0.35 (-0.83, 0.16)	-0.25 (-0.67, 0.17)
2002	-0.35 (-0.83, 0.16)	-0.10 (-0.51, 0.30)
2003	-0.44 (-0.89, 0.05)	-0.17 (-0.56, 0.23)
2004	0.30 (-0.14, 0.78)	-0.06 (-0.46, 0.34)
2005	0.51 (0.07, 1.00)	0.11 (-0.31, 0.53)
2006	0.90 (0.46, 1.38)	-0.94 (-1.36, -0.50)
2007	0.56 (-0.15, 1.27)	0.23 (-0.18, 0.64)
<b>SES*Visit year, reference group: Low SES*1999 or Low SES*2000</b>		
Mid SES*2000	-0.49 (-1.38, 0.36)	0.46 (-0.29, 1.25)
Mid SES*2001	-0.45 (-1.28, 0.38)	0.48 (-0.26, 1.25)
Mid SES*2002	-0.20 (-1.01, 0.57)	0.10 (-0.62, 0.85)
Mid SES*2003	-0.53 (-1.32, 0.23)	-0.05 (-0.77, 0.71)
Mid SES*2004	-0.66 (-1.43, 0.08)	0.24 (-0.47, 0.98)
Mid SES*2005	-0.69 (-1.47, 0.04)	0.31 (-0.41, 1.05)
Mid SES*2006	-0.69 (-1.46, 0.03)	0.54 (-0.16, 1.29)
Mid SES*2007	-0.63 (-1.60, 0.35)	0.37 (-0.31, 1.10)
High SES*2000	-0.67 (-1.50, 0.11)	0.36 (-0.32, 1.11)
High SES*2001	-0.52 (-1.29, 0.21)	0.48 (-0.18, 1.20)
High SES*2002	-0.18 (-0.93, 0.50)	0.39 (-0.23, 1.10)
High SES*2003	-0.57 (-1.31, 0.10)	0.35 (-0.26, 1.04)
High SES*2004	-0.39 (-1.10, 0.25)	0.35 (-0.27, 1.05)
High SES*2005	-0.46 (-1.18, 0.18)	0.62 (-0.01, 1.32)
High SES*2006	-0.51 (-1.23, 0.13)	0.45 (-0.18, 1.15)
High SES*2007	-0.45 (-1.78, 0.82)	0.22 (-0.38, 0.89)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>		

Age, reference group: <60 years		
Age: 60-74 years	0.01 (-0.06, 0.09)	0.00 (-0.11, 0.11)
Age: 75+ years	0.38 (0.28, 0.47)	-0.11 (-0.25, 0.03)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9	-0.01 (-0.09, 0.06)	0.47 (0.34, 0.60)
Duration 10+	0.18 (0.09, 0.27)	1.60 (1.47, 1.73)
Ethnicity, reference group: White		
South Asian	0.22 (0.05, 0.38)	-0.16 (-0.39, 0.08)
Other Ethnicity	0.30 (-0.07, 0.67)	0.53 (0.08, 0.95)
Male	0.24 (0.18, 0.31)	0.25 (0.16, 0.34)
Smoking status, reference group: non smoker		
Smoker	0.26 (0.17, 0.36)	-0.13 (-0.27, 0.00)
Ex-smoker	0.07 (0.00, 0.14)	-0.12 (-0.22, -0.03)
BMI status, reference group: under & normal weight		
Overweight	-0.01 (-0.11, 0.08)	-0.09 (-0.22, 0.04)
Obese	0.06 (-0.04, 0.16)	-0.10 (-0.23, 0.03)
HbA1c	0.18 (0.11, 0.24)	0.05 (0.02, 0.08)
Hypertensive	0.03 (0.00, 0.06)	0.33 (0.25, 0.42)
Cholesterol	0.09 (0.06, 0.11)	-0.02 (-0.06, 0.03)
eGFR		-0.01 (-0.01, -0.01)
<b>Interventions</b>		
Quality of care level, reference group: Low quality OR Mid quality		
Mid quality	-0.13 (-0.54, 0.25)	
High quality	-0.25 (-0.66, 0.14)	-0.07 (-0.17, 0.04)
Diabetes treatment, reference group diet alone		
Sulphonylureas/metformin only	0.14 (0.04, 0.24)	0.40 (0.24, 0.56)
Combination, no insulin	0.02 (-0.10, 0.13)	0.70 (0.52, 0.87)
Insulin only	0.18 (0.04, 0.32)	1.05 (0.86, 1.23)
Combination, with insulin	0.20 (0.10, 0.30)	1.16 (0.96, 1.36)
Blood pressure treatment, reference group: No treatments		
ACE inhibitors only	0.36 (0.25, 0.47)	0.32 (0.17, 0.46)
ACE & other(s)	0.52 (0.43, 0.61)	0.22 (0.10, 0.35)
Other combination	0.32 (0.22, 0.41)	0.13 (0.00, 0.26)
Aspirin	0.08 (0.01, 0.14)	0.05 (-0.04, 0.15)
Lipid therapy	-0.05 (-0.12, 0.02)	-0.06 (-0.16, 0.04)
Middlesbrough PCT	0.58 (0.29, 0.86)	-0.05 (-0.22, 0.13)
Shared care	-0.92 (-1.01, -0.83)	0.52 (0.42, 0.63)
<b>Cons</b>	-2.73 (-3.43, -2.11)	-2.83 (-3.48, -2.19)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.21 (0.13, 0.34)	0.05 (0.03, 0.10)
<b>Patient level</b>	0.01 (0.00, 0.03)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	25480.02	14536.53
<b>Available cases (N)</b>	23,304	18,665

Comparing the five models in Table 10 and Table 11 there were some statistically significant results indicating improvements in the incidences of stroke or TIA, PVD and, in particular, ICD over the study period. In contrast, in 2005 and 2006, rates of microalbuminuria were significantly worse than 1999. These results support the graphical analyses displayed in chapter five. Increasing age, in contrast to the intermediate outcomes, was a statistically significant predictor of higher rates of all long-term complications with the exception of retinopathy. Interestingly, increased duration of diabetes was associated with lower incidences of ICD and stroke or TIA but higher incidences of PVD. South Asian patients had significantly lower

incidences of PVD but higher of microalbuminuria compared to white patients. Interestingly, the relationship between ethnicity and microalbuminuria was only significant following the introduction of intervention data suggesting a potential interaction effect not included in the model. Patients of an 'other' ethnicity had significantly higher rates of retinopathy compared to white patients. There were no other significant relationships between ethnicity and rates of vascular disease modelled here. The results show that non-smokers had lower rates of complications in comparison to smokers and/or ex-smokers, but these findings were not consistent.

When looking at the health status data, increased HbA1c was significantly associated with higher rates of ICD, microalbuminuria and retinopathy. Being hypertensive was significantly associated with lower incidences of ICD but higher rates of retinopathy. Increased cholesterol was significantly associated with lower incidences of ICD and stroke or TIA but higher rates of retinopathy. There was a small but significant association with increased eGFR with lower rates of retinopathy. Interestingly, BMI status was not significantly associated with any of outcomes. However, patients classified as obese were significantly more likely to have had ICD and microalbuminuria prior to the intervention data being included in the final models, suggesting that diabetes care mediates this relationship.

The results of the diabetes interventions indicators showed that increased quality of care was a significant predictor of lower incidences of ICD but was not associated with any other long-term complication. Where diabetes treatments were significant, the results showed they were associated with lower incidences of ICD and stroke or TIA incidences but were higher rates of microalbuminuria and retinopathy, compared to being prescribed no treatments. Where prescriptions for BP treatments, aspirin and lipid therapies were significant, they were associated with higher rates of long-term complications. Receiving shared care was significantly associated with all long-term complications, with the exception of microalbuminuria where it was associated with lower rates. Interestingly, being managed in Middlesbrough PCT was a significant predictor of lower incidences of ICD and higher rates of microalbuminuria, compared to being managed in Redcar & Cleveland PCT.

The Bayesian DIC statistics from the stepwise models indicated that model fit increasingly improved with the inclusion of each set of variables. The ICC of practice level in the null model indicated that 5.74% and 6.44% of the variance of rates of PVD and microalbuminuria respectively were explained by patients' general practice. Approximately 2% or less of the variance were explained by general practice with the other outcomes.

## Principle findings

The results in this chapter showed some evidence of SES inequalities in intermediate outcomes and long-term complications over time. However, the results showed that this did not always favour the same SES group. In particular, high SES patients were significantly more likely to have lower HbA1c but in contrast, they were significantly more likely to have higher cholesterol over the study period. In contrast, there were no statistically significant interactions between SES and visit year with any long-term complication. There was one exception, however, which indicated mid SES patients were more likely to have higher incidences of ICD than low SES patients.

## Chapter 7: Are there socio-economic inequalities in interventions associated with type 2 diabetes over time?

This chapter describes the multilevel models that were fitted to examine whether there were socio-economic inequalities in the rate of type 2 diabetes interventions reported by SES over the study period once other explanatory variables and clustering of data within individuals and general practices have been taken into account.

### Timeliness of diagnosis

Table 12 shows the results for the saturated linear regression multilevel model that examined timeliness of diagnosis by SES. There were 3,071 available cases were modelled.

Table 12: Saturated linear regression multilevel model examining timeliness of diagnosis with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Timeliness of diagnosis
<b>Social-economic status</b>	
Social-economic status, reference group: Low	
Mid	0.69 (-0.20, 1.55)
High	0.27 (-0.49, 1.03)
Visit year, reference group: 1999	
2000	-0.24 (-0.90, 0.39)
2001	-0.76 (-1.37, -0.18)
2002	-0.12 (-0.68, 0.42)
2003	-0.42 (-0.95, 0.12)
2004	-0.42 (-0.97, 0.11)
2005	-0.59 (-1.14, -0.05)
2006	-0.99 (-1.54, -0.44)
2007	-1.14 (-1.73, -0.57)
SES*Visit year, reference group: Low SES*1999	
Mid SES*2000	-0.40 (-1.50, 0.71)
Mid SES*2001	0.25 (-0.80, 1.31)
Mid SES*2002	-1.11 (-2.08, -0.12)
Mid SES*2003	-0.64 (-1.60, 0.32)
Mid SES*2004	-0.91 (-1.84, 0.04)
Mid SES*2005	-0.41 (-1.35, 0.54)
Mid SES*2006	-0.37 (-1.33, 0.60)
Mid SES*2007	-0.45 (-1.41, 0.55)
High SES*2000	-0.76 (-1.75, 0.23)

High SES*2001	-0.03 (-0.96, 0.90)
High SES*2002	-0.70 (-1.53, 0.16)
High SES*2003	-0.50 (-1.33, 0.34)
High SES*2004	-0.33 (-1.14, 0.49)
High SES*2005	-0.44 (-1.28, 0.39)
High SES*2006	-0.43 (-1.26, 0.42)
High SES*2007	-0.24 (-1.08, 0.61)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>	
Age at diagnosis, reference group: <60	
60-74	-0.08 (-0.22, 0.07)
75+	-0.12 (-0.33, 0.08)
Ethnicity, reference group: white	
South Asian	<b>0.47 (0.17, 0.77)</b>
Other Ethnicity	0.32 (-0.32, 0.98)
Male	0.05 (-0.08, 0.17)
Smoking status, reference group: non-smoker	
Smoker	<b>0.21 (0.05, 0.36)</b>
Ex-smoker	0.03 (-0.11, 0.16)
Obesity status, reference group: Under and normal weight	
Overweight	0.16 (-0.03, 0.35)
Obese	0.05 (-0.14, 0.24)
Hypertensive	0.04 (-0.08, 0.17)
Cholesterol	<b>0.16 (0.12, 0.20)</b>
Creatinine > 300	0.53 (-1.78, 2.75)
eGFR	0.00 (0.00, 0.01)
Ischaemic Cardiac	0.09 (-0.09, 0.26)
Stroke or TIA	-0.10 (-0.37, 0.18)
PVD	-0.01 (-0.41, 0.39)
<b>Interventions</b>	
Care level, reference group: <7	
Care level: 7	<b>-0.20 (-0.34, -0.05)</b>
Care level: 8	-0.11 (-0.29, 0.08)
Diabetes treatment, reference group diet alone	
Metformin/sulphonylureas only	<b>1.01 (0.87, 1.15)</b>
Combination, no insulin	<b>1.03 (0.82, 1.25)</b>
Insulin only	<b>2.00 (1.65, 2.35)</b>
Combination with insulin	<b>1.64 (1.44, 1.84)</b>
BP treatment, reference group no treatments	
ACE inhibitors only	<b>-0.32 (-0.54, -0.10)</b>
ACE & other(s)	<b>-0.25 (-0.42, -0.07)</b>
Other BP	-0.13 (-0.29, 0.03)
Aspirin	0.04 (-0.11, 0.19)
Lipid therapy	-0.11 (-0.24, 0.03)
Shared care	<b>0.20 (0.01, 0.38)</b>
Middlesbrough PCT	0.14 (-0.09, 0.38)
<b>Cons</b>	<b>6.79 (6.06, 7.50)</b>
<b>Practice level</b>	0.10 (0.05, 0.18)
<b>Patient level</b>	2.57 (2.45, 2.71)
<b>Bayesian DIC</b>	11700.65

N = 3,071

The results in Table 12 show that there were no statistically significant differences by SES in the timeliness of diagnosis as measured by HbA1c at time of diagnosis. This was consistent over each step of analysis (Table 63, Appendix G). However, there was some evidence of statistically

significant differences in timeliness of diagnosis over time, with mid SES patients more likely to have a lower HbA1c at diagnosis in 2002 compared to low SES in 1999. There was more evidence in earlier steps, however, these become non-significant following the introduction of other covariates, particularly intervention data. This suggests that there were differences in timeliness of diagnosis, however, diabetes care initiated during the first year mediates the effect of these differences.

Prior to intervention data being added to the model, age had a significant negative association with HbA1c at diagnosis, with the older patients the more likely to have a more favourable HbA1c at diagnosis. However, being South Asian, a smoker and increasing cholesterol were significantly associated with a greater HbA1c at diagnosis. Once intervention data were added, age was no longer significant. Mid quality of care compared to low quality, and being treated with ACEIs either alone or in combination with other BP treatments had a significant negative association with HbA1c at diagnosis, suggesting a relationship with earlier diagnosis. In contrast, being treated with any diabetes treatment and receiving shared care were significantly associated with a higher HbA1c at diagnosis, suggesting a later diagnosis results in the initiation of treatments and specialist care within a year.

The Bayesian DIC statistics indicated that more variance was explained as each set of variables were added to the model, improving model fit. In the null model, 3.57% of variation in timeliness of diagnosis was accounted for by the practice the patient was registered with, as measured by ICC (Table 63, Appendix G).

## Quality of care

Table 13 shows the results for the saturated linear regression multilevel model that examined quality of care by SES. There were 33,115 available cases for this model. The results show that there was a statistically significant difference in the quality of care, with high SES associated with lower quality. However, the interaction effect resulted in a statistically significant association between high SES and time, suggesting that high SES patients have received greater quality of care compared to low SES patients from 2002 to 2007 compared to 1999. These patterns remained consistent across each step of the analyses suggesting that the relationship occurs regardless of a patients' other characteristics and health care needs. The significant positive association between visit year and quality of care suggests that quality of care has

increased over time, with the exception of 2007. This reflects the results from the graphical analyses.

Prior to the interventions being added to the model, being aged 75 years and over, South Asian, a smoker and increasing cholesterol had a significant negative association with quality of care. Duration categories of more than 3 years had a significant positive association with quality of care. Once intervention data were added having diabetes 10 years or more was no longer significant. Interestingly, being aged 60-74 become significant and indicated that this age group were likely to receive greater quality of care compared to those aged under 60 years old.

Other intervention data were added to the model to determine whether these were related to the level of quality of care. The results show that being treated with insulin and other diabetes treatments, ACEI solely and shared care were also significant predictors of increased quality of care. The latter relationship suggests that place of care was an important determinant of quality care. However, the ICC at practice level variance in the null model was 6%, indicating that patients' general practice makes only a small contribution to the level of care they receive. The Bayesian DIC indicates that more variance was explained as each set of variables were added to the model, improving model fit.

Table 13: Saturated linear regression multilevel model examining quality of care with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Quality of care
<b>Socio-economic status</b>	
Social-economic status, reference group: Low	
Mid	-0.04 (-0.16, 0.09)
High	-0.16 (-0.28, -0.05)
Visit year, reference group: 1999	
2000	0.05 (-0.03, 0.14)
2001	0.11 (0.02, 0.19)
2002	0.06 (-0.02, 0.14)
2003	0.06 (-0.01, 0.14)
2004	0.15 (0.07, 0.23)
2005	0.02 (-0.06, 0.10)
2006	0.27 (0.19, 0.34)
2007	-0.40 (-0.47, -0.32)
SES*Visit year, reference group: Low SES*1999	
Mid SES*2000	-0.03 (-0.18, 0.12)
Mid SES*2001	-0.01 (-0.16, 0.13)
Mid SES*2002	0.11 (-0.03, 0.24)
Mid SES*2003	0.07 (-0.07, 0.21)
Mid SES*2004	-0.01 (-0.14, 0.13)
Mid SES*2005	0.03 (-0.10, 0.17)
Mid SES*2006	0.06 (-0.07, 0.20)
Mid SES*2007	0.08 (-0.05, 0.22)

High SES*2000	0.09 (-0.05, 0.22)
High SES*2001	0.11 (-0.02, 0.24)
High SES*2002	0.21 (0.09, 0.33)
High SES*2003	0.17 (0.05, 0.29)
High SES*2004	0.18 (0.06, 0.30)
High SES*2005	0.19 (0.07, 0.31)
High SES*2006	0.21 (0.09, 0.32)
High SES*2007	0.16 (0.03, 0.28)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>	
Age, reference group: <60	
60-74	0.04 (0.02, 0.06)
75+	-0.01 (-0.03, 0.02)
Duration of diabetes, reference group: 0-3 years	
Duration: 4-9 years	0.06 (0.04, 0.07)
Duration 10+ years	0.01 (-0.01, 0.03)
Ethnicity, reference group: white	
South Asian	-0.08 (-0.12, -0.04)
Other Ethnicity	-0.08 (-0.16, 0.00)
Male	-0.01 (-0.03, 0.00)
Smoking status, reference group: non-smoker	
Smoker	-0.06 (-0.09, -0.04)
Ex-smoker	0.02 (0.00, 0.04)
Obesity status, reference group: Under and normal weight	
Overweight	0.03 (0.01, 0.05)
Obese	0.00 (-0.02, 0.02)
Hypertensive	-0.03 (-0.04, -0.01)
HbA1c	-0.01 (-0.02, -0.01)
Cholesterol	-0.02 (-0.03, -0.02)
Creatinine > 300	-0.07 (-0.19, 0.06)
eGFR	0.00 (0.00, 0.00)
Ischaemic Cardiac	-0.03 (-0.05, -0.01)
Stroke or TIA	-0.01 (-0.03, 0.02)
PVD	0.02 (-0.01, 0.04)
<b>Interventions</b>	
Diabetes treatment, reference group diet alone	
Sulphonylureas / metformin only	0.05 (0.03, 0.08)
Diab. comb. no insulin	0.02 (-0.01, 0.04)
Insulin only	0.02 (-0.01, 0.05)
Insulin & other diab. treatments	0.04 (0.02, 0.07)
BP treatment, reference group no treatments	
ACE inhibitors only	0.04 (0.01, 0.06)
ACE & other(s)	0.02 (0.00, 0.05)
Other BP	0.02 (0.00, 0.04)
Aspirin	0.00 (-0.02, 0.01)
Lipid therapy	0.02 (0.00, 0.03)
Shared care	0.27 (0.25, 0.29)
Middlesbrough PCT	-0.07 (-0.18, 0.03)
<b>Cons</b>	7.06 (6.93, 7.19)
<b>Practice level</b>	0.03 (0.02, 0.05)
<b>Patient level</b>	0.00 (0.00, 0.01)
<b>Visit year level</b>	0.42 (0.41, 0.43)
<b>Bayesian DIC</b>	65285.29
<b>Available cases (N)</b>	33,115

## Diabetes treatments

Table 14 shows the results for the saturated logistic regression multilevel model that examined diabetes treatments by SES. There were 36,161 available cases available for each set of analyses.

Table 14: Saturated logistic regression multilevel model examining diabetes treatments with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Diet alone	Metformin or sulphonylureas	Combination, no insulin	Insulin only	Insulin & others
<b>Social-economic status</b>					
Social-economic status, reference group: Low					
Mid	-0.31 (-0.65, 0.03)	0.27 (-0.04, 0.59)	0.24 (-0.06, 0.53)	-0.26 (-0.61, 0.09)	-0.10 (-0.42, 0.20)
High	-0.05 (-0.34, 0.25)	-0.49 (-0.87, -0.14)	0.00 (-0.28, 0.27)	-0.11 (-0.45, 0.24)	0.22 (-0.06, 0.50)
Visit year, reference group: 1999					
2000	-0.45 (-0.71, -0.17)	0.11 (-0.14, 0.37)	-0.25 (-0.51, 0.00)	0.06 (-0.22, 0.33)	0.29 (0.06, 0.52)
2001	-0.69 (-0.96, -0.43)	0.10 (-0.15, 0.34)	-0.39 (-0.64, -0.14)	0.11 (-0.17, 0.39)	0.47 (0.24, 0.70)
2002	-0.62 (-0.87, -0.37)	0.17 (-0.05, 0.39)	-0.45 (-0.68, -0.23)	-0.23 (-0.49, 0.03)	0.64 (0.44, 0.84)
2003	-0.74 (-0.97, -0.51)	0.17 (-0.04, 0.39)	-0.72 (-0.94, -0.49)	-0.21 (-0.46, 0.04)	0.80 (0.61, 1.00)
2004	-0.81 (-1.04, -0.58)	0.30 (0.09, 0.51)	-1.10 (-1.32, -0.88)	-0.14 (-0.39, 0.11)	0.96 (0.76, 1.15)
2005	-0.95 (-1.18, -0.72)	0.31 (0.10, 0.52)	-1.17 (-1.40, -0.94)	-0.12 (-0.38, 0.13)	1.04 (0.84, 1.24)
2006	-1.15 (-1.37, -0.92)	0.34 (0.14, 0.55)	-1.25 (-1.48, -1.02)	-0.05 (-0.31, 0.21)	1.03 (0.84, 1.23)
2007	-1.21 (-1.45, -0.98)	0.53 (0.32, 0.75)	-1.48 (-1.73, -1.24)	-0.03 (-0.29, 0.24)	1.08 (0.88, 1.27)
SES*Visit year, reference group: Low SES*1999					
Mid SES*2000	0.10 (-0.38, 0.57)	-0.28 (-0.73, 0.16)	0.03 (-0.37, 0.44)	-0.01 (-0.50, 0.46)	0.16 (-0.24, 0.56)
Mid SES*2001	0.27 (-0.17, 0.72)	-0.52 (-0.95, -0.08)	-0.11 (-0.51, 0.29)	-0.11 (-0.59, 0.37)	0.36 (-0.02, 0.76)
Mid SES*2002	0.17 (-0.26, 0.57)	-0.34 (-0.73, 0.05)	-0.27 (-0.64, 0.12)	0.46 (0.00, 0.90)	0.12 (-0.23, 0.50)
Mid SES*2003	0.37 (-0.04, 0.77)	-0.31 (-0.68, 0.06)	-0.19 (-0.55, 0.19)	0.40 (-0.04, 0.84)	0.04 (-0.29, 0.39)
Mid SES*2004	0.29 (-0.11, 0.67)	-0.25 (-0.60, 0.10)	-0.06 (-0.42, 0.31)	0.19 (-0.25, 0.62)	0.00 (-0.33, 0.35)
Mid SES*2005	0.54 (0.15, 0.94)	-0.34 (-0.70, 0.01)	-0.33 (-0.71, 0.05)	0.36 (-0.06, 0.80)	0.01 (-0.33, 0.36)
Mid SES*2006	0.39 (0.00, 0.76)	-0.40 (-0.76, -0.05)	-0.25 (-0.62, 0.12)	0.37 (-0.06, 0.80)	0.14 (-0.20, 0.48)
Mid SES*2007	0.47 (0.08, 0.87)	-0.44 (-0.80, -0.09)	-0.28 (-0.68, 0.13)	0.40 (-0.04, 0.84)	0.13 (-0.20, 0.48)
High SES*2000	0.10 (-0.33, 0.53)	-0.09 (-0.59, 0.40)	0.08 (-0.30, 0.47)	0.34 (-0.12, 0.80)	-0.24 (-0.61, 0.13)
High SES*2001	0.09 (-0.32, 0.49)	0.22 (-0.23, 0.68)	0.19 (-0.18, 0.57)	0.07 (-0.38, 0.52)	-0.25 (-0.61, 0.11)
High SES*2002	0.01 (-0.37, 0.38)	0.23 (-0.19, 0.66)	0.17 (-0.17, 0.53)	0.38 (-0.04, 0.80)	-0.30 (-0.64, 0.03)

High SES*2003	0.05 (-0.31, 0.40)	0.33 (-0.06, 0.76)	0.22 (-0.12, 0.56)	0.38 (-0.03, 0.79)	-0.38 (-0.71, -0.06)
High SES*2004	0.05 (-0.30, 0.39)	0.31 (-0.08, 0.72)	0.16 (-0.18, 0.50)	0.31 (-0.10, 0.72)	-0.30 (-0.60, 0.02)
High SES*2005	0.15 (-0.20, 0.48)	0.45 (0.07, 0.87)	0.05 (-0.29, 0.40)	0.23 (-0.19, 0.65)	-0.33 (-0.64, -0.01)
High SES*2006	0.14 (-0.20, 0.47)	0.43 (0.05, 0.84)	0.02 (-0.31, 0.37)	0.46 (0.05, 0.86)	-0.35 (-0.66, -0.04)
High SES*2007	0.25 (-0.10, 0.59)	0.41 (0.03, 0.82)	0.04 (-0.32, 0.42)	0.32 (-0.11, 0.73)	-0.36 (-0.67, -0.04)

### Socio-demographic, anthropometric, lifestyle and health covariates

Age, reference group: <60 years					
Age: 60-74 years	0.19 (0.11, 0.27)	-0.03 (-0.09, 0.04)	0.21 (0.12, 0.30)	-0.46 (-0.55, -0.36)	0.00 (-0.06, 0.06)
Age: 75+ years	0.44 (0.34, 0.54)	-0.01 (-0.10, 0.08)	0.52 (0.41, 0.63)	-0.63 (-0.76, -0.50)	-0.31 (-0.39, -0.22)
Duration of diabetes, reference group: 0-3 years					
Duration: 4-9 yrs	-1.07 (-1.14, -1.00)	-0.42 (-0.48, -0.36)	0.14 (0.07, 0.22)	0.57 (0.46, 0.68)	1.14 (1.08, 1.20)
Duration 10+ yrs	-1.64 (-1.74, -1.55)	-1.20 (-1.28, -1.11)	-0.27 (-0.36, -0.18)	1.42 (1.31, 1.53)	1.43 (1.36, 1.50)
Ethnicity, reference group: white					
South Asian	0.13 (-0.06, 0.32)	0.07 (-0.08, 0.21)	0.12 (-0.06, 0.29)	-0.58 (-0.77, -0.39)	0.08 (-0.05, 0.21)
Other Ethnicity	0.10 (-0.33, 0.51)	-0.37 (-0.73, -0.04)	-0.11 (-0.56, 0.31)	0.55 (0.19, 0.89)	-0.18 (-0.45, 0.09)
Male	0.28 (0.22, 0.35)	-0.18 (-0.24, -0.12)	0.34 (0.26, 0.41)	-0.07 (-0.15, 0.01)	-0.17 (-0.23, -0.12)
Smoking status, reference group: non-smoker					
Smoker	-0.07 (-0.16, 0.03)	0.06 (-0.02, 0.14)	0.07 (-0.03, 0.17)	0.16 (0.05, 0.27)	-0.05 (-0.13, 0.02)
Ex-smoker	-0.09 (-0.16, -0.02)	0.03 (-0.03, 0.09)	0.02 (-0.06, 0.09)	0.00 (-0.08, 0.08)	0.05 (-0.01, 0.10)
BMI status, reference group: under and normal weight					
Overweight	-0.23 (-0.32, -0.14)	0.46 (0.36, 0.56)	-0.25 (-0.34, -0.17)	-0.40 (-0.51, -0.30)	0.32 (0.24, 0.40)
Obese	-0.43 (-0.52, -0.34)	0.63 (0.54, 0.73)	-0.70 (-0.79, -0.61)	-0.87 (-0.97, -0.76)	0.72 (0.65, 0.81)
HbA1c	-0.82 (-0.85, -0.79)	-0.07 (-0.09, -0.06)	0.01 (-0.01, 0.04)	0.24 (0.22, 0.26)	0.27 (0.25, 0.28)
Hypertensive	0.03 (-0.04, 0.09)	-0.02 (-0.08, 0.04)	0.02 (-0.05, 0.09)	-0.13 (-0.21, -0.05)	0.04 (-0.01, 0.09)
Cholesterol	0.24 (0.21, 0.27)	-0.01 (-0.04, 0.02)	0.00 (-0.03, 0.03)	0.02 (-0.01, 0.05)	-0.18 (-0.20, -0.15)
Creatinine > 300	-0.18 (-0.75, 0.34)	-3.11 (-6.17, -1.20)	0.25 (-0.21, 0.69)	0.46 (-0.01, 0.93)	-1.15 (-1.79, -0.56)
eGFR	0.00 (-0.01, 0.00)	0.01 (0.01, 0.01)	-0.01 (-0.02, -0.01)	-0.02 (-0.03, -0.02)	0.01 (0.01, 0.01)
Ischaemic Cardiac Stroke or TIA	0.06 (-0.01, 0.13)	-0.04 (-0.11, 0.02)	-0.01 (-0.08, 0.06)	0.18 (0.10, 0.27)	-0.11 (-0.17, -0.06)
PVD	-0.08 (-0.19, 0.03)	-0.07 (-0.17, 0.03)	0.06 (-0.04, 0.17)	0.15 (0.04, 0.26)	-0.04 (-0.12, 0.04)
	-0.33 (-0.46, -0.19)	-0.06 (-0.18, 0.05)	-0.09 (-0.22, 0.03)	0.38 (0.27, 0.50)	-0.09 (-0.18, 0.00)

### Interventions

Care level, reference group: <7					
Care level: 7	-0.15 (-0.22, -0.07)	0.08 (0.00, 0.15)	0.06 (-0.03, 0.14)	-0.07 (-0.17, 0.03)	0.10 (0.03, 0.16)
Care level: 8	-0.14 (-0.23, -0.05)	0.15 (0.06, 0.23)	-0.05 (-0.15, 0.05)	-0.04 (-0.15, 0.07)	0.12 (0.05, 0.20)
Shared care	-1.33 (-1.43, -1.23)	-0.54 (-0.61, -0.46)	-0.62 (-0.71, -0.53)	2.12 (2.04, 2.21)	0.09 (0.03, 0.15)
M'brough PCT	0.01 (-0.34, 0.36)	0.14 (-0.05, 0.32)	-0.01 (-0.19, 0.18)	-0.13 (-0.35, 0.08)	-0.08 (-0.28, 0.10)
Cons	5.43	-1.73	-0.22	-3.42	-4.76

	(5.02, 5.88)	(-2.08, -1.40)	(-0.65, 0.19)	(-3.88, -2.97)	(-5.12, -4.41)
<b>Variance estimate at:</b>					
<b>Practice level</b>	0.32 (0.20, 0.51)	0.09 (0.05, 0.14)	0.07 (0.04, 0.13)	0.11 (0.06, 0.18)	0.09 (0.05, 0.14)
<b>Patient level</b>	0.01 (0.00, 0.03)	0.00 (0.00, 0.02)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.01 (0.00, 0.02)
<b>Bayesian DIC</b>	27148.92	32952.79	25654.38	19561.96	40361.07

*N* = 36,161

With the exception of being treated by a combination of diabetes treatments with no insulin, Table 14 shows that there were statistically significant differences in diabetes treatment regimens by SES over time. Mid SES patients were more likely to be treated by diet alone and less likely to be prescribed a mono-therapy of metformin or sulphonylureas compared to low SES patients in particular years. In addition, high SES patients were significantly more likely to be prescribed with a mono-therapy of metformin or sulphonylureas or insulin only and less likely to be treated with a combination of insulin with other diabetes treatment(s) compared to low SES patients in particular years. These relationships were not explained by demographic, anthropometric, lifestyle and health care needs.

The final saturated model which examined differences in patients having their blood glucose levels managed by diet alone shows that there were no significant differences by SES overall. However, prior to adding intervention data into the model, there were statistically significant results indicating that mid SES were more likely to be treated by diet alone, both overall and over time (Table 65, Appendix G). The stepwise models in Table 66 in Appendix G show that high SES patients were significantly more likely to be prescribed a mono-therapy of metformin or sulphonylureas overall and that this relationship was not explained by other covariates. In contrast, there were no statistically significant differences by SES in being prescribed any of the remaining treatment regimens in any step of the analyses (Table 67, Table 68 and Table 69, Appendix G).

Increasing age was significantly associated with being more likely to be treated with diet alone and combination of diabetes treatments with no insulin and being less likely to be prescribed insulin only and in combination with other diabetes treatments. The direction of the relationship between treatment regimens and duration of diabetes follows expectations: with increased duration associated with being less likely to be treated with diet alone and mono-therapies of metformin or sulphonylureas and more likely to be treated with insulin either solely or in combination. There were significant differences between ethnicity and sex and treatment regimens which were not easily explained.

Being a smoker and ex-smoker were significantly associated with being less likely to be treated by diet alone. Being a smoker was also significantly associated with being more likely to be treated with insulin only. Increased BMI was also a significant predictor of diabetes treatment regimens but not in a consistent manner as was demonstrated with duration of diabetes. As expected, increased HbA1c had a significant negative association with being treated by diet alone and a mono-therapy of metformin or sulphonylureas and positive association with being treated with insulin either alone or in combination with other diabetes treatment(s). Other indicators of patients' health status showed some significant relationships with diabetes treatment outcomes but not in a consistent way.

The ICC of the null models indicate that the general practice at which patients were registered with explained more of the variation in being treated by diet alone than other treatment regimens with 10.51% of the variation explained at this level. 4.26% of the variation in being prescribed insulin only and approximately 2% of the variation in prescription for combinations of diabetes treatments without insulin and for diabetes treatments with insulin were explained at this level. In all sets of analyses the Bayesian statistics indicate improved model fit with the introduction of set of variables.

## Blood pressure treatments

Table 15: Saturated logistic regression multilevel model examining BP treatments with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	No BP	ACEI only	ACEI comb.	Other comb.
<b>Social-economic status</b>				
<b>Social-economic status, reference group: Low</b>				
Mid	0.11 (-0.18, 0.42)	0.30 (-0.14, 0.74)	0.00 (-0.37, 0.35)	-0.23 (-0.52, 0.08)
High	<b>0.35 (0.06, 0.63)</b>	0.33 (-0.07, 0.73)	-0.36 (-0.72, 0.00)	<b>-0.31 (-0.62, -0.01)</b>
Visit year, reference group: 1999				
2000	-0.21 (-0.45, 0.03)	<b>0.40 (0.05, 0.75)</b>	<b>0.30 (0.03, 0.58)</b>	-0.23 (-0.46, 0.00)
2001	<b>-0.27 (-0.50, -0.04)</b>	<b>0.46 (0.12, 0.80)</b>	<b>0.57 (0.30, 0.83)</b>	<b>-0.44 (-0.66, -0.21)</b>
2002	<b>-0.45 (-0.66, -0.24)</b>	<b>0.48 (0.16, 0.79)</b>	<b>0.78 (0.52, 1.03)</b>	<b>-0.51 (-0.72, -0.30)</b>
2003	<b>-0.73 (-0.94, -0.52)</b>	<b>0.44 (0.14, 0.75)</b>	<b>0.89 (0.65, 1.13)</b>	<b>-0.40 (-0.60, -0.19)</b>
2004	<b>-0.81 (-1.01, -0.60)</b>	<b>0.37 (0.07, 0.67)</b>	<b>0.96 (0.71, 1.20)</b>	<b>-0.38 (-0.58, -0.18)</b>
2005	<b>-0.99 (-1.20, -0.79)</b>	<b>0.50 (0.21, 0.81)</b>	<b>0.99 (0.75, 1.22)</b>	<b>-0.36 (-0.56, -0.15)</b>
2006	<b>-0.92 (-1.12, -0.71)</b>	<b>0.56 (0.27, 0.87)</b>	<b>0.98 (0.73, 1.22)</b>	<b>-0.43 (-0.63, -0.23)</b>
2007	<b>-1.11 (-1.33, -0.89)</b>	<b>0.60 (0.31, 0.91)</b>	<b>1.06 (0.82, 1.30)</b>	<b>-0.42 (-0.63, -0.21)</b>
SES*Visit year, reference group: Low SES*1999				
Mid SES*2000	-0.17 (-0.59, 0.24)	0.04 (-0.53, 0.62)	0.02 (-0.44, 0.50)	0.11 (-0.29, 0.50)
Mid SES*2001	-0.18 (-0.58, 0.21)	-0.02 (-0.56, 0.51)	0.00 (-0.42, 0.44)	0.16 (-0.23, 0.55)
Mid SES*2002	-0.18 (-0.55, 0.18)	-0.17 (-0.68, 0.33)	-0.03 (-0.42, 0.38)	0.25 (-0.10, 0.60)
Mid SES*2003	-0.06 (-0.42, 0.30)	-0.31 (-0.81, 0.19)	0.09 (-0.29, 0.49)	0.11 (-0.24, 0.46)
Mid SES*2004	0.02 (-0.33, 0.37)	-0.28 (-0.77, 0.21)	-0.06 (-0.45, 0.34)	0.18 (-0.16, 0.51)
Mid SES*2005	-0.09 (-0.44, 0.26)	-0.25 (-0.74, 0.24)	-0.01 (-0.40, 0.38)	0.20 (-0.14, 0.54)

Mid SES*2006	-0.14 (-0.48, 0.21)	-0.23 (-0.71, 0.25)	-0.01 (-0.40, 0.38)	0.22 (-0.12, 0.54)
Mid SES*2007	-0.18 (-0.56, 0.18)	-0.13 (-0.61, 0.35)	0.01 (-0.38, 0.41)	0.14 (-0.20, 0.47)
High SES*2000	0.00 (-0.38, 0.38)	-0.26 (-0.79, 0.28)	0.22 (-0.23, 0.68)	0.02 (-0.37, 0.42)
High SES*2001	-0.20 (-0.56, 0.17)	-0.36 (-0.87, 0.15)	0.07 (-0.35, 0.50)	<b>0.43 (0.05, 0.81)</b>
High SES*2002	<b>-0.42 (-0.76, -0.07)</b>	-0.2 (-0.66, 0.28)	0.18 (-0.21, 0.58)	<b>0.47 (0.12, 0.82)</b>
High SES*2003	<b>-0.39 (-0.72, -0.06)</b>	-0.21 (-0.67, 0.26)	0.34 (-0.05, 0.72)	0.32 (-0.02, 0.67)
High SES*2004	-0.29 (-0.61, 0.05)	-0.27 (-0.72, 0.19)	0.37 (-0.01, 0.76)	0.23 (-0.11, 0.57)
High SES*2005	-0.27 (-0.59, 0.06)	-0.34 (-0.79, 0.12)	0.26 (-0.12, 0.65)	<b>0.37 (0.03, 0.71)</b>
High SES*2006	-0.30 (-0.61, 0.03)	-0.32 (-0.76, 0.13)	0.21 (-0.17, 0.59)	<b>0.43 (0.11, 0.76)</b>
High SES*2007	-0.26 (-0.59, 0.08)	-0.23 (-0.67, 0.23)	0.22 (-0.16, 0.60)	<b>0.35 (0.01, 0.68)</b>
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years	<b>-0.43 (-0.50, -0.36)</b>	<b>-0.13 (-0.21, -0.05)</b>	<b>0.19 (0.13, 0.26)</b>	<b>0.39 (0.32, 0.45)</b>
Age: 75+ years	<b>-0.41 (-0.51, -0.31)</b>	<b>-0.37 (-0.49, -0.25)</b>	0.01 (-0.07, 0.09)	<b>0.59 (0.50, 0.67)</b>
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 yrs	<b>-0.24 (-0.31, -0.18)</b>	<b>0.32 (0.24, 0.40)</b>	<b>0.23 (0.17, 0.29)</b>	<b>-0.18 (-0.24, -0.13)</b>
Duration 10+ yrs	<b>-0.21 (-0.29, -0.13)</b>	<b>0.42 (0.33, 0.52)</b>	<b>0.39 (0.33, 0.46)</b>	<b>-0.44 (-0.51, -0.37)</b>
Ethnicity, reference: White				
South Asian	<b>0.44 (0.31, 0.58)</b>	-0.05 (-0.22, 0.12)	<b>-0.53 (-0.68, -0.39)</b>	-0.03 (-0.17, 0.10)
Other Ethnicity	0.20 (-0.09, 0.49)	<b>-1.01 (-1.55, -0.53)</b>	-0.10 (-0.41, 0.19)	0.24 (-0.06, 0.53)
Male	0.01 (-0.05, 0.07)	<b>0.25 (0.18, 0.33)</b>	<b>0.27 (0.22, 0.32)</b>	<b>-0.40 (-0.46, -0.35)</b>
Smoking status, reference: non-smoker				
Smoker	<b>0.25 (0.17, 0.33)</b>	0.01 (-0.10, 0.11)	<b>-0.11 (-0.19, -0.04)</b>	<b>-0.10 (-0.18, -0.03)</b>
Ex-smoker	-0.07 (-0.14, 0.00)	0.04 (-0.03, 0.12)	0.02 (-0.03, 0.08)	0.01 (-0.04, 0.07)
BMI status, reference: under and normal weight				
Overweight	<b>-0.37 (-0.45, -0.29)</b>	0.01 (-0.09, 0.11)	<b>0.29 (0.21, 0.37)</b>	0.03 (-0.05, 0.11)
Obese	<b>-0.87 (-0.95, -0.78)</b>	-0.10 (-0.20, 0.00)	<b>0.58 (0.50, 0.66)</b>	<b>0.17 (0.10, 0.25)</b>
sBP	<b>-0.02 (-0.02, -0.02)</b>	0.00 (0.00, 0.00)	<b>0.01 (0.01, 0.01)</b>	0.00 (0.00, 0.01)
dBp	0.01 (0.00, 0.01)	<b>0.01 (0.01, 0.02)</b>	<b>-0.01 (-0.01, -0.01)</b>	0.00 (0.00, 0.00)
HbA1c	<b>0.07 (0.05, 0.08)</b>	0.01 (-0.01, 0.04)	<b>-0.03 (-0.05, -0.01)</b>	<b>-0.04 (-0.06, -0.02)</b>
Cholesterol	<b>0.14 (0.12, 0.17)</b>	-0.02 (-0.05, 0.01)	<b>-0.11 (-0.14, -0.09)</b>	0.00 (-0.03, 0.02)
eGFR	<b>0.02 (0.02, 0.02)</b>	0.01 (0.00, 0.01)	<b>-0.01 (-0.02, -0.01)</b>	0.00 (-0.01, 0.00)
Ischaemic Cardiac	<b>-1.66 (-1.75, -1.58)</b>	<b>-0.93 (-1.03, -0.84)</b>	<b>0.81 (0.76, 0.86)</b>	<b>0.48 (0.43, 0.54)</b>
Stroke or TIA	<b>-0.49 (-0.61, -0.37)</b>	<b>0.2 (0.09, 0.32)</b>	<b>0.16 (0.08, 0.23)</b>	-0.04 (-0.12, 0.04)
PVD	<b>-0.32 (-0.46, -0.19)</b>	<b>0.29 (0.16, 0.41)</b>	0.02 (-0.07, 0.11)	-0.06 (-0.15, 0.03)
<b>Interventions</b>				
Care level, reference group: <7				
Care level: 7	-0.03 (-0.10, 0.05)	0.04 (-0.05, 0.13)	0.01 (-0.05, 0.08)	0.00 (-0.06, 0.07)
Care level: 8	<b>-0.14 (-0.22, -0.05)</b>	0.10 (0.00, 0.20)	0.04 (-0.03, 0.12)	0.02 (-0.05, 0.10)
M'brough PCT	0.08 (-0.12, 0.28)	0.01 (-0.21, 0.25)	-0.08 (-0.22, 0.07)	0.00 (-0.14, 0.15)
Shared care	0.05 (-0.03, 0.12)	0.01 (-0.07, 0.09)	0.03 (-0.03, 0.10)	<b>-0.16 (-0.23, -0.10)</b>
<b>Cons</b>	1.05 (0.55, 1.49)	-3.81 (-4.33, -3.28)	-1.96 (-2.39, -1.60)	-0.68 (-1.11, -0.28)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.10 (0.06, 0.16)	0.12 (0.07, 0.20)	0.04 (0.02, 0.08)	0.05 (0.03, 0.09)
<b>Patient level</b>	0.00 (0.00, 0.02)	0.01 (0.00, 0.03)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)
<b>Bayesian DIC</b>	31019.58	24581.11	39653.78	39748.14

N = 34,231

Table 15 shows the results for the saturated logistic regression multilevel model that examined BP treatments by SES. There were 34,231 available cases for each set of analyses. The results in **Table 15** show that there were some statistically significant differences by SES, both overall and over time, in being prescribed no BP treatments and combination of BP treatments without ACEI. High SES patients were more likely to receive no BP treatments overall but in 2002 and 2003 were less likely to be prescribed no BP treatments compared to low SES patients. In contrast, high SES patients were less likely to be prescribed another combination of BP treatments and yet, in a number of years, these patients were more likely to be prescribed this treatment regimen compared to low SES patients. There was evidence of inequalities in the

prescriptions of ACEI, alone and in combination with other BP treatments, by SES (Table 71 & Table 72, Appendix G) however, these relationships were no longer significant following the introduction of other covariates.

As expected the time trends which were noted in the four figures displayed in chapter five were supported by the statistically significant association between visit year and BP treatment regimens in the final models in Table 15. Age also followed expectations, with increasing age significantly associated with being less likely to be prescribed no BP treatments and ACEI only, and more likely to be prescribed a combination of BP treatments. The relationship between age and the two BP combination treatment regimens were expected as BP usually deteriorates with increased age [65]. The relationship between BMI categories and treatment outcomes did follow an expected pattern with obese patients less likely to be prescribed no BP treatments and more likely to receive combinations of BP treatments. Interestingly, duration of diabetes was significantly associated with all BP treatments regimens but did not follow a linear pattern. Increased duration was negatively associated with no BP treatments and other combinations of BP treatments and positively associated with ACEI alone and in combination. Like with diabetes treatments, there were significant differences by ethnicity and sex.

The variables measuring patients' health produced some unexpected results. Increased HbA1c, cholesterol and eGFR significantly associated with being more likely to be prescribed no BP treatments and less likely to be prescribed ACEI in combination with other BP treatments. However, the relationships between the outcome variables and history of vascular disease were more predictable. History of ICD, stroke or TIA and PVD were negatively associated with being prescribed no BP treatments, with the former also negatively associated with being prescribed ACEI only. There were significant positive associations between history of ICD and both BP treatment combinations, history of stroke or TIA and ACEI only and in combination with other BP treatments, and finally history of PVD with ACEI only.

Quality of care and receiving shared care were not associated with BP treatments which should be expected, however, there were exceptions. High quality of care was negatively associated with receiving no BP treatments compared to low quality of care. The Bayesian statistics indicated that there was improved model fit in each set of analyses with the introduction of other variables, but with the exception of when intervention data were added to the modelling of ACEI alone and in combination with BP treatments. This reflects the lack of statistical significance of these variables. Around 3% or less variation was explained at the level of general practice as measured by the ICC of the null models. This suggests that the general practice at

which patients are registered with played a very small role in determining their BP management.

## Antithrombotic and lipid profile treatments

Table 16 shows the results for the saturated logistic regression multilevel model that examined prescriptions for lipid therapies and aspirin by SES. There were 33,603 available cases for each set of analyses. The results show that there were no significant differences in the prescription of aspirin and lipid therapies by SES either overall or over time. These findings were consistent over each step of analyses (Table 74 and Table 75, Appendix G).

Both models indicated a statistically significant increase in prescriptions for these treatments over the study period, again supporting the graphical analyses in chapter five. The direction of the relationship between the treatment outcomes and the status of patients BMI, smoking and health were very similar in both final models. Smokers, ex-smokers, increased BMI, decreased cholesterol and history of ICD, stroke or TIA and PVD all had a significant positive association with both treatment outcomes. HbA1c also had a significant positive associated with prescription of lipid therapies but not with aspirin. Increased cholesterol was significantly associated with being less likely to be prescribed these treatments. This may be a reflection of patients cholesterol being reduced by the treatment as patients with high cholesterol are considered at high risk of CV complications [46].

Interestingly, increased quality of care had a significant positive association with patients being prescribed lipid therapies but not aspirin. In addition, shared care was significantly associated with both treatment outcomes but with different directions. These findings were not expected, especially the level of significant variables in the lipid therapies as there was a relatively high prescription rates with over 70% of patients receiving this treatment from 2005 onwards.

Bayesian DIC statistics indicated improvement in model fit when each set of variables were added to both models. Only between 2% and 3% of variation was explained at practice level.

Table 16: Saturated logistic regression multilevel model examining lipid therapies and aspirin with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Lipid therapies	Aspirin
<b>Social-economic status</b>		
<b>Social-economic status, reference group: Low</b>		
Mid	-0.05 (-0.52, 0.41)	-0.02 (-0.56, 0.45)
High	0.11 (-0.34, 0.52)	-0.19 (-0.67, 0.23)
Visit year, reference group: 1999		
2000	0.16 (-0.17, 0.47)	0.06 (-0.29, 0.38)
2001	0.39 (0.07, 0.69)	0.16 (-0.17, 0.48)
2002	0.89 (0.59, 1.17)	0.14 (-0.18, 0.44)
2003	1.56 (1.27, 1.84)	0.42 (0.11, 0.72)
2004	2.11 (1.81, 2.38)	0.55 (0.23, 0.84)
2005	2.38 (2.08, 2.66)	0.64 (0.32, 0.94)
2006	2.56 (2.27, 2.84)	0.72 (0.40, 1.02)
2007	2.70 (2.39, 2.98)	0.86 (0.53, 1.16)
SES*Visit year, reference group: Low SES*1999		
Mid SES*2000	0.15 (-0.40, 0.70)	0.10 (-0.45, 0.70)
Mid SES*2001	0.15 (-0.38, 0.67)	-0.08 (-0.62, 0.50)
Mid SES*2002	0.13 (-0.37, 0.63)	0.14 (-0.38, 0.69)
Mid SES*2003	0.03 (-0.46, 0.52)	-0.01 (-0.51, 0.54)
Mid SES*2004	0.12 (-0.38, 0.61)	0.04 (-0.45, 0.59)
Mid SES*2005	0.12 (-0.38, 0.61)	0.08 (-0.41, 0.63)
Mid SES*2006	-0.01 (-0.50, 0.49)	0.06 (-0.44, 0.61)
Mid SES*2007	0.11 (-0.40, 0.62)	0.11 (-0.39, 0.66)
High SES*2000	-0.08 (-0.58, 0.43)	-0.06 (-0.56, 0.48)
High SES*2001	-0.16 (-0.62, 0.32)	-0.28 (-0.75, 0.26)
High SES*2002	-0.12 (-0.56, 0.35)	0.04 (-0.42, 0.55)
High SES*2003	-0.28 (-0.71, 0.20)	0.02 (-0.43, 0.53)
High SES*2004	-0.08 (-0.51, 0.39)	0.16 (-0.27, 0.66)
High SES*2005	-0.13 (-0.57, 0.33)	0.12 (-0.31, 0.62)
High SES*2006	-0.08 (-0.51, 0.39)	0.21 (-0.23, 0.71)
High SES*2007	-0.19 (-0.63, 0.29)	0.08 (-0.36, 0.58)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	0.01 (-0.06, 0.08)	0.47 (0.40, 0.53)
Age: 75+ years	-0.67 (-0.75, -0.58)	0.46 (0.37, 0.54)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	0.04 (-0.02, 0.10)	0.07 (0.01, 0.13)
Duration 10+ years	-0.10 (-0.17, -0.03)	0.20 (0.14, 0.27)
Ethnicity, reference: White		
South Asian	-0.35 (-0.49, -0.22)	0.03 (-0.11, 0.16)
Other Ethnicity	-0.71 (-0.98, -0.42)	-0.01 (-0.31, 0.29)
Male	-0.33 (-0.39, -0.28)	0.27 (0.21, 0.32)
Smoking status, reference: non-smoker		
Smoker	0.09 (0.01, 0.17)	0.22 (0.14, 0.30)
Ex-smoker	0.10 (0.04, 0.15)	0.10 (0.04, 0.15)
BMI status, reference: under and normal weight		
Overweight	0.43 (0.35, 0.51)	0.22 (0.14, 0.30)
Obese	0.45 (0.37, 0.53)	0.32 (0.24, 0.40)
Hypertensive	-0.01 (-0.07, 0.04)	0.06 (0.01, 0.11)
HbA1c	0.06 (0.04, 0.08)	0.00 (-0.02, 0.01)
Cholesterol	-0.28 (-0.30, -0.25)	-0.11 (-0.13, -0.08)
eGFR	-0.01 (-0.01, 0.00)	0.00 (0.00, 0.00)
Ischaemic Cardiac	0.95 (0.89, 1.01)	1.73 (1.67, 1.79)
Stroke or TIA	0.23 (0.15, 0.32)	0.86 (0.78, 0.95)
PVD	0.17 (0.07, 0.27)	0.33 (0.23, 0.43)

<b>Interventions</b>		
Care level, reference group: <7		
Care level: 7	0.14 (0.07, 0.21)	0.04 (-0.03, 0.10)
Care level: 8	0.10 (0.03, 0.18)	-0.01 (-0.09, 0.07)
M. PCT	0.17 (-0.03, 0.37)	0.12 (-0.14, 0.38)
Shared care	-0.10 (-0.16, -0.03)	0.29 (0.22, 0.36)
<b>Cons</b>	-0.41 (-0.98, 1.64)	-1.78 (-2.22, -1.24)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.09 (0.05, 0.15)	0.16 (0.10, 0.26)
<b>Patient level</b>	0.41 (0.00, 5.15)	0.00 (0.00, 0.01)
<b>Bayesian DIC</b>	36560.43	37629.34
N = 33,603		

## Shared care

Table 17: Saturated logistic regression multilevel model examining shared care with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	<b>Shared care</b>
<b>Social-economic status</b>	
Social-economic status, reference group: Low	
Mid	0.25 (-0.27, 0.80)
High	-0.61 (-1.08, -0.17)
Visit year, reference group: 1999	
2000	-0.82 (-1.19, -0.46)
2001	-1.51 (-1.88, -1.17)
2002	-1.91 (-2.26, -1.59)
2003	-2.35 (-2.70, -2.03)
2004	-2.84 (-3.20, -2.52)
2005	-3.16 (-3.51, -2.84)
2006	-3.52 (-3.88, -3.19)
2007	-3.08 (-3.44, -2.75)
SES*Visit year, reference group: Low SES*1999	
Mid SES*2000	-0.30 (-0.93, 0.33)
Mid SES*2001	-0.51 (-1.13, 0.09)
Mid SES*2002	-0.42 (-1.01, 0.15)
Mid SES*2003	-0.33 (-0.93, 0.24)
Mid SES*2004	-0.04 (-0.63, 0.53)
Mid SES*2005	0.14 (-0.45, 0.71)
Mid SES*2006	0.15 (-0.43, 0.72)
Mid SES*2007	0.13 (-0.46, 0.72)
High SES*2000	0.47 (-0.06, 1.01)
High SES*2001	0.44 (-0.06, 0.97)
High SES*2002	0.72 (0.24, 1.23)
High SES*2003	0.76 (0.29, 1.26)
High SES*2004	0.76 (0.28, 1.25)
High SES*2005	0.85 (0.37, 1.34)
High SES*2006	0.72 (0.24, 1.22)
High SES*2007	0.69 (0.20, 1.19)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>	
Age, reference group: <60 years	
60-74	-0.46 (-0.53, -0.38)
75+	-0.85 (-0.96, -0.75)

Duration of diabetes, reference group: 0-3 years	
Duration: 4-9 years	0.01 (-0.07, 0.09)
Duration 10+ years	0.54 (0.45, 0.63)
Ethnicity, reference: White	
South Asian	0.13 (-0.02, 0.28)
Other Ethnicity	0.72 (0.39, 1.05)
Male	0.06 (0.00, 0.13)
Smoking status, reference: non-smoker	
Smoker	-0.32 (-0.42, -0.22)
Ex-smoker	-0.04 (-0.12, 0.03)
BMI status, reference: under and normal weight	
Overweight	0.08 (-0.02, 0.18)
Obese	0.39 (0.28, 0.49)
HbA1c	0.11 (0.09, 0.13)
Hypertensive	0.47 (0.41, 0.54)
Cholesterol	-0.08 (-0.11, -0.05)
Creatinine > 300	0.06 (-0.47, 0.58)
eGFR	-0.01 (-0.01, 0.00)
Ischaemic Cardiac	0.21 (0.13, 0.29)
Stroke or TIA	0.17 (0.06, 0.27)
PVD	0.7 (0.59, 0.81)
<b>Interventions</b>	
Quality of care level, reference group: Low quality	
Care level: 7	0.60 (0.52, 0.69)
Care level: 8	1.25 (1.15, 1.35)
Diabetes treatment, reference group diet alone	
Metformin/sulphonylureas only	0.75 (0.62, 0.88)
Comb. no insulin	0.78 (0.65, 0.92)
Insulin only	3.28 (3.14, 3.43)
Insulin & others	1.59 (1.47, 1.71)
BP treatment, reference group no treatments	
ACE inhibitors only	-0.04 (-0.15, 0.07)
ACE & other(s)	-0.01 (-0.10, 0.08)
Other BP	-0.10 (-0.19, -0.01)
Aspirin	0.22 (0.15, 0.30)
Lipid therapy	-0.20 (-0.28, -0.13)
Middlesbrough PCT	0.65 (0.17, 1.21)
<b>Cons</b>	-1.19 (-1.77, -0.50)
<b>Variance estimate at:</b>	
<b>Practice level</b>	1.00 (0.62, 1.62)
<b>Patient level</b>	0.01 (0.00, 0.03)
<b>Bayesian DIC</b>	25638.09

N = 33,115

Table 17 shows the results for the saturated logistic regression multilevel model that examined shared care by SES. There were 33,115 available cases available for each set of analyses. The results shows that were significant differences in the receipt of shared care by SES with high SES patients less likely to receive shared care, however, over time these were more likely to receive shared care compared to low SES patients in 1999. These findings suggest a possible increase in differences by SES compared to 1999. These results remained consistent following the introduction of other covariates into the model (Table 76, Appendix G).

Increased age, being a smoker and higher cholesterol all had a significant negative association with receiving shared care. Having diabetes for 10 years or more was significantly associated with receiving shared care compared to those who had the condition for less than 4 years. This was likely to reflect the progressive nature of diabetes and the longer patients have diabetes the more complications they were likely to have. The idea that having poorer control and more complex care needs was also supported by the significant positive associations of being obese, hypertensive, increased HbA1c, having a history of ICD, stroke or TIA and PVD and being prescribed any diabetes treatment combination and aspirin with this outcome. However, this does not explain the significant negative association that cholesterol, being prescribed other BP treatments and lipid therapies have with receipt of shared care.

Increased quality of care also had a significant positive association with shared care. This may support the theory here that patients with more complex needs receive greater levels of care. It may also reflect that greater quality of care occurs in shared care than primary care and/or the quality of recording from these different locations than the actual care patients receive. Interestingly patients under the responsibility of Middlesbrough PCT were more likely to receive shared care. There are many potential explanations for this. On a macro level Middlesbrough PCT may be more inclined to fund patients' referral to specialist care. However, it may be more a reflection in terms of access as the Diabetes Care Centre is located within Middlesbrough PCT and the majority of Redcar & Cleveland PCT is rural requiring extensive journey time to attend this clinic. The results also show that there has been a significant reduction in the rate of patients receiving shared care over the study period. This reflects the national policy trend of moving chronic disease management into primary from secondary care.

The ICC showed that 23.26% of variation in the receipt of shared care was explained at the practice level. This was notably higher than the variation of any of the other interventions modelled in this chapter. This should be explored further as it may indicate that patients in certain practices are being denied access to specialist services or in contrast, it may be because some practices may be more effective at managing patients within a primary care setting and therefore there is potential to identify best practice techniques.

## Principle findings

Overall, these analyses found socio-economic inequalities in quality of care, some diabetes and BP treatments regimens, and the receipt of shared care over time. There was also one statistically significant result that indicated inequalities in timeliness of diagnosis over time. These results were not explained by controlling for other relevant variables.

## Chapter 8: Are there intervention-generated inequalities in type 2 diabetes care?

The previous two chapters have examined inequalities in intermediate outcomes, long-term complications and type 2 diabetes interventions by SES over time. This chapter moves on from these analyses and aims to establish whether the same interventions differ in their association with patients' health outcomes by SES. This was achieved by modelling health outcomes with interaction effects between SES and interventions. Significant results in the interaction effects would indicate that the intervention differed in its association with the health outcome according to the patients SES and therefore could indicate the presence of intervention generated inequalities.

Whilst health variables were used as the dependent variable in this section, the focus was on the possible differential effect of interventions as measured by differences in health by SES. As such, this section was organised by interventions with a variety of health outcomes examined. The description of the results focuses on the intervention of interest and the interaction with SES results whilst in the discussion evidence from the previous two chapters were drawn upon to highlight where there was evidence of intervention generated inequalities.

### Timeliness of diagnosis

The graphical analyses in chapter five showed some evidence of differences in timeliness of diagnosis by SES; with low SES patients having a higher HbA1c to another status groups in 2002 and from 2004 to 2007 (Figure 19). In the saturated multilevel analyses in chapter 7, there was only one incidence of evidence of statistically significant differences in timeliness of diagnosis over time, with mid SES more likely to have a lower HbA1c at diagnosis in 2002 compared to low SES patients in 1999 (Table 12).

Table 18 shows the results for the saturated logistic regression multilevel models comparing retinopathy and microalbuminuria with interactions of timeliness of diagnosis and SES. There were 6,957 available cases for the model with retinopathy as the dependent variable and 8,260 available cases for the model with microalbuminuria as the dependent variable. The results in

Table 18 show that delay of diagnosis, as measured by the level of HbA1c at diagnosis, was a significant positive predictor of retinopathy but microalbuminuria. There was no significant interaction effect between timeliness of diagnosis and SES in either model suggesting that there was no difference by SES in the effect of timeliness of diagnosis on these outcomes.

Table 18: Saturated logistic regression multilevel models examining retinopathy and microalbuminuria with interaction effect between SES and HbA1c at diagnosis by 1999 to 2007, conditional on relevant explanatory variables

	Retinopathy	Microalbuminuria
<b>Interactions</b>		
HbA1c at diagnosis, reference group: Low SES*HbA1c at diagnosis		
Mid SES*HbA1c at diagnosis	-0.02 (-0.13, 0.09)	0.05 (-0.01, 0.12)
High SES*HbA1c at diagnosis	0.00 (-0.10, 0.11)	0.03 (-0.04, 0.09)
<b>Social-economic status, reference group: low</b>		
Mid	0.08 (-0.88, 1.03)	-0.54 (-1.13, 0.01)
High	0.05 (-0.85, 0.91)	-0.28 (-0.81, 0.25)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	0.06 (-0.18, 0.31)	-0.11 (-0.24, 0.02)
Age: 75+ years	0.13 (-0.20, 0.45)	0.16 (-0.01, 0.34)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	0.16 (-0.07, 0.38)	-0.01 (-0.14, 0.11)
Ethnicity, reference group: White		
South Asian	0.10 (-0.58, 0.70)	0.22 (-0.10, 0.54)
Other Ethnicity	0.72 (-0.17, 1.55)	0.20 (-0.59, 0.95)
Male	0.15 (-0.05, 0.36)	<b>0.19 (0.07, 0.30)</b>
Smoking status, reference: non-smoker		
Smoker	-0.13 (-0.43, 0.17)	<b>0.37 (0.21, 0.53)</b>
Ex-smoker	0.00 (-0.21, 0.23)	<b>0.21 (0.09, 0.33)</b>
BMI status, reference group: under and normal weight		
Overweight	-0.24 (-0.54, 0.06)	0.13 (-0.04, 0.30)
Obese	<b>-0.40 (-0.69, -0.10)</b>	<b>0.19 (0.03, 0.37)</b>
eGFR	-0.01 (-0.02, 0.00)	-0.01 (-0.01, 0.00)
Hypertensive	<b>0.48 (0.29, 0.68)</b>	<b>0.17 (0.06, 0.29)</b>
Cholesterol	-0.06 (-0.15, 0.04)	0.03 (-0.02, 0.08)
<b>Interventions</b>		
HbA1c at diagnosis	<b>0.10 (0.04, 0.17)</b>	-0.54 (-1.13, 0.01)
Quality of care, reference group: Low (Microalbuminuria) or Mid (Retinopathy)		
Mid		0.24 (-0.58, 1.05)
High	-0.05 (-0.30, 0.21)	0.01 (-0.81, 0.82)
Diabetes treatment, reference group diet alone		
Metformin or sulphonylureas only	<b>0.34 (0.09, 0.60)</b>	<b>0.19 (0.05, 0.33)</b>
Combination, with no insulin	<b>0.56 (0.25, 0.87)</b>	0.05 (-0.14, 0.24)
Insulin only	0.33 (-0.15, 0.79)	0.09 (-0.25, 0.41)
Combination with insulin	<b>0.68 (0.20, 1.14)</b>	<b>0.32 (0.16, 0.48)</b>
BP treatment, reference group: no treatment		
ACE Inhibitors only	<b>0.47 (0.15, 0.81)</b>	<b>0.25 (0.07, 0.44)</b>
Combination with ACEI	<b>0.45 (0.16, 0.75)</b>	<b>0.34 (0.19, 0.50)</b>
Combination no ACEI	0.21 (-0.07, 0.52)	0.14 (-0.01, 0.29)
Aspirin	0.00 (-0.20, 0.20)	-0.04 (-0.15, 0.07)
Lipid therapy	-0.11 (-0.33, 0.12)	0.13 (0.00, 0.25)
Shared care	0.28 (0.01, 0.54)	<b>-1.26 (-1.46, -1.07)</b>
Middlesbrough PCT	-0.17 (-0.49, 0.15)	<b>0.77 (0.35, 1.18)</b>

Visit year, reference group: 1999		
2000	-0.80 (-1.98, 0.37)	-1.52 (-2.61, -0.32)
2001	-0.38 (-1.38, 0.64)	-1.53 (-2.42, -0.48)
2002	-0.34 (-1.28, 0.63)	-1.75 (-2.61, -0.74)
2003	-0.36 (-1.28, 0.59)	-1.56 (-2.39, -0.57)
2004	-0.13 (-1.06, 0.82)	-0.63 (-1.45, 0.36)
2005	0.26 (-0.65, 1.22)	-0.44 (-1.26, 0.55)
2006	-1.16 (-2.09, -0.18)	0.00 (-0.81, 0.99)
2007	0.47 (-0.44, 1.41)	-0.49 (-1.42, 0.61)
<b>Cons</b>	-2.77 (-4.07, -1.41)	-1.32 (-2.76, -0.16)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.15 (0.05, 0.30)	0.38 (0.23, 0.62)
<b>Patient level</b>	0.01 (0.00, 0.07)	0.01 (0.00, 0.04)
<b>Bayesian DIC</b>	3516.17	8919.12
<b>Available case (N)</b>	6957	8260

## Quality of care

In chapter 5, increased quality of care was a statistically significant predictor of lower levels of HbA1c, retinopathy and incidences of PVD (Table 2 and Table 3). The graphical analyses (Figure 20) in the same chapter found some, but inconsistent, evidence of statistically significant differences in the level of care patients receive over time. Patients with low SES were found to have a lower mean number of care processes in most years compared to another status group. The multilevel analyses found statistically significant differences in quality of care, with high SES associated with a lower quality of care. In contrast, the interaction effect between SES and visit year resulted in a statistically significant association between high SES and time, suggesting that high SES patients have received greater quality of care compared to low SES patients from 2002 to 2007 compared to 1999. As such if low SES patients were likely to receive poorer care over time and high quality of care was associated with more favourable health outcomes this suggests the potential that quality of care may contribute to health inequalities by SES.

Table 19, Table 20 and Table 21 show the results of the saturated linear and logistic regression multilevel models examining health outcomes with interaction effects between SES and quality of care. The results in Table 19 show that quality of care had a differential relationship with HbA1c by SES, but not with cholesterol. High SES patients receiving high quality care were significantly more likely to have poorer HbA1c compared to low SES patients. However, overall high SES patients were more likely to have more favourable HbA1c levels than low SES patients and low quality of care. There were no differences in cholesterol levels by SES. The results in Table 20 and Table 21 show that overall the relationship between quality of care and long-term complications did not differ by SES. However, there was one exception. Mid SES patients were

significantly more likely to have PVD compared to low SES patients. Overall, however, mid status patients were less likely to have PVD.

Overall, the association between quality of care and health outcomes was consistent across SES. However, there was some, but not consistent evidence, that quality of care could have a differential impact on patients HbA1c, an important indicator of patients' diabetes control, and incidences of PVD.

Table 19: Saturated linear regression multilevel models examining HbA1c and cholesterol levels with interaction effect between SES and quality of care by 1999 to 2007, conditional on relevant explanatory variables

	HbA1c	Cholesterol
<b>Social-economic status, reference group: low</b>		
Mid	-0.07 (-0.15, 0.01)	0.06 (0.00, 0.12)
High	-0.14 (-0.21, -0.07)	-0.02 (-0.07, 0.03)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	-0.33 (-0.36, -0.29)	-0.20 (-0.23, -0.17)
Age: 75+ years	-0.41 (-0.46, -0.37)	-0.26 (-0.30, -0.23)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	0.06 (0.02, 0.09)	-0.09 (-0.11, -0.06)
Duration 10+ years	0.05 (0.01, 0.10)	-0.13 (-0.16, -0.10)
Ethnicity, reference group: White		
South Asian	0.46 (0.39, 0.53)	-0.08 (-0.14, -0.02)
Other Ethnicity	0.48 (0.31, 0.64)	0.08 (-0.05, 0.20)
Male	-0.06 (-0.09, -0.03)	-0.34 (-0.36, -0.32)
Smoking status, reference: non-smoker		
Smoker	0.23 (0.19, 0.27)	0.08 (0.05, 0.11)
E*-smoker	0.05 (0.02, 0.08)	0.00 (-0.03, 0.02)
Obesity category, reference group: under and normal weight		
Overweight	0.02 (-0.02, 0.07)	0.03 (0.00, 0.07)
Obese	0.08 (0.04, 0.13)	0.03 (0.00, 0.07)
Creatinine > 300	-0.80 (-1.04, -0.56)	
Hypertensive	0.10 (0.07, 0.13)	0.14 (0.12, 0.16)
ICD	0.00 (-0.04, 0.03)	-0.13 (-0.15, -0.10)
Stroke or TIA	-0.06 (-0.10, -0.01)	-0.02 (-0.06, 0.01)
PVD	-0.06 (-0.12, -0.01)	0.01 (-0.03, 0.06)
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.14 (-0.19, -0.09)	-0.09 (-0.13, -0.05)
High	-0.19 (-0.25, -0.13)	-0.16 (-0.20, -0.11)
Diabetes treatment, reference group diet alone		
Metformin or sulphonylureas only	0.81 (0.77, 0.84)	
Combination, with no insulin	1.25 (1.20, 1.29)	
Insulin only	1.67 (1.61, 1.73)	
Combination with insulin	1.75 (1.69, 1.82)	
Aspirin		-0.09 (-0.11, -0.06)
Lipid therapy		-0.28 (-0.31, -0.26)
Middlesbrough PCT	0.10 (0.01, 0.20)	-0.03 (-0.09, 0.03)
Shared care	0.17 (0.13, 0.21)	-0.07 (-0.10, -0.04)
Visit year, reference group: 1999		

2000	-0.32 (-0.40, -0.23)	-0.20 (-0.29, -0.11)
2001	-0.47 (-0.55, -0.39)	-0.21 (-0.30, -0.12)
2002	-0.55 (-0.63, -0.47)	-0.22 (-0.31, -0.13)
2003	-0.59 (-0.67, -0.52)	-0.37 (-0.46, -0.29)
2004	-0.63 (-0.71, -0.56)	-0.58 (-0.66, -0.50)
2005	-0.72 (-0.79, -0.64)	-0.74 (-0.82, -0.65)
2006	-1.18 (-1.26, -1.11)	-0.84 (-0.92, -0.75)
2007	-1.12 (-1.20, -1.05)	-0.93 (-1.02, -0.85)
<b>Interactions</b>		
Quality of care, reference group: Low SES & Low quality		
Mid SES*Mid quality	0.01 (-0.08, 0.10)	-0.05 (-0.12, 0.03)
Mid SES*High quality	0.07 (-0.03, 0.17)	-0.02 (-0.10, 0.06)
High SES*Mid quality	0.05 (-0.03, 0.14)	0.00 (-0.07, 0.06)
High SES*High quality	<b>0.10 (0.01, 0.19)</b>	0.05 (-0.02, 0.12)
<b>Cons</b>	7.62 (7.49, 7.74)	5.92 (5.81, 6.03)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.02 (0.01, 0.04)	0.01 (0.01, 0.01)
<b>Patient level</b>	0.00 (0.00, 0.02)	0.00 (0.00, 0.01)
<b>Visit year</b>	1.91 (1.88, 1.94)	1.14 (1.13, 1.16)
<b>Bayesian DIC</b>	133976.66	110338.38
<b>Available cases (N)</b>	38,413	37,085

Table 20: Saturated logistic regression multilevel models examining incidences of ICD, stroke or TIA and PVD with interaction effect between SES and quality of care by 2000 to 2007, conditional on relevant explanatory variables

	ICD	Stroke or TIA	PVD
<b>Social-economic status, reference group: low</b>			
Mid	-0.26 (-0.55, 0.03)	-0.30 (-0.73, 0.11)	<b>-0.62 (-1.19, -0.09)</b>
High	-0.19 (-0.44, 0.07)	-0.14 (-0.52, 0.23)	-0.30 (-0.77, 0.17)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>			
Age, reference group: <60 years			
Age: 60-74 years	<b>0.34 (0.19, 0.48)</b>	<b>0.74 (0.50, 0.99)</b>	<b>0.61 (0.37, 0.87)</b>
Age: 75+ years	<b>0.60 (0.41, 0.79)</b>	<b>1.10 (0.83, 1.38)</b>	<b>0.81 (0.51, 1.11)</b>
Duration of diabetes, reference group: 0-3 years			
Duration: 4-9 years	<b>-0.59 (-0.73, -0.45)</b>	<b>-0.31 (-0.50, -0.12)</b>	0.13 (-0.09, 0.35)
Duration 10+ years	<b>-0.63 (-0.80, -0.46)</b>	-0.18 (-0.39, 0.02)	<b>0.38 (0.14, 0.62)</b>
Ethnicity, reference group: White			
South Asian	0.08 (-0.25, 0.41)	0.11 (-0.33, 0.52)	<b>-0.80 (-1.51, -0.17)</b>
Other Ethnicity	-0.67 (-1.54, 0.10)	-1.17 (-3.02, 0.15)	-0.06 (-1.14, 0.83)
Male	<b>0.32 (0.20, 0.44)</b>	-0.11 (-0.27, 0.05)	<b>0.39 (0.20, 0.59)</b>
Smoking status, reference: non-smoker			
Smoker	0.15 (-0.03, 0.32)	<b>0.28 (0.05, 0.51)</b>	<b>0.94 (0.68, 1.19)</b>
E*-smoker	<b>0.32 (0.19, 0.45)</b>	0.14 (-0.03, 0.31)	<b>0.37 (0.16, 0.59)</b>
Obesity category, reference group: under and normal weight			
Overweight	-0.02 (-0.19, 0.16)	-0.09 (-0.30, 0.13)	-0.20 (-0.46, 0.05)
Obese	0.12 (-0.05, 0.30)	-0.19 (-0.41, 0.03)	-0.25 (-0.50, 0.00)
eGFR	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)
Hypertensive	<b>-0.27 (-0.39, -0.16)</b>	0.14 (-0.01, 0.30)	0.13 (-0.05, 0.31)
Cholesterol	<b>-0.23 (-0.29, -0.17)</b>	<b>-0.09 (-0.16, -0.01)</b>	-0.05 (-0.13, 0.03)
HbA1c	<b>0.07 (0.03, 0.11)</b>	0.02 (-0.04, 0.07)	0.01 (-0.05, 0.07)
<b>Interventions</b>			
Quality of care, reference group: Low			
Mid	<b>-0.30 (-0.49, -0.11)</b>	<b>-0.32 (-0.59, -0.05)</b>	<b>-0.35 (-0.68, -0.03)</b>
High	<b>-0.50 (-0.73, -0.27)</b>	-0.10 (-0.41, 0.20)	0.10 (-0.22, 0.43)
Diabetes treatment, reference group diet alone			
Metformin or sulphonylureas only	<b>-0.28 (-0.43, -0.14)</b>	-0.18 (-0.38, 0.03)	-0.05 (-0.31, 0.21)

Combination, with no insulin	-0.40 (-0.60, -0.21)	-0.29 (-0.56, -0.03)	-0.11 (-0.42, 0.19)
Insulin only	0.12 (-0.13, 0.36)	-0.07 (-0.38, 0.24)	0.34 (0.00, 0.68)
Combination with insulin	-0.31 (-0.61, -0.03)	-0.28 (-0.66, 0.08)	0.35 (-0.03, 0.73)
BP treatment, reference group: no treatment			
ACE inhibitors only	0.27 (0.01, 0.53)	0.30 (0.01, 0.59)	0.48 (0.16, 0.81)
Combination, with ACEI	1.51 (1.31, 1.70)	0.15 (-0.08, 0.40)	0.42 (0.14, 0.71)
Combination, no ACEI	1.25 (1.05, 1.45)	0.18 (-0.06, 0.42)	0.37 (0.09, 0.66)
Aspirin	1.37 (1.25, 1.49)	1.06 (0.89, 1.23)	0.58 (0.39, 0.76)
Lipid therapy	0.61 (0.47, 0.75)	0.09 (-0.09, 0.27)	0.10 (-0.10, 0.29)
Shared care	0.28 (0.12, 0.44)	0.44 (0.24, 0.64)	0.84 (0.62, 1.05)
Middlesbrough PCT	-0.20 (-0.39, -0.01)	-0.12 (-0.39, 0.15)	-0.19 (-0.56, 0.19)
Visit year, reference group: 2000			
2001	-0.29 (-0.59, 0.01)	0.21 (-0.18, 0.61)	-0.23 (-0.65, 0.18)
2002	-0.30 (-0.57, -0.03)	0.03 (-0.34, 0.40)	-0.25 (-0.62, 0.13)
2003	-0.40 (-0.66, -0.14)	0.14 (-0.21, 0.50)	0.03 (-0.33, 0.39)
2004	-0.67 (-0.93, -0.40)	0.03 (-0.33, 0.39)	0.00 (-0.36, 0.36)
2005	-1.25 (-1.52, -0.97)	-0.20 (-0.57, 0.18)	-0.30 (-0.68, 0.08)
2006	-1.67 (-1.97, -1.38)	-0.81 (-1.21, -0.40)	-0.79 (-1.21, -0.37)
2007	-1.81 (-2.11, -1.51)	-0.73 (-1.14, -0.32)	-1.14 (-1.60, -0.68)
<b>Interactions</b>			
Quality of care, reference group: Low SES & Low quality			
Mid SES*Mid quality	0.01 (-0.33, 0.37)	0.40 (-0.09, 0.90)	0.71 (0.09, 1.37)
Mid SES*High quality	0.23 (-0.16, 0.61)	0.32 (-0.19, 0.85)	0.38 (-0.24, 1.03)
High SES*Mid quality	-0.02 (-0.34, 0.30)	0.26 (-0.19, 0.71)	0.14 (-0.44, 0.70)
High SES*High quality	0.15 (-0.21, 0.51)	0.04 (-0.45, 0.54)	0.08 (-0.48, 0.64)
<b>Cons</b>	-3.89 (-4.89, -2.90)	-4.88 (-6.37, -3.29)	-5.82 (-7.13, -4.70)
<b>Variance estimate at:</b>			
<b>Practice level</b>	0.05 (0.02, 0.11)	0.11 (0.04, 0.22)	0.27 (0.14, 0.48)
<b>Patient level</b>	1.98 (0.53, 6.31)	1.41 (0.33, 5.19)	1.39 (0.34, 4.41)
<b>Bayesian DIC</b>	9200.75	6137.51	5025.34

Table 21: Saturated logistic regression multilevel models examining recorded microalbuminuria and retinopathy with interaction effect between SES and quality of care by 2000 to 2007, conditional on relevant explanatory variables

	Microalbuminuria	Retinopathy
<b>Social-economic status, reference group: low</b>		
Mid	0.24 (-0.71, 1.10)	4.06 (-1.65, 9.97)
High	-0.60 (-1.41, 0.25)	-5.83 (-11.03, 0.13)
<b>Covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	0.01 (-0.06, 0.09)	0.00 (-0.11, 0.11)
Age: 75+ years	0.38 (0.28, 0.47)	-0.11 (-0.25, 0.04)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	-0.01 (-0.09, 0.06)	0.47 (0.34, 0.60)
Duration 10+ years	0.18 (0.09, 0.27)	1.60 (1.46, 1.73)
Ethnicity, reference group: White		
South Asian	0.21 (0.05, 0.38)	-0.15 (-0.39, 0.08)
Other Ethnicity	0.30 (-0.08, 0.68)	0.52 (0.08, 0.96)
Male	0.24 (0.18, 0.31)	0.25 (0.16, 0.34)
Smoking status, reference: non-smoker		
Smoker	0.26 (0.17, 0.36)	-0.13 (-0.26, 0.00)
Ex-smoker	0.07 (0.00, 0.14)	-0.12 (-0.22, -0.02)
Obesity category, reference group: under and normal weight		
Overweight	-0.01 (-0.11, 0.09)	-0.09 (-0.22, 0.05)
Obese	0.06 (-0.04, 0.16)	-0.10 (-0.23, 0.04)
eGFR		-0.01 (-0.01, -0.01)

Hypertensive	0.18 (0.11, 0.24)	0.34 (0.25, 0.42)
Cholesterol	0.03 (0.00, 0.06)	-0.02 (-0.06, 0.02)
HbA1c	0.09 (0.06, 0.11)	0.05 (0.02, 0.08)
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.19 (-0.84, 0.44)	-0.19 (-1.90, 1.67)
High	-0.30 (-0.94, 0.34)	-0.28 (-2.00, 1.58)
Diabetes treatment, reference group diet alone		
Metformin/sulphonylureas only	0.14 (0.04, 0.24)	0.40 (0.24, 0.57)
Combination, with no insulin	0.02 (-0.10, 0.13)	0.70 (0.53, 0.87)
Insulin only	0.18 (0.04, 0.32)	1.05 (0.85, 1.23)
Combination with insulin	0.20 (0.10, 0.30)	1.16 (0.96, 1.37)
BP treatments, reference group: No BP treatment		
ACE Inhibitors only	0.37 (0.26, 0.47)	0.31 (0.17, 0.46)
Combination with ACEI	0.53 (0.43, 0.62)	0.22 (0.09, 0.34)
Combination no ACEI	0.32 (0.23, 0.41)	0.12 (-0.01, 0.25)
Aspirin	0.08 (0.01, 0.14)	0.05 (-0.04, 0.14)
Lipid therapy	-0.05 (-0.12, 0.02)	-0.06 (-0.16, 0.03)
Shared care	-0.93 (-1.02, -0.84)	0.53 (0.42, 0.63)
Middlesbrough PCT	0.59 (0.31, 0.89)	-0.05 (-0.22, 0.14)
Visit year, reference group: 1999		
2000	-0.36 (-0.70, -0.03)	0.01 (-0.29, 0.31)
2001	-0.58 (-0.90, -0.27)	-0.02 (-0.32, 0.27)
2002	-0.81 (-1.12, -0.50)	0.00 (-0.28, 0.28)
2003	-0.69 (-1.00, -0.40)	-0.12 (-0.40, 0.17)
2004	0.07 (-0.23, 0.36)	0.06 (-0.22, 0.35)
2005	0.26 (-0.04, 0.55)	0.33 (0.04, 0.64)
2006	0.63 (0.32, 0.92)	-0.70 (-1.00, -0.40)
2007	0.34 (-0.10, 0.76)	0.36 (0.06, 0.65)
<b>Interactions</b>		
Quality of care, reference group: Low SES & Low quality		
Mid SES*Mid quality	-0.33 (-1.21, 0.63)	-4.13 (-10.02, 1.61)
Mid SES*High quality	-0.33 (-1.21, 0.62)	-4.14 (-10.04, 1.55)
High SES*Mid quality	0.53 (-0.33, 1.34)	5.69 (-0.25, 10.90)
High SES*High quality	0.49 (-0.37, 1.31)	5.81 (-0.13, 11.01)
<b>Cons</b>	-2.40 (-3.18, -1.69)	-2.75 (-4.71, -0.95)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.21 (0.13, 0.34)	0.05 (0.03, 0.10)
<b>Patient level</b>	0.01 (0.00, 0.03)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	25472.88	14526.88

## Diabetes treatments

In chapter 6, all diabetes treatments regimens had a statistically significant association with poorer levels of HbA1c compared to patients who were treated through lifestyle modification. This result was somewhat expected as it is when patients HbA1c deteriorates that these treatments would be initiated. However, when examining the results where long-term complications were modelled the results show that most of the diabetes treatment regimens were significantly associated with lower rates of ICD but with higher rates of retinopathy. Being treated with combination of diabetes treatments without insulin was also significantly associated with lower incidences of stroke or TIA.

The graphical analyses in chapter 5 showed some evidence of differences in diabetes treatments by SES over time (Figure 23, Figure 22, Figure 25, Figure 26 and Figure 27). This was particularly evident in being prescribed no diabetes treatments (Figure 23). The results from the multilevel modelling showed that there were statistically significant differences in the prescription of diabetes treatments over time whilst taking into account patients' health status, and other variables (Table 14). These results may indicate that these treatments were not being prescribed methodically across patients groups and could potentially account for the divergent associations between diabetes treatments regimen and long-term complications.

Table 22, Table 23 and Table 24 contain the results from modelling patient's health outcomes with an interaction effect between SES and diabetes interventions. Whilst the majority of the results from these models indicated that there were no significant differences in the impact of diabetes treatments on health by SES, there was some evidence of potential intervention generated inequalities.

The results from Table 22 show that, for high SES patients, there was a significant association between having lower HbA1c and prescriptions for insulin, either alone or in combination with other diabetes treatments compared to low SES patients. The results from long-term complications in Table 23 and Table 24 shows that for high SES patients, compared to low SES patients, there were significant associations between lower rates of ICD and retinopathy and prescriptions for insulin in combination with other diabetes treatment and insulin only respectively. In contrast, mid SES patients prescribed insulin only were significantly more likely to have higher microalbuminuria rates compared to low SES patients. There were no other statistically significant results indicating that in general diabetes interventions were not associated with differences in health by SES.

Table 22: Saturated linear regression multilevel models examining HbA1c levels with interaction effect between SES and diabetes treatment regimens by 1999 to 2007, conditional on relevant explanatory variables

	HbA1c
<b>Social-economic status, reference group: low</b>	
Mid	0.01 (-0.07, 0.08)
High	0.00 (-0.07, 0.07)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>	
Age, reference group: <60 years	
Age: 60-74 years	-0.33 (-0.36, -0.29)
Age: 75+ years	-0.41 (-0.46, -0.37)
Duration of diabetes, reference group: 0-3 years	
Duration: 4-9 years	0.06 (0.02, 0.09)
Duration 10+ years	0.06 (0.02, 0.10)
Ethnicity, reference group: White	
South Asian	0.46 (0.39, 0.53)
Other Ethnicity	0.46 (0.30, 0.63)
Male	-0.06 (-0.09, -0.03)
Smoking status, reference: non-smoker	
Smoker	0.23 (0.19, 0.27)
Ex-smoker	0.05 (0.02, 0.08)
Obesity category, reference group: under and normal weight	
Overweight	0.03 (-0.02, 0.07)
Obese	0.08 (0.04, 0.13)
Creatinine > 300	-0.80 (-1.05, -0.56)
Hypertensive	0.10 (0.07, 0.13)
Ischaemic Cardiac	-0.01 (-0.04, 0.02)
Stroke or TIA	-0.06 (-0.10, -0.01)
PVD	-0.07 (-0.12, -0.01)
<b>Interventions</b>	
Quality of care, reference group: Low	
Mid	-0.13 (-0.16, -0.09)
High	-0.15 (-0.19, -0.11)
Diabetes treatment, reference group diet alone	
Metformin or sulphonylureas only	0.84 (0.78, 0.89)
Combination, with no insulin	1.31 (1.24, 1.37)
Insulin only	1.77 (1.69, 1.85)
Combination with insulin	1.82 (1.73, 1.91)
Middlesbrough PCT	0.10 (0.00, 0.20)
Shared care	0.17 (0.13, 0.21)
Visit year, reference group: 1999	
2000	-0.32 (-0.40, -0.23)
2001	-0.47 (-0.55, -0.39)
2002	-0.55 (-0.63, -0.47)
2003	-0.59 (-0.67, -0.51)
2004	-0.63 (-0.71, -0.56)
2005	-0.71 (-0.79, -0.64)
2006	-1.18 (-1.26, -1.10)
2007	-1.12 (-1.20, -1.04)
<b>Interaction</b>	
Diabetes treatment, reference group: Diet alone & Low SES	
Mid SES*Metformin/sulphonylureas only	-0.04 (-0.13, 0.06)
Mid SES*Combination with no insulin	-0.11 (-0.22, 0.00)
Mid SES*Insulin only	-0.11 (-0.24, 0.02)
Mid SES*Combination with insulin	-0.02 (-0.16, 0.13)

High SES*Metformin/sulphonylureas only	-0.05 (-0.14, 0.03)
High SES*Combination, with no insulin	-0.10 (-0.20, 0.00)
High SES*Insulin only	-0.24 (-0.36, -0.13)
High SES*Combination with insulin	-0.23 (-0.37, -0.10)
<b>Cons</b>	7.55 (7.42, 7.68)
<b>Variance estimate at:</b>	
<b>Practice level</b>	0.02 (0.01, 0.04)
<b>Patient level</b>	0.00 (0.00, 0.02)
<b>Visit year</b>	1.91 (1.88, 1.94)
<b>Bayesian DIC</b>	133960.84

Table 23: Saturated logistic regression multilevel models examining incidences of ICD, stroke or TIA and PVD with interaction effect between SES and diabetes treatments by 2000 to 2007, conditional on relevant explanatory variables

	ICD	Stroke or TIA	PVD
<b>Social-economic status, reference group: low</b>			
Mid	-0.29 (-0.55, -0.03)	0.06 (-0.30, 0.44)	-0.15 (-0.60, 0.32)
High	-0.18 (-0.42, 0.05)	0.07 (-0.27, 0.42)	-0.49 (-0.99, 0.00)
<b>Covariates</b>			
Age, reference group: <60 years			
Age: 60-74 years	0.34 (0.19, 0.49)	0.74 (0.50, 0.98)	0.61 (0.36, 0.86)
Age: 75+ years	0.60 (0.42, 0.79)	1.09 (0.82, 1.37)	0.81 (0.51, 1.11)
Duration of diabetes, reference group: 0-3 years			
Duration: 4-9 years	-0.59 (-0.73, -0.45)	-0.30 (-0.50, -0.11)	0.13 (-0.09, 0.35)
Duration 10+ years	-0.62 (-0.79, -0.46)	-0.17 (-0.39, 0.04)	0.38 (0.14, 0.61)
Ethnicity, reference group: White			
South Asian	0.09 (-0.25, 0.40)	0.10 (-0.35, 0.51)	-0.82 (-1.52, -0.20)
Other Ethnicity	-0.66 (-1.52, 0.12)	-1.18 (-3.02, 0.15)	-0.07 (-1.13, 0.81)
Male	0.32 (0.20, 0.44)	-0.11 (-0.28, 0.05)	0.39 (0.20, 0.59)
Smoking status, reference: non-smoker			
Smoker	0.16 (-0.02, 0.33)	0.27 (0.04, 0.50)	0.93 (0.67, 1.17)
E*-smoker	0.32 (0.19, 0.44)	0.13 (-0.04, 0.30)	0.37 (0.16, 0.58)
Obesity category, reference group: under and normal weight			
Overweight	-0.01 (-0.19, 0.17)	-0.09 (-0.30, 0.13)	-0.20 (-0.45, 0.05)
Obese	0.12 (-0.06, 0.29)	-0.19 (-0.41, 0.03)	-0.26 (-0.50, -0.01)
eGFR	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)
Hypertensive	-0.28 (-0.40, -0.16)	0.14 (-0.01, 0.30)	0.13 (-0.04, 0.31)
Cholesterol	-0.23 (-0.29, -0.17)	-0.09 (-0.16, -0.01)	-0.05 (-0.13, 0.03)
HbA1c	0.07 (0.03, 0.11)	0.02 (-0.04, 0.07)	0.01 (-0.05, 0.07)
<b>Interventions</b>			
Quality of care, reference group: Low			
Mid	-0.30 (-0.44, -0.16)	-0.16 (-0.36, 0.04)	-0.19 (-0.42, 0.05)
High	-0.40 (-0.56, -0.23)	-0.03 (-0.25, 0.19)	0.17 (-0.08, 0.41)
Diabetes treatment, reference group diet alone			
Metformin/sulphonylureas only	-0.40 (-0.61, -0.20)	-0.16 (-0.46, 0.15)	-0.12 (-0.47, 0.26)
Combination, with no insulin	-0.35 (-0.61, -0.09)	-0.29 (-0.67, 0.10)	-0.38 (-0.83, 0.05)
Insulin only	0.11 (-0.20, 0.42)	0.08 (-0.33, 0.49)	0.37 (-0.06, 0.81)
Combination with insulin	-0.22 (-0.59, 0.15)	-0.08 (-0.59, 0.41)	0.20 (-0.28, 0.70)
BP treatment, reference group: no treatment			
ACE inhibitors only	0.27 (0.01, 0.53)	0.30 (0.00, 0.59)	0.48 (0.16, 0.79)
Combination, with ACEI	1.51 (1.32, 1.70)	0.15 (-0.09, 0.40)	0.41 (0.14, 0.69)
Combination, no ACEI	1.25 (1.06, 1.45)	0.18 (-0.06, 0.43)	0.36 (0.08, 0.65)
Aspirin	1.37 (1.26, 1.50)	1.06 (0.89, 1.22)	0.57 (0.39, 0.76)
Lipid therapy	0.61 (0.47, 0.74)	0.08 (-0.09, 0.26)	0.10 (-0.10, 0.30)
Shared care	0.28 (0.12, 0.43)	0.44 (0.24, 0.65)	0.85 (0.63, 1.06)

Middlesbrough PCT	-0.20 (-0.39, -0.01)	-0.11 (-0.37, 0.16)	-0.19 (-0.56, 0.18)
Visit year, reference group: 2000			
2001	-0.29 (-0.59, 0.01)	0.20 (-0.19, 0.59)	-0.24 (-0.65, 0.17)
2002	-0.30 (-0.57, -0.03)	0.04 (-0.32, 0.40)	-0.25 (-0.63, 0.13)
2003	-0.39 (-0.66, -0.13)	0.14 (-0.21, 0.50)	0.02 (-0.34, 0.38)
2004	-0.66 (-0.93, -0.39)	0.03 (-0.33, 0.39)	-0.02 (-0.38, 0.34)
2005	-1.24 (-1.52, -0.95)	-0.20 (-0.56, 0.18)	-0.31 (-0.69, 0.08)
2006	-1.67 (-1.96, -1.37)	-0.8 (-1.21, -0.41)	-0.80 (-1.21, -0.38)
2007	-1.81 (-2.11, -1.50)	-0.72 (-1.12, -0.31)	-1.14 (-1.61, -0.68)
<b>Interactions</b>			
Diabetes treatment, reference group: Diet alone & Low SES			
Mid SES*Metformin/sulphonylurea only	0.27 (-0.07, 0.61)	0.05 (-0.42, 0.52)	-0.04 (-0.63, 0.54)
Mid SES*Combination with no insulin	-0.06 (-0.50, 0.36)	-0.17 (-0.78, 0.41)	0.28 (-0.41, 0.94)
Mid SES*Insulin only	0.02 (-0.49, 0.51)	-0.10 (-0.72, 0.50)	-0.38 (-1.08, 0.29)
Mid SES*Combination with insulin	0.16 (-0.45, 0.74)	-0.81 (-1.75, 0.04)	0.09 (-0.68, 0.85)
High SES*Metformin/sulphonylurea only	0.21 (-0.11, 0.53)	-0.09 (-0.55, 0.37)	0.26 (-0.35, 0.88)
High SES*Combination, with no insulin	-0.16 (-0.58, 0.24)	0.13 (-0.42, 0.67)	0.69 (0.00, 1.38)
High SES*Insulin only	-0.01 (-0.46, 0.44)	-0.53 (-1.16, 0.07)	0.15 (-0.48, 0.80)
High SES*Combination with insulin	-0.73 (-1.45, -0.05)	-0.16 (-0.91, 0.56)	0.41 (-0.35, 1.17)
<b>Cons</b>	-3.99 (-5.39, -2.83)	0.05 (-0.42, 0.52)	-5.76 (-7.03, -4.62)
<b>Variance estimate at:</b>			
<b>Practice level</b>	0.05 (0.02, 0.11)	0.11 (0.04, 0.22)	0.27 (0.14, 0.48)
<b>Patient level</b>	2.19 (0.56, 7.35)	1.39 (0.34, 4.85)	1.35 (0.32, 4.48)
<b>Bayesian DIC</b>	9198.43	6140.12	5031.91

Table 24: Saturated logistic regression multilevel models examining microalbuminuria and retinopathy rates with interaction effect between SES and diabetes treatments by 2000 to 2007, conditional on relevant explanatory variables

	Microalbuminuria	Retinopathy
<b>Social-economic status, reference group: low</b>		
Mid	-0.14 (-0.31, 0.03)	0.00 (-0.35, 0.34)
High	-0.10 (-0.26, 0.06)	0.23 (-0.11, 0.56)
<b>Covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	0.02 (-0.06, 0.09)	0.00 (-0.11, 0.11)
Age: 75+ years	0.38 (0.29, 0.48)	-0.11 (-0.24, 0.03)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	-0.02 (-0.09, 0.06)	0.47 (0.34, 0.60)
Duration 10+ years	0.18 (0.09, 0.27)	1.61 (1.47, 1.74)
Ethnicity, reference group: White		
South Asian	0.22 (0.06, 0.38)	-0.16 (-0.40, 0.07)
Other Ethnicity	0.30 (-0.08, 0.68)	0.49 (0.05, 0.92)
Male	0.24 (0.18, 0.31)	0.25 (0.16, 0.34)
Smoking status, reference: non-smoker		
Smoker	0.26 (0.17, 0.36)	-0.14 (-0.27, 0.00)
E*-smoker	0.07 (0.00, 0.14)	-0.12 (-0.22, -0.02)
Obesity category, reference group: under and normal weight		
Overweight	-0.01 (-0.11, 0.08)	-0.09 (-0.22, 0.04)
Obese	0.06 (-0.04, 0.15)	-0.10 (-0.24, 0.03)
HbA1c	0.09 (0.06, 0.11)	0.05 (0.02, 0.08)
Hypertensive	0.18 (0.11, 0.25)	0.34 (0.25, 0.43)
Cholesterol	0.03 (0.00, 0.06)	-0.01 (-0.06, 0.02)
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.10 (-0.49, 0.27)	-0.66 (-2.24, 0.85)

High	-0.21 (-0.60, 0.16)	-0.73 (-2.29, 0.79)
Diabetes treatment, reference group diet alone		
Metformin or sulphonylureas only	0.12 (-0.03, 0.26)	0.52 (0.27, 0.78)
Combination, with no insulin	0.00 (-0.17, 0.17)	0.79 (0.53, 1.06)
Insulin only	0.06 (-0.12, 0.25)	1.29 (1.02, 1.57)
Combination with insulin	0.21 (0.07, 0.35)	1.24 (0.96, 1.54)
BP treatments, reference group: No BP treatment		
ACE Inhibitors only	0.36 (0.25, 0.47)	0.32 (0.17, 0.47)
Combination with ACEI	0.53 (0.44, 0.62)	0.22 (0.09, 0.34)
Combination no ACEI	0.32 (0.23, 0.41)	0.12 (-0.01, 0.26)
Aspirin	0.08 (0.01, 0.14)	0.05 (-0.04, 0.14)
Lipid therapy	-0.05 (-0.12, 0.02)	-0.06 (-0.16, 0.04)
Shared care	-0.93 (-1.02, -0.84)	0.53 (0.43, 0.64)
Middlesbrough PCT	0.61 (0.31, 0.90)	-0.04 (-0.22, 0.14)
Visit year, reference group: 1999		
2000	-0.41 (-0.74, -0.08)	0.03 (-0.27, 0.33)
2001	-0.61 (-0.93, -0.31)	0.00 (-0.30, 0.30)
2002	-0.85 (-1.15, -0.55)	0.02 (-0.27, 0.31)
2003	-0.74 (-1.04, -0.45)	-0.09 (-0.39, 0.20)
2004	0.02 (-0.27, 0.30)	0.08 (-0.21, 0.38)
2005	0.21 (-0.09, 0.49)	0.35 (0.05, 0.66)
2006	0.58 (0.29, 0.87)	-0.68 (-0.98, -0.37)
2007	0.27 (-0.15, 0.68)	0.38 (0.08, 0.67)
<b>Interactions</b>		
Diabetes treatment, reference group: Diet alone & Low SES		
Mid SES*Metformin/sulphonylureas only	0.01 (-0.23, 0.24)	-0.16 (-0.55, 0.23)
Mid SES*Combination with no insulin	-0.05 (-0.33, 0.23)	-0.05 (-0.44, 0.35)
Mid SES*Insulin only	0.51 (0.22, 0.80)	-0.16 (-0.57, 0.26)
Mid SES*Combination with insulin	0.01 (-0.20, 0.22)	0.08 (-0.35, 0.51)
High SES*Metformin/sulphonylureas only	0.08 (-0.14, 0.30)	-0.22 (-0.60, 0.16)
High SES*Combination, with no insulin	0.09 (-0.18, 0.34)	-0.22 (-0.60, 0.17)
High SES*Insulin only	-0.01 (-0.28, 0.26)	-0.62 (-1.00, -0.22)
High SES*Combination with insulin	-0.04 (-0.24, 0.16)	-0.31 (-0.74, 0.11)
<b>Cons</b>	-2.49 (-3.00, -1.94)	-2.46 (-3.96, -0.89)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.21 (0.13, 0.34)	0.05 (0.03, 0.10)
<b>Patient level</b>	0.01 (0.00, 0.03)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	25462.24	14526.17

## Blood pressure treatments

In general the results in chapter six, BP regimens were found to be significant predictors of higher incidences of long-term complications compared to no BP treatments (Table 10 and Table 11). The graphical analyses in chapter 5 showed, in general, there were no differences in the prescription of BP treatments by SES over time, however, there were some significant differences in particular years in not having a prescription for any BP treatment. The multilevel analyses enabled other variables to be taken into account and the results indicated the presence of significant differences over time in patients being prescribed no BP treatments and a

combination of BP treatments excluding ACEI. Interestingly, high SES were significantly less likely to receive no BP and more likely to receive a combination without ACEI in comparison to low SES. However, these results were not consistent over time.

The evidence from the previous two chapters therefore suggests that BP treatments were not necessarily prescribed in accordance with patients' health outcomes, as measured in these models and that these differences were stratified by patients' SES. In this chapter, the results from Table 25 and Table 26 indicate that there were no differences in the association between these treatments and long-term complications by SES, though there were two exceptions. High SES patients prescribed a combination of BP treatments excluding ACEI were significantly more likely to have microalbuminuria and significantly more likely have retinopathy when prescribed ACEI only compared to low SES patients.

The results presented in this section indicate that there was limited evidence of differences in the association with BP treatments by SES in health outcomes suggesting that overall BP management was unlikely to be a cause of subsequent inequalities in type 2 diabetes patients' health.

Table 25: Saturated logistic regression multilevel models examining incidences of ICD, stroke or TIA and PVD with interaction effect between SES and BP treatments by 2000 to 2007, conditional on relevant explanatory variables

	ICD	Stroke or TIA	PVD
<b>Social-economic status, reference group: low</b>			
Mid	-0.21 (-0.64, 0.22)	0.02 (-0.45, 0.49)	-0.56 (-1.19, 0.02)
High	-0.02 (-0.41, 0.37)	-0.29 (-0.77, 0.18)	-0.31 (-0.86, 0.20)
<b>Socio-demographic, anthropometric, lifestyle and health</b>			
Age, reference group: <60 years			
Age: 60-74 years	0.34 (0.19, 0.49)	0.75 (0.52, 0.99)	0.62 (0.37, 0.88)
Age: 75+ years	0.60 (0.42, 0.79)	1.11 (0.84, 1.39)	0.82 (0.52, 1.12)
Duration of diabetes, reference group: 0-3 years			
Duration: 4-9 years	-0.59 (-0.73, -0.45)	-0.31 (-0.50, -0.12)	0.13 (-0.09, 0.35)
Duration 10+ years	-0.63 (-0.80, -0.46)	-0.19 (-0.40, 0.03)	0.38 (0.14, 0.62)
Ethnicity, reference group: White			
South Asian	0.09 (-0.25, 0.41)	0.11 (-0.33, 0.52)	-0.80 (-1.54, -0.16)
Other Ethnicity	-0.67 (-1.53, 0.10)	-1.19 (-3.09, 0.14)	-0.06 (-1.13, 0.83)
Male			
	0.31 (0.19, 0.44)	-0.11 (-0.27, 0.05)	0.39 (0.20, 0.58)
Smoking status, reference: non-smoker			
Smoker	0.15 (-0.02, 0.33)	0.27 (0.04, 0.50)	0.93 (0.68, 1.18)
Ex-smoker	0.31 (0.18, 0.44)	0.14 (-0.04, 0.31)	0.37 (0.16, 0.58)
Obesity category, reference group: under and normal weight			
Overweight	-0.01 (-0.19, 0.17)	-0.10 (-0.31, 0.13)	-0.20 (-0.45, 0.05)
Obese	0.13 (-0.05, 0.31)	-0.20 (-0.42, 0.02)	-0.26 (-0.51, -0.01)
eGFR			
	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)
Hypertensive			
	-0.28 (-0.40, -0.16)	0.14 (-0.01, 0.30)	0.13 (-0.05, 0.31)
Cholesterol			
	-0.23 (-0.29, -0.17)	-0.09 (-0.16, -0.01)	-0.05 (-0.14, 0.03)
HbA1c			
	0.07 (0.03, 0.11)	0.02 (-0.03, 0.07)	0.01 (-0.04, 0.07)

<b>Interventions</b>			
Quality of care, reference group: Low			
Mid	-0.31 (-0.45, -0.17)	-0.16 (-0.35, 0.04)	-0.18 (-0.41, 0.05)
High	-0.40 (-0.56, -0.24)	-0.02 (-0.25, 0.19)	0.18 (-0.06, 0.42)
Diabetes treatment, reference group diet alone			
Metformin or sulphonylureas only	-0.28 (-0.42, -0.13)	-0.17 (-0.38, 0.04)	-0.06 (-0.32, 0.20)
Combination, with no insulin	-0.40 (-0.59, -0.21)	-0.29 (-0.56, -0.03)	-0.13 (-0.45, 0.18)
Insulin only	0.12 (-0.13, 0.36)	-0.07 (-0.38, 0.24)	0.33 (0.00, 0.67)
Combination with insulin	-0.31 (-0.6, -0.02)	-0.28 (-0.66, 0.09)	0.34 (-0.04, 0.71)
BP treatment, reference group: no treatment			
ACE inhibitors only	0.36 (0.00, 0.72)	0.2 (-0.24, 0.63)	0.44 (0.01, 0.87)
Combination, with ACEI	1.54 (1.29, 1.82)	0.06 (-0.27, 0.41)	0.28 (-0.08, 0.64)
Combination, no ACEI	1.28 (1.02, 1.57)	0.14 (-0.19, 0.48)	0.26 (-0.11, 0.63)
Aspirin	1.37 (1.26, 1.49)	1.06 (0.89, 1.23)	0.58 (0.40, 0.76)
Lipid therapy	0.61 (0.47, 0.74)	0.09 (-0.09, 0.26)	0.10 (-0.10, 0.30)
Shared care	0.28 (0.12, 0.44)	0.44 (0.24, 0.64)	0.84 (0.62, 1.06)
Middlesbrough PCT	-0.20 (-0.39, -0.02)	-0.12 (-0.38, 0.15)	-0.20 (-0.60, 0.19)
Visit year, reference group: 2000			
2001	-0.29 (-0.58, 0.02)	0.21 (-0.17, 0.60)	-0.23 (-0.65, 0.18)
2002	-0.30 (-0.56, -0.02)	0.04 (-0.33, 0.41)	-0.25 (-0.62, 0.14)
2003	-0.39 (-0.65, -0.12)	0.14 (-0.20, 0.51)	0.02 (-0.35, 0.40)
2004	-0.66 (-0.93, -0.39)	0.03 (-0.32, 0.40)	-0.02 (-0.38, 0.36)
2005	-1.24 (-1.52, -0.96)	-0.2 (-0.57, 0.18)	-0.31 (-0.70, 0.09)
2006	-1.67 (-1.96, -1.37)	-0.81 (-1.20, -0.41)	-0.80 (-1.21, -0.37)
2007	-1.81 (-2.10, -1.51)	-0.71 (-1.12, -0.30)	-1.14 (-1.62, -0.68)
<b>Interactions</b>			
BP treatments, reference group: No BP treatment & Low SES			
Mid SES*ACEI only	0.22 (-0.44, 0.87)	0.11 (-0.62, 0.84)	0.42 (-0.41, 1.24)
Mid SES*Combo. w. ACEI	-0.01 (-0.49, 0.47)	-0.02 (-0.59, 0.53)	0.48 (-0.19, 1.17)
Mid SES*Combo. no ACEI	0.03 (-0.47, 0.51)	-0.10 (-0.67, 0.46)	0.39 (-0.31, 1.11)
High SES*ACEI only	-0.54 (-1.16, 0.10)	0.27 (-0.44, 0.97)	-0.21 (-0.97, 0.57)
High SES*Combo w. ACEI	-0.11 (-0.55, 0.33)	0.39 (-0.16, 0.95)	0.19 (-0.40, 0.79)
High SES* Combo. no ACEI	-0.14 (-0.60, 0.31)	0.27 (-0.29, 0.83)	0.15 (-0.48, 0.79)
<b>Cons</b>	-3.88 (-5.00, -2.69)	-4.99 (-6.39, -3.83)	-5.80 (-7.32, -4.57)
<b>Variance estimate at:</b>			
<b>Practice level</b>	0.05 (0.02, 0.11)	0.11 (0.04, 0.21)	0.28 (0.14, 0.49)
<b>Patient level</b>	2.09 (0.53, 6.98)	1.36 (0.33, 4.57)	1.40 (0.34, 4.83)
<b>Bayesian DIC</b>	9201.75	6142.00	5030.83

Table 26: Saturated logistic regression multilevel models examining microalbuminuria and retinopathy rates with interaction effect between SES and BP treatments by 1999 to 2007, conditional on relevant explanatory variables

	Microalbuminuria	Retinopathy
<b>Social-economic status, reference group: low</b>		
Mid	-0.20 (-0.37, -0.02)	-0.12 (-0.37, 0.13)
High	-0.23 (-0.39, -0.07)	-0.12 (-0.34, 0.11)
<b>Covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	0.01 (-0.06, 0.09)	0.00 (-0.11, 0.11)
Age: 75+ years	0.37 (0.28, 0.47)	-0.11 (-0.25, 0.03)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	-0.01 (-0.09, 0.06)	0.47 (0.34, 0.59)
Duration 10+ years	0.18 (0.10, 0.27)	1.60 (1.47, 1.73)
Ethnicity, reference group: White		
South Asian	0.21 (0.04, 0.37)	-0.15 (-0.39, 0.08)
Other Ethnicity	0.29 (-0.09, 0.66)	0.52 (0.08, 0.94)
Male	0.24 (0.18, 0.31)	0.25 (0.16, 0.34)
Smoking status, reference: non-smoker		
Smoker	0.26 (0.16, 0.35)	-0.13 (-0.26, 0.01)
Ex-smoker	0.07 (0.00, 0.14)	-0.12 (-0.22, -0.02)
Obesity category, reference group: under and normal weight		
Overweight	-0.01 (-0.11, 0.09)	-0.09 (-0.22, 0.04)
Obese	0.06 (-0.04, 0.16)	-0.10 (-0.23, 0.03)
eGFR		-0.01 (-0.01, -0.01)
Hypertensive	0.18 (0.12, 0.25)	0.34 (0.25, 0.43)
Cholesterol	0.03 (0.00, 0.06)	-0.01 (-0.06, 0.02)
HbA1c	0.08 (0.06, 0.11)	0.05 (0.02, 0.08)
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.14 (-0.53, 0.29)	-0.29 (-1.96, 1.27)
High	-0.26 (-0.64, 0.18)	-0.35 (-2.02, 1.19)
Diabetes treatment, reference group diet alone		
Metformin/ sulphonylureas only	0.14 (0.05, 0.24)	0.40 (0.23, 0.56)
Combination, with no insulin	0.02 (-0.09, 0.14)	0.70 (0.53, 0.87)
Insulin only	0.18 (0.04, 0.32)	1.04 (0.85, 1.23)
Combination with insulin	0.20 (0.11, 0.30)	1.16 (0.96, 1.36)
BP treatments, reference group: No BP treatment		
ACE Inhibitors only	0.34 (0.19, 0.50)	0.22 (0.00, 0.44)
Combination with ACEI	0.48 (0.35, 0.60)	0.18 (0.00, 0.36)
Combination no ACEI	0.17 (0.05, 0.30)	0.15 (-0.03, 0.34)
Aspirin	0.08 (0.01, 0.14)	0.05 (-0.04, 0.14)
Lipid therapy	-0.05 (-0.12, 0.02)	-0.06 (-0.16, 0.04)
Shared care	-0.93 (-1.02, -0.84)	0.53 (0.42, 0.63)
Middlesbrough PCT	0.59 (0.28, 0.89)	-0.04 (-0.22, 0.13)
Visit year, reference group: 1999		
2000	-0.34 (-0.66, 0.02)	0.03 (-0.27, 0.32)
2001	-0.55 (-0.86, -0.21)	0.00 (-0.30, 0.29)
2002	-0.78 (-1.08, -0.45)	0.02 (-0.27, 0.29)
2003	-0.67 (-0.97, -0.34)	-0.10 (-0.38, 0.18)
2004	0.09 (-0.19, 0.42)	0.09 (-0.20, 0.37)
2005	0.29 (0.00, 0.61)	0.36 (0.06, 0.65)
2006	0.66 (0.37, 0.99)	-0.68 (-0.98, -0.38)
2007	0.36 (-0.06, 0.79)	0.38 (0.08, 0.67)
<b>Interactions</b>		
BP treatments, reference group: No BP treatment & Low SES		
Mid SES*ACEI only	0.10 (-0.18, 0.38)	-0.06 (-0.44, 0.31)

Mid SES*Comb. w. ACEI	0.08 (-0.14, 0.28)	0.11 (-0.18, 0.40)
Mid SES*Comb. no ACEI	0.22 (-0.01, 0.44)	0.02 (-0.29, 0.32)
High SES*ACEI only	0.00 (-0.26, 0.26)	<b>0.36 (0.03, 0.71)</b>
High SES*Comb. w. ACEI	0.12 (-0.08, 0.31)	0.04 (-0.23, 0.31)
High SES* Comb. no ACEI	<b>0.33 (0.12, 0.53)</b>	-0.11 (-0.40, 0.18)
<b>Cons</b>	<b>-2.35 (-2.90, -1.73)</b>	<b>-2.70 (-4.24, -1.11)</b>
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.21 (0.13, 0.34)	0.05 (0.03, 0.09)
<b>Patient level</b>	0.01 (0.00, 0.03)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	25466.08	14525.92

## Antithrombotic and Lipid profile treatments

In chapter 6, both prescriptions of aspirin and lipid therapies were statistically significant predictors of lower cholesterol levels; this was contrary to expectation of diabetes treatments, which was associated with poorer HbA1c (Table 9). In the models with long-term complications as the dependent variable, aspirin was a significant predictor of incidences of ICD, stroke or TIA, PVD and microalbuminuria and lipid therapies was a significant predictor of ICD. The graphical analyses in chapter 5 showed that, in general, there were some significant differences in these treatments over time with one year in each figure displaying statistically significant differences (Figure 32 and Figure 33). However, once other variables and the structured of the data were taken into account, no differences were found between either variable by SES overall or over time. In contrast, the results here show that there was some evidence of statistically significant differences between these treatments and health outcomes by SES.

The results in Table 27 shows that mid SES patients prescribed aspirin were significantly more likely to have lower cholesterol levels compared to low SES patients, however, this treatment was not associated with differences with long-term complications by SES (Table 28 and Table 29). In Table 30 the results show that mid and high SES patients prescribed lipid therapies were significantly more likely to have lower cholesterol levels compared to low SES. Like with aspirin, the results in Table 31 and Table 32 show that there were no significant interaction effects between SES and lipid therapies associated with long-term complications, with one exception. Mid SES patients prescribed lipid therapies were significantly more likely to have more favourable microalbuminuria rates compared to low SES patients.

Table 27: Saturated linear regression multilevel models examining cholesterol levels with interaction effect between SES and aspirin by 1999 to 2007, conditional on relevant explanatory variables

<b>Cholesterol</b>	
<b>Social-economic status, reference group: low</b>	
Mid	0.06 (0.03, 0.10)
High	0.02 (-0.02, 0.05)
<b>Socio-demographic, anthropometric, lifestyle and health</b>	
Age, reference group: <60 years	
Age: 60-74 years	-0.20 (-0.23, -0.17)
Age: 75+ years	-0.26 (-0.30, -0.23)
Duration of diabetes, reference group: 0-3 years	
Duration: 4-9 years	-0.09 (-0.11, -0.06)
Duration 10+ years	-0.13 (-0.16, -0.10)
Ethnicity, reference group: White	
South Asian	-0.08 (-0.14, -0.02)
Other Ethnicity	0.08 (-0.04, 0.21)
Male	-0.34 (-0.36, -0.32)
Smoking status, reference: non-smoker	
Smoker	0.08 (0.04, 0.11)
Ex-smoker	0.00 (-0.03, 0.02)
Obesity category, reference group: under and normal weight	
Overweight	0.03 (0.00, 0.07)
Obese	0.03 (0.00, 0.07)
Hypertensive	0.14 (0.12, 0.16)
Ischaemic Cardiac	-0.13 (-0.15, -0.10)
Stroke or TIA	-0.02 (-0.06, 0.01)
PVD	0.01 (-0.03, 0.05)
<b>Interventions</b>	
Quality of care, reference group: Low	
Mid	-0.10 (-0.13, -0.08)
High	-0.15 (-0.18, -0.11)
Aspirin	-0.06 (-0.09, -0.02)
Lipid therapy	-0.28 (-0.31, -0.26)
Middlesbrough PCT	-0.03 (-0.09, 0.04)
Shared care	-0.07 (-0.10, -0.04)
Visit year, reference group: 1999	
2000	-0.20 (-0.29, -0.11)
2001	-0.21 (-0.30, -0.12)
2002	-0.22 (-0.30, -0.14)
2003	-0.37 (-0.46, -0.29)
2004	-0.58 (-0.66, -0.50)
2005	-0.73 (-0.82, -0.65)
2006	-0.83 (-0.92, -0.75)
2007	-0.93 (-1.02, -0.85)
<b>Interactions</b>	
Quality of care, reference group: Low SES & Aspirin	
Mid SES*Aspirin	-0.07 (-0.13, -0.01)
High SES*Aspirin	-0.05 (-0.10, 0.00)
<b>Cons</b>	5.90 (5.80, 6.01)
<b>Variance estimate at:</b>	
<b>Practice level</b>	0.01 (0.00, 0.01)
<b>Patient level</b>	0.00 (0.00, 0.01)
<b>Visit year</b>	1.14 (1.13, 1.16)
<b>Bayesian DIC</b>	110332.92

Table 28: Saturated logistic regression multilevel models examining incidences in ICD, stroke or TIA and PVD with interaction effect between SES and aspirin by 2000 to 2007, conditional on relevant explanatory variables

	ICD	Stroke or TIA	PVD
<b>Social-economic status, reference group: low</b>			
Mid	-0.13 (-0.37, 0.10)	0.12 (-0.22, 0.44)	-0.08 (-0.43, 0.26)
High	-0.13 (-0.35, 0.08)	0.02 (-0.29, 0.32)	0.02 (-0.31, 0.33)
<b>Socio-demographic, anthropometric, lifestyle and health</b>			
Age, reference group: <60 years			
Age: 60-74 years	0.34 (0.19, 0.49)	0.73 (0.49, 0.97)	0.61 (0.37, 0.87)
Age: 75+ years	0.60 (0.42, 0.79)	1.09 (0.81, 1.36)	0.81 (0.50, 1.11)
Duration of diabetes, reference group: 0-3 years			
Duration: 4-9 years	-0.59 (-0.73, -0.45)	-0.31 (-0.50, -0.11)	0.12 (-0.10, 0.34)
Duration 10+ years	-0.62 (-0.79, -0.46)	-0.18 (-0.40, 0.03)	0.38 (0.14, 0.62)
Ethnicity, reference group: White			
South Asian	0.09 (-0.25, 0.42)	0.11 (-0.32, 0.53)	-0.80 (-1.53, -0.15)
Other Ethnicity	-0.67 (-1.54, 0.09)	-1.16 (-3.04, 0.19)	-0.03 (-1.08, 0.86)
Male	0.32 (0.20, 0.44)	-0.11 (-0.28, 0.05)	0.39 (0.20, 0.58)
Smoking status, reference: non-smoker			
Smoker	0.15 (-0.02, 0.33)	0.28 (0.05, 0.51)	0.93 (0.68, 1.19)
Ex-smoker	0.31 (0.19, 0.44)	0.14 (-0.04, 0.31)	0.37 (0.16, 0.59)
Obesity category, reference group: under and normal weight			
Overweight	-0.02 (-0.20, 0.16)	-0.09 (-0.30, 0.12)	-0.21 (-0.45, 0.04)
Obese	0.12 (-0.06, 0.30)	-0.19 (-0.41, 0.02)	-0.26 (-0.50, -0.01)
eGFR	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)	-0.01 (-0.02, 0.00)
Hypertensive	-0.27 (-0.39, -0.16)	0.14 (-0.01, 0.30)	0.13 (-0.05, 0.31)
Cholesterol	-0.23 (-0.28, -0.17)	-0.09 (-0.17, -0.02)	-0.06 (-0.14, 0.03)
HbA1c	0.07 (0.03, 0.11)	0.02 (-0.04, 0.07)	0.01 (-0.05, 0.07)
<b>Interventions</b>			
Quality of care, reference group: Low			
Mid	-0.30 (-0.44, -0.16)	-0.16 (-0.35, 0.04)	-0.18 (-0.41, 0.06)
High	-0.40 (-0.56, -0.23)	-0.03 (-0.24, 0.19)	0.18 (-0.06, 0.43)
Diabetes treatment, reference group diet alone			
Metformin/ sulphonylureas only	-0.28 (-0.43, -0.14)	-0.18 (-0.38, 0.03)	-0.06 (-0.32, 0.20)
Combination, with no insulin	-0.41 (-0.60, -0.21)	-0.30 (-0.56, -0.03)	-0.12 (-0.43, 0.20)
Insulin only	0.11 (-0.13, 0.35)	-0.08 (-0.39, 0.23)	0.33 (-0.01, 0.65)
Combination with insulin	-0.32 (-0.61, -0.03)	-0.28 (-0.66, 0.09)	0.34 (-0.04, 0.72)
BP treatment, reference group: no treatment			
ACE inhibitors only	0.27 (0.01, 0.52)	0.30 (0.00, 0.59)	0.48 (0.15, 0.80)
Combination, with ACEI	1.51 (1.32, 1.70)	0.16 (-0.08, 0.40)	0.42 (0.15, 0.70)
Combination, no ACEI	1.25 (1.06, 1.44)	0.18 (-0.06, 0.42)	0.37 (0.09, 0.65)
Aspirin	1.40 (1.24, 1.57)	1.12 (0.89, 1.36)	0.72 (0.47, 0.98)
Lipid therapy	0.61 (0.47, 0.74)	0.09 (-0.09, 0.26)	0.09 (-0.11, 0.29)
Shared care	0.28 (0.12, 0.44)	0.44 (0.23, 0.64)	0.84 (0.62, 1.06)
Middlesbrough PCT	-0.20 (-0.39, -0.01)	-0.13 (-0.40, 0.14)	-0.20 (-0.59, 0.18)
Visit year, reference group: 2000			
2001	-0.28 (-0.58, 0.02)	0.20 (-0.18, 0.59)	-0.23 (-0.65, 0.19)
2002	-0.30 (-0.57, -0.02)	0.02 (-0.34, 0.40)	-0.25 (-0.63, 0.14)
2003	-0.39 (-0.65, -0.13)	0.13 (-0.22, 0.49)	0.02 (-0.33, 0.40)
2004	-0.66 (-0.93, -0.39)	0.02 (-0.34, 0.38)	-0.02 (-0.37, 0.37)
2005	-1.24 (-1.53, -0.95)	-0.21 (-0.57, 0.16)	-0.31 (-0.69, 0.09)
2006	-1.67 (-1.96, -1.37)	-0.83 (-1.22, -0.42)	-0.80 (-1.21, -0.38)
2007	-1.80 (-2.11, -1.51)	-0.73 (-1.12, -0.32)	-1.14 (-1.61, -0.67)
<b>Interactions</b>			
Aspirin, reference group: Low SES & Aspirin			
Mid SES*Aspirin	-0.08 (-0.37, 0.20)	-0.19 (-0.58, 0.20)	-0.16 (-0.58, 0.27)
High SES*Aspirin	-0.04 (-0.31, 0.23)	-0.05 (-0.41, 0.33)	-0.39 (-0.80, 0.02)

<b>Cons</b>	-4.18 (-5.76, -3.07)	-4.96 (-6.37, -3.76)	-5.87 (-7.23, -4.52)
<b>Variance estimate at:</b>			
<b>Practice level</b>	0.05 (0.02, 0.11)	0.11 (0.04, 0.21)	0.28 (0.14, 0.49)
<b>Patient level</b>	2.32 (0.56, 7.71)	1.40 (0.33, 4.81)	1.41 (0.34, 4.63)
<b>Bayesian DIC</b>	9198.05	6135.98	5023.44

Table 29: Saturated logistic regression multilevel models examining microalbuminuria and retinopathy rates with interaction effect between SES and aspirin by 1999 to 2007, conditional on relevant explanatory variables

	<b>Microalbuminuria</b>	<b>Retinopathy</b>
<b>Social-economic status, reference group: low</b>		
Mid	-0.12 (-0.22, -0.01)	-0.03 (-0.19, 0.12)
High	-0.12 (-0.22, -0.02)	-0.04 (-0.18, 0.10)
<b>Socio-demographic, anthropometric, lifestyle and health</b>		
Age, reference group: <60 years		
Age: 60-74 years	0.01 (-0.07, 0.09)	0.01 (-0.11, 0.11)
Age: 75+ years	0.37 (0.28, 0.47)	-0.10 (-0.25, 0.04)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	-0.01 (-0.09, 0.06)	0.47 (0.34, 0.59)
Duration 10+ years	0.18 (0.09, 0.27)	1.60 (1.47, 1.73)
Ethnicity, reference group: White		
South Asian	0.21 (0.05, 0.38)	-0.15 (-0.39, 0.07)
Other Ethnicity	0.29 (-0.08, 0.67)	0.52 (0.08, 0.94)
Male	0.24 (0.18, 0.31)	0.25 (0.16, 0.34)
Smoking status, reference: non-smoker		
Smoker	0.26 (0.17, 0.36)	-0.13 (-0.27, 0.00)
Ex-smoker	0.07 (0.00, 0.14)	-0.12 (-0.22, -0.02)
Obesity category, reference group: under and normal weight		
Overweight	-0.01 (-0.11, 0.08)	-0.09 (-0.22, 0.04)
Obese	0.06 (-0.04, 0.15)	-0.10 (-0.23, 0.03)
eGFR		-0.01 (-0.01, -0.01)
Hypertensive	0.18 (0.11, 0.25)	0.33 (0.24, 0.42)
Cholesterol	0.03 (0.00, 0.06)	-0.02 (-0.06, 0.03)
HbA1c	0.08 (0.06, 0.11)	0.05 (0.02, 0.08)
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.15 (-0.54, 0.29)	-0.05 (-1.25, 1.82)
High	-0.26 (-0.67, 0.18)	-0.12 (-1.31, 1.78)
Diabetes treatment, reference group diet alone		
Metformin/sulphonylureas only	0.14 (0.04, 0.24)	0.40 (0.24, 0.56)
Combination, with no insulin	0.02 (-0.10, 0.13)	0.70 (0.53, 0.87)
Insulin only	0.18 (0.04, 0.31)	1.04 (0.85, 1.23)
Combination with insulin	0.20 (0.10, 0.30)	1.16 (0.96, 1.36)
BP treatments, reference group: No BP treatment		
ACE Inhibitors only	0.37 (0.26, 0.48)	0.31 (0.16, 0.46)
Combination with ACEI	0.53 (0.44, 0.62)	0.22 (0.09, 0.34)
Combination no ACEI	0.32 (0.23, 0.41)	0.12 (0.00, 0.26)
Aspirin	0.05 (-0.05, 0.14)	0.09 (-0.03, 0.22)
Lipid therapy	-0.05 (-0.12, 0.02)	-0.06 (-0.16, 0.04)
Shared care	-0.93 (-1.01, -0.84)	0.53 (0.42, 0.64)
Middlesbrough PCT	0.58 (0.27, 0.86)	-0.04 (-0.22, 0.14)
Visit year, reference group: 1999		
2000	-0.44 (-0.79, -0.08)	0.04 (-0.27, 0.34)
2001	-0.65 (-0.98, -0.30)	0.01 (-0.30, 0.31)
2002	-0.88 (-1.21, -0.54)	0.03 (-0.26, 0.32)
2003	-0.77 (-1.09, -0.43)	-0.08 (-0.39, 0.21)

2004	-0.01 (-0.33, 0.32)	0.10 (-0.20, 0.40)
2005	0.17 (-0.15, 0.51)	0.37 (0.06, 0.67)
2006	0.55 (0.22, 0.88)	-0.67 (-0.99, -0.36)
2007	0.24 (-0.22, 0.69)	0.39 (0.09, 0.70)
<b>Interactions</b>		
Aspirin, reference group: Low SES & Aspirin		
Mid SES*Aspirin	0.05 (-0.10, 0.21)	-0.10 (-0.30, 0.11)
High SES* Aspirin	0.06 (-0.08, 0.20)	-0.06 (-0.26, 0.14)
<b>Cons</b>	-2.37 (-3.01, -1.71)	-3.00 (-4.71, -1.70)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.21 (0.13, 0.34)	0.05 (0.03, 0.10)
<b>Patient level</b>	0.01 (0.00, 0.03)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	25471.46	14529.81

Table 30: Saturated linear regression multilevel models examining cholesterol levels with interaction effect between SES and lipid therapies by 1999 to 2007, conditional on relevant explanatory variables

	<b>Cholesterol</b>	
<b>Social-economic status, reference group: low</b>		
Mid	0.10 (0.06, 0.15)	
High	0.06 (0.02, 0.11)	
<b>Socio-demographic, anthropometric, lifestyle and health</b>		
Age, reference group: <60 years		
Age: 60-74 years	-0.20 (-0.22, -0.17)	
Age: 75+ years	-0.26 (-0.30, -0.23)	
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	-0.09 (-0.11, -0.06)	
Duration 10+ years	-0.14 (-0.17, -0.10)	
Ethnicity, reference group: White		
South Asian	-0.08 (-0.14, -0.02)	
Other Ethnicity	0.08 (-0.04, 0.21)	
Male	-0.34 (-0.36, -0.32)	
Smoking status, reference: non-smoker		
Smoker	0.08 (0.04, 0.11)	
Ex-smoker	0.00 (-0.03, 0.02)	
Obesity category, reference group: under and normal weight		
Overweight	0.03 (0.00, 0.07)	
Obese	0.03 (0.00, 0.06)	
Hypertensive	0.14 (0.12, 0.16)	
Ischaemic Cardiac	-0.13 (-0.15, -0.10)	
Stroke or TIA	-0.02 (-0.06, 0.01)	
PVD	0.01 (-0.03, 0.05)	
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.10 (-0.13, -0.08)	
High	-0.15 (-0.18, -0.11)	
Aspirin	-0.09 (-0.11, -0.06)	
Lipid therapy	-0.22 (-0.26, -0.19)	
Middlesbrough PCT	-0.03 (-0.09, 0.03)	
Shared care	-0.07 (-0.10, -0.04)	
Visit year, reference group: 1999		
2000	-0.20 (-0.29, -0.11)	
2001	-0.21 (-0.29, -0.12)	
2002	-0.22 (-0.30, -0.13)	
2003	-0.37 (-0.46, -0.29)	
2004	-0.58 (-0.66, -0.50)	

2005	-0.73 (-0.82, -0.65)
2006	-0.83 (-0.92, -0.75)
2007	-0.93 (-1.02, -0.85)
<b>Interactions</b>	
Lipid therapy, reference group: Low SES & Lipid therapy	
Mid SES* Lipid therapy	-0.11 (-0.17, -0.06)
High SES* Lipid therapy	-0.11 (-0.16, -0.06)
<b>Cons</b>	5.88 (5.77, 5.99)
<b>Variance estimate at:</b>	
<b>Practice level</b>	0.01 (0.00, 0.01)
<b>Patient level</b>	0.00 (0.00, 0.01)
<b>Visit year</b>	1.14 (1.13, 1.16)
<b>Bayesian DIC</b>	110316.16

Table 31: Saturated logistic regression multilevel models incidences of ICD, stroke or TIA and PVD with interaction effect between SES and lipid therapies by 2000 to 2007, conditional on relevant explanatory variables

	ICD	Stroke or TIA	PVD
<b>Social-economic status, reference group: low</b>			
Mid	-0.20 (-0.46, 0.05)	0.02 (-0.29, 0.33)	-0.06 (-0.40, 0.27)
High	-0.20 (-0.45, 0.04)	-0.12 (-0.42, 0.19)	-0.20 (-0.55, 0.13)
<b>Socio-demographic, anthropometric, lifestyle and health</b>			
Age, reference group: <60 years			
Age: 60-74 years	0.34 (0.18, 0.49)	0.73 (0.50, 0.97)	0.61 (0.37, 0.86)
Age: 75+ years	0.60 (0.41, 0.79)	1.08 (0.81, 1.36)	0.80 (0.51, 1.09)
Duration of diabetes, reference group: 0-3 years			
Duration: 4-9 years	-0.59 (-0.73, -0.45)	-0.31 (-0.50, -0.12)	0.13 (-0.09, 0.35)
Duration 10+ years	-0.62 (-0.79, -0.46)	-0.18 (-0.40, 0.03)	0.38 (0.14, 0.61)
Ethnicity, reference group: White			
South Asian	0.09 (-0.25, 0.41)	0.11 (-0.33, 0.52)	-0.8 (-1.53, -0.16)
Other Ethnicity	-0.67 (-1.58, 0.10)	-1.16 (-3.02, 0.18)	-0.04 (-1.10, 0.83)
Male	0.32 (0.20, 0.45)	-0.11 (-0.28, 0.05)	0.39 (0.20, 0.58)
Smoking status, reference: non-smoker			
Smoker	0.16 (-0.02, 0.33)	0.28 (0.05, 0.50)	0.93 (0.69, 1.18)
Ex-smoker	0.31 (0.19, 0.44)	0.14 (-0.04, 0.30)	0.37 (0.16, 0.58)
Obesity category, reference group: under and normal weight			
Overweight	-0.01 (-0.19, 0.16)	-0.09 (-0.30, 0.13)	-0.21 (-0.46, 0.04)
Obese	0.12 (-0.05, 0.29)	-0.19 (-0.41, 0.03)	-0.26 (-0.52, -0.01)
eGFR	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)
Hypertensive	-0.27 (-0.39, -0.16)	0.14 (-0.01, 0.30)	0.13 (-0.05, 0.31)
Cholesterol	-0.23 (-0.28, -0.16)	-0.09 (-0.17, -0.02)	-0.06 (-0.14, 0.02)
HbA1c	0.07 (0.03, 0.11)	0.02 (-0.04, 0.07)	0.01 (-0.05, 0.07)
<b>Interventions</b>			
Quality of care, reference group: Low			
Mid	-0.30 (-0.44, -0.17)	-0.16 (-0.36, 0.04)	-0.18 (-0.41, 0.07)
High	-0.40 (-0.56, -0.24)	-0.03 (-0.24, 0.20)	0.18 (-0.06, 0.42)
Diabetes treatment, reference group diet alone			
Metformin/sulphonylureas only	-0.28 (-0.43, -0.14)	-0.18 (-0.38, 0.03)	-0.06 (-0.32, 0.19)
Combination, with no insulin	-0.40 (-0.60, -0.21)	-0.30 (-0.56, -0.03)	-0.12 (-0.44, 0.20)
Insulin only	0.11 (-0.13, 0.35)	-0.08 (-0.40, 0.23)	0.33 (-0.01, 0.66)
Combination with insulin	-0.32 (-0.61, -0.03)	-0.29 (-0.68, 0.09)	0.33 (-0.05, 0.71)
BP treatment, reference group: no treatment			
ACE inhibitors only	0.27 (0.01, 0.53)	0.30 (0.01, 0.58)	0.48 (0.16, 0.80)
Combination, with ACEI	1.50 (1.31, 1.70)	0.15 (-0.08, 0.39)	0.42 (0.15, 0.70)
Combination, no ACEI	1.25 (1.05, 1.45)	0.18 (-0.06, 0.41)	0.37 (0.09, 0.65)
Aspirin	1.37 (1.26, 1.49)	1.06 (0.89, 1.23)	0.57 (0.39, 0.76)

Lipid therapy	0.58 (0.40, 0.77)	0.06 (-0.17, 0.30)	0.15 (-0.12, 0.42)
Shared care	0.28 (0.12, 0.43)	0.44 (0.24, 0.63)	0.84 (0.62, 1.05)
Middlesbrough PCT	-0.20 (-0.38, 0.00)	-0.12 (-0.39, 0.14)	-0.19 (-0.57, 0.20)
Visit year, reference group: 2000			
2001	-0.28 (-0.57, 0.02)	0.20 (-0.19, 0.60)	-0.24 (-0.65, 0.17)
2002	-0.30 (-0.56, -0.02)	0.02 (-0.34, 0.40)	-0.26 (-0.64, 0.12)
2003	-0.39 (-0.65, -0.11)	0.13 (-0.22, 0.50)	0.01 (-0.33, 0.38)
2004	-0.66 (-0.92, -0.38)	0.02 (-0.34, 0.38)	-0.03 (-0.38, 0.34)
2005	-1.23 (-1.51, -0.94)	-0.21 (-0.58, 0.16)	-0.32 (-0.69, 0.06)
2006	-1.66 (-1.95, -1.36)	-0.82 (-1.22, -0.42)	-0.81 (-1.21, -0.41)
2007	-1.80 (-2.10, -1.50)	-0.73 (-1.13, -0.32)	-1.15 (-1.61, -0.70)
<b>Interactions</b>			
Lipid therapy, reference group: Low SES & Lipid therapy			
Mid SES*Lipid therapy	0.03 (-0.28, 0.33)	-0.06 (-0.44, 0.33)	-0.18 (-0.61, 0.24)
High SES*Lipid therapy	0.06 (-0.21, 0.35)	0.15 (-0.21, 0.52)	-0.02 (-0.42, 0.40)
<b>Cons</b>	-3.98 (-5.26, -2.61)	-4.85 (-6.13, -3.63)	-5.83 (-6.96, -4.51)
<b>Variance estimate at:</b>			
<b>Practice level</b>	0.05 (0.02, 0.11)	0.11 (0.04, 0.22)	0.28 (0.14, 0.48)
<b>Patient level</b>	2.18 (0.54, 7.22)	1.35 (0.32, 4.88)	1.41 (0.33, 4.95)
<b>Bayesian DIC</b>	9199.07	6136.29	5025.41

Table 32: Saturated logistic regression multilevel models microalbuminuria and retinopathy rates with interaction effect between SES and lipid therapies by 1999 to 2007, conditional on relevant explanatory variables

	Microalbuminuria	Retinopathy
<b>Social-economic status, reference group: low</b>		
Mid	0.08 (-0.05, 0.21)	-0.15 (-0.32, 0.03)
High	-0.02 (-0.15, 0.11)	-0.12 (-0.28, 0.04)
<b>Socio-demographic, anthropometric, lifestyle and health</b>		
Age, reference group: <60 years		
Age: 60-74 years	0.01 (-0.06, 0.09)	0.00 (-0.11, 0.11)
Age: 75+ years	0.38 (0.28, 0.47)	-0.11 (-0.25, 0.04)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	-0.01 (-0.09, 0.06)	0.47 (0.34, 0.59)
Duration 10+ years	0.18 (0.09, 0.27)	1.60 (1.47, 1.72)
Ethnicity, reference group: White		
South Asian	0.22 (0.05, 0.38)	-0.15 (-0.39, 0.08)
Other Ethnicity	0.31 (-0.07, 0.67)	0.52 (0.08, 0.96)
Male	0.24 (0.18, 0.31)	0.25 (0.16, 0.34)
Smoking status, reference: non-smoker		
Smoker	0.26 (0.17, 0.36)	-0.13 (-0.27, 0.00)
Ex-smoker	0.07 (0.00, 0.14)	-0.12 (-0.22, -0.03)
Obesity category, reference group: under and normal weight		
Overweight	-0.01 (-0.10, 0.09)	-0.09 (-0.22, 0.04)
Obese	0.06 (-0.03, 0.16)	-0.10 (-0.23, 0.03)
eGFR		-0.01 (-0.01, -0.01)
Hypertensive	0.18 (0.11, 0.24)	0.33 (0.25, 0.42)
Cholesterol	0.03 (0.00, 0.06)	-0.01 (-0.05, 0.03)
HbA1c	0.09 (0.06, 0.11)	0.05 (0.02, 0.08)
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.09 (-0.48, 0.34)	-0.43 (-1.75, 0.85)
High	-0.20 (-0.60, 0.22)	-0.49 (-1.81, 0.77)
Diabetes treatment, reference group diet alone		
Metformin/sulphonylureas only	0.14 (0.04, 0.24)	0.40 (0.24, 0.56)
Combination, with no insulin	0.02 (-0.09, 0.14)	0.70 (0.52, 0.87)

Insulin only	0.18 (0.04, 0.31)	1.04 (0.86, 1.23)
Combination with insulin	0.20 (0.10, 0.30)	1.16 (0.96, 1.36)
BP treatments, reference group: No BP treatment		
ACE Inhibitors only	0.36 (0.25, 0.47)	0.32 (0.17, 0.47)
Combination with ACEI	0.53 (0.44, 0.62)	0.22 (0.09, 0.34)
Combination no ACEI	0.32 (0.23, 0.41)	0.13 (0.00, 0.26)
Lipid therapy	0.04 (-0.06, 0.14)	-0.11 (-0.25, 0.03)
Aspirin	0.07 (0.01, 0.14)	0.05 (-0.04, 0.15)
Shared care	-0.92 (-1.01, -0.83)	0.53 (0.42, 0.64)
Middlesbrough PCT	0.60 (0.32, 0.88)	-0.04 (-0.22, 0.14)
Visit year, reference group: 1999		
2000	-0.41 (-0.74, -0.10)	0.04 (-0.25, 0.34)
2001	-0.63 (-0.93, -0.33)	0.01 (-0.29, 0.30)
2002	-0.86 (-1.16, -0.56)	0.03 (-0.26, 0.31)
2003	-0.75 (-1.04, -0.46)	-0.08 (-0.37, 0.21)
2004	0.01 (-0.28, 0.28)	0.10 (-0.19, 0.39)
2005	0.19 (-0.10, 0.47)	0.37 (0.07, 0.67)
2006	0.57 (0.28, 0.84)	-0.66 (-0.96, -0.36)
2007	0.27 (-0.15, 0.68)	0.39 (0.10, 0.68)
<b>Interactions</b>		
Lipid therapy, reference group: Low SES & Lipid therapy		
Mid SES*Lipid therapy	-0.25 (-0.41, -0.09)	0.12 (-0.09, 0.33)
High SES*Lipid therapy	-0.11 (-0.26, 0.04)	0.08 (-0.13, 0.29)
<b>Cons</b>	-2.55 (-3.22, -1.98)	-2.57 (-3.88, -1.19)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.21 (0.13, 0.34)	0.05 (0.03, 0.10)
<b>Patient level</b>	0.01 (0.00, 0.03)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	25462.27	14529.00

## Shared care

In chapter 6, shared care was consistently a statistically significant predictor of health outcomes in all the multilevel models. With the exception of cholesterol levels, shared care was associated with poorer health outcomes. As stated previously, these results were in general to be expected as referrals to specialist care should be for patients with poor control and complex needs. The graphical analyses of shared care by SES over time in chapter five revealed that it was high and low SES patients who had higher rates of receiving shared compared to mid SES patients, this pattern achieved statistical significance in a number of years (Figure 34). In contrast, when patients health statuses and other variables were taken into account the results from the multilevel analyses showed evidence that high SES patients were more likely to receive shared care over time compared to low SES patients (Table 17).

The results from this section indicate that in most circumstances there were no differences in the association between shared care and health outcomes by SES. However, there were two exceptions: high SES patients receiving shared care were significantly more likely to have lower HbA1c levels and microalbuminuria rates compared to low SES patients receiving shared care. In contrast, mid SES patients receiving shared were significantly more likely to have higher rates of microalbuminuria compared to low SES patients receiving shared care.

Table 33: Saturated linear regression multilevel models examining cholesterol levels with interaction effect between SES and lipid therapies by 1999 to 2007, conditional on relevant explanatory variables

	HbA1c	Cholesterol
<b>Social-economic status, reference group: low</b>		
Mid	-0.03 (-0.07, 0.01)	0.04 (0.01, 0.08)
High	-0.05 (-0.10, -0.01)	0.01 (-0.02, 0.04)
<b>Socio-demographic, anthropometric, lifestyle and health</b>		
Age, reference group: <60 years		
Age: 60-74 years	-0.33 (-0.36, -0.30)	-0.20 (-0.23, -0.17)
Age: 75+ years	-0.41 (-0.46, -0.37)	-0.26 (-0.30, -0.23)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	0.06 (0.02, 0.09)	-0.09 (-0.11, -0.06)
Duration 10+ years	0.06 (0.02, 0.10)	-0.13 (-0.16, -0.10)
Ethnicity, reference group: White		
South Asian	0.46 (0.39, 0.54)	-0.08 (-0.14, -0.03)
Other Ethnicity	0.47 (0.30, 0.63)	0.08 (-0.05, 0.20)
Male	-0.06 (-0.09, -0.03)	-0.34 (-0.36, -0.32)
Smoking status, reference: non-smoker		
Smoker	0.23 (0.19, 0.27)	0.08 (0.04, 0.11)
E*-smoker	0.05 (0.02, 0.08)	0.00 (-0.03, 0.02)
Obesity category, reference group: under and normal weight		

Overweight	0.03 (-0.02, 0.07)	0.03 (0.00, 0.07)
Obese	0.08 (0.04, 0.13)	0.03 (0.00, 0.07)
Creatinine > 300	-0.81 (-1.06, -0.56)	
Hypertensive	0.10 (0.07, 0.13)	0.14 (0.12, 0.16)
Ischaemic Cardiac	0.00 (-0.04, 0.03)	-0.13 (-0.15, -0.10)
Stroke or TIA	-0.06 (-0.10, -0.01)	-0.02 (-0.06, 0.01)
PVD	-0.07 (-0.12, -0.01)	0.01 (-0.03, 0.05)
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.13 (-0.16, -0.09)	-0.10 (-0.13, -0.08)
High	-0.15 (-0.19, -0.11)	-0.15 (-0.18, -0.11)
Diabetes treatment, reference group diet alone		
Metformin/sulphonylureas only	0.81 (0.77, 0.85)	
Combination, with no insulin	1.25 (1.20, 1.29)	
Insulin only	1.67 (1.61, 1.73)	
Combination with insulin	1.75 (1.69, 1.82)	
Aspirin		-0.09 (-0.11, -0.06)
Lipid therapy		-0.28 (-0.31, -0.26)
Middlesbrough PCT	0.10 (0.01, 0.21)	-0.03 (-0.09, 0.03)
Shared care	0.22 (0.17, 0.26)	-0.05 (-0.08, -0.01)
Visit year, reference group: 1999		
2000	-0.32 (-0.40, -0.23)	-0.20 (-0.29, -0.11)
2001	-0.47 (-0.55, -0.39)	-0.21 (-0.30, -0.12)
2002	-0.55 (-0.63, -0.47)	-0.22 (-0.30, -0.13)
2003	-0.59 (-0.67, -0.52)	-0.37 (-0.45, -0.29)
2004	-0.63 (-0.71, -0.56)	-0.58 (-0.66, -0.49)
2005	-0.71 (-0.79, -0.64)	-0.73 (-0.82, -0.65)
2006	-1.18 (-1.25, -1.10)	-0.83 (-0.92, -0.75)
2007	-1.12 (-1.20, -1.04)	-0.93 (-1.02, -0.85)
<b>Interactions</b>		
Shared care, reference group: Low SES & Shared care		
Mid SES*Shared care	-0.05 (-0.13, 0.03)	-0.04 (-0.10, 0.03)
High SES*Shared care	-0.13 (-0.20, -0.05)	-0.06 (-0.12, 0.00)
<b>Cons</b>	7.58 (7.46, 7.70)	5.91 (5.8, 6.02)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.02 (0.01, 0.04)	0.01 (0.00, 0.01)
<b>Patient level</b>	0.00 (0.00, 0.02)	0.00 (0.00, 0.01)
<b>Visit year</b>	1.91 (1.88, 1.94)	1.14 (1.13, 1.16)
<b>Bayesian DIC</b>	133966.20	110335.80

Table 34: Saturated logistic regression multilevel models incidences of ICD, stroke or TIA and PVD with interaction effect between SES and lipid therapies by 2000 to 2007, conditional on relevant explanatory variables

	ICD	Stroke or TIA	PVD
<b>Social-economic status, reference group: low</b>			
Mid	-0.19 (-0.35, -0.01)	0.09 (-0.15, 0.33)	-0.20 (-0.50, 0.09)
High	-0.12 (-0.27, 0.05)	0.09 (-0.13, 0.32)	-0.07 (-0.35, 0.22)
<b>Covariates</b>			
Age, reference group: <60 years			
Age: 60-74 years	0.33 (0.19, 0.48)	0.74 (0.51, 0.98)	0.62 (0.38, 0.87)
Age: 75+ years	0.60 (0.41, 0.79)	1.10 (0.83, 1.38)	0.82 (0.52, 1.12)
Duration of diabetes, reference group: 0-3 years			
Duration: 4-9 years	-0.59 (-0.73, -0.45)	-0.31 (-0.50, -0.12)	0.13 (-0.10, 0.35)
Duration 10+ years	-0.63 (-0.80, -0.46)	-0.18 (-0.39, 0.03)	0.38 (0.15, 0.62)
Ethnicity, reference group: White			

South Asian	0.09 (-0.25, 0.41)	0.11 (-0.33, 0.53)	-0.81 (-1.52, -0.18)
Other Ethnicity	-0.67 (-1.55, 0.09)	-1.18 (-3.11, 0.16)	-0.07 (-1.10, 0.82)
Male	0.32 (0.20, 0.44)	-0.11 (-0.27, 0.06)	0.39 (0.20, 0.58)
Smoking status, reference: non-smoker			
Smoker	0.15 (-0.02, 0.33)	0.27 (0.05, 0.50)	0.93 (0.68, 1.18)
Ex-smoker	0.31 (0.19, 0.44)	0.14 (-0.04, 0.31)	0.37 (0.15, 0.58)
Obesity category, reference group: under and normal weight			
Overweight	-0.02 (-0.19, 0.16)	-0.08 (-0.30, 0.13)	-0.20 (-0.45, 0.05)
Obese	0.12 (-0.05, 0.29)	-0.19 (-0.40, 0.02)	-0.25 (-0.51, 0.00)
eGFR	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)
Hypertensive	-0.27 (-0.39, -0.16)	0.15 (-0.01, 0.30)	0.13 (-0.05, 0.31)
Cholesterol	-0.23 (-0.29, -0.17)	-0.09 (-0.16, -0.02)	-0.06 (-0.13, 0.03)
HbA1c	0.07 (0.03, 0.11)	0.02 (-0.04, 0.08)	0.01 (-0.05, 0.07)

### Interventions

Quality of care, reference group: Low			
Mid	-0.31 (-0.44, -0.17)	-0.16 (-0.35, 0.03)	-0.19 (-0.42, 0.05)
High	-0.40 (-0.56, -0.23)	-0.03 (-0.24, 0.19)	0.17 (-0.07, 0.42)
Diabetes treatment, reference group diet alone			
Metformin or sulphonylureas only	-0.28 (-0.43, -0.13)	-0.17 (-0.37, 0.03)	-0.05 (-0.31, 0.20)
Combination, with no insulin	-0.40 (-0.60, -0.21)	-0.29 (-0.56, -0.03)	-0.11 (-0.42, 0.19)
Insulin only	0.12 (-0.12, 0.36)	-0.07 (-0.38, 0.23)	0.34 (0.01, 0.67)
Combination with insulin	-0.31 (-0.60, -0.02)	-0.28 (-0.65, 0.10)	0.34 (-0.03, 0.72)
BP treatment, reference group: no treatment			
ACE inhibitors only	0.27 (0.01, 0.53)	0.30 (0.00, 0.59)	0.48 (0.16, 0.81)
Combination, with ACEI	1.50 (1.31, 1.70)	0.16 (-0.08, 0.40)	0.43 (0.15, 0.71)
Combination, no ACEI	1.25 (1.05, 1.45)	0.18 (-0.05, 0.42)	0.38 (0.10, 0.66)
Aspirin	1.37 (1.26, 1.49)	1.06 (0.89, 1.23)	0.58 (0.40, 0.76)
Lipid therapy	0.61 (0.48, 0.74)	0.09 (-0.08, 0.27)	0.10 (-0.10, 0.30)
Shared care	0.31 (0.11, 0.51)	0.60 (0.34, 0.86)	0.91 (0.63, 1.19)
Middlesbrough PCT	-0.2 (-0.38, -0.01)	-0.12 (-0.38, 0.16)	-0.19 (-0.59, 0.20)
Visit year, reference group: 2000			
2001	-0.29 (-0.59, 0.01)	0.21 (-0.18, 0.60)	-0.23 (-0.63, 0.19)
2002	-0.31 (-0.58, -0.03)	0.04 (-0.32, 0.41)	-0.24 (-0.62, 0.13)
2003	-0.40 (-0.66, -0.13)	0.15 (-0.21, 0.50)	0.03 (-0.32, 0.40)
2004	-0.67 (-0.93, -0.40)	0.03 (-0.32, 0.38)	-0.01 (-0.37, 0.36)
2005	-1.25 (-1.53, -0.96)	-0.19 (-0.55, 0.17)	-0.31 (-0.70, 0.09)
2006	-1.68 (-1.96, -1.39)	-0.80 (-1.20, -0.40)	-0.79 (-1.19, -0.38)
2007	-1.82 (-2.11, -1.52)	-0.71 (-1.11, -0.31)	-1.15 (-1.61, -0.69)

### Interactions

Shared care, reference group: Low SES & Shared care			
Mid SES*Shared care	0.01 (-0.31, 0.33)	-0.30 (-0.72, 0.11)	0.07 (-0.35, 0.51)
High SES*Shared care	-0.16 (-0.47, 0.14)	-0.34 (-0.72, 0.04)	-0.32 (-0.72, 0.09)
<b>Cons</b>	-3.88 (-5.04, -2.9)	-5.14 (-6.50, -4.03)	-5.95 (-7.37, -4.81)

### Variance estimate at:

<b>Practice level</b>	0.05 (0.02, 0.11)	0.11 (0.04, 0.22)	0.28 (0.15, 0.50)
<b>Patient level</b>	2.07 (0.55, 6.4)	1.35 (0.33, 4.51)	1.35 (0.33, 4.45)
<b>Bayesian DIC</b>	9197.41	6132.89	5023.01
<b>Available cases (N)</b>			30053

Table 35: Saturated logistic regression multilevel models examining incidences of ICD, stroke or TIA and PVD with interaction effect between SES and lipid therapies by 2000 to 2007, conditional on relevant explanatory variables

	Microalbuminuria	Retinopathy
<b>Social-economic status, reference group: low</b>		
Mid	-0.18 (-0.27, -0.09)	-0.07 (-0.21, 0.08)
High	-0.05 (-0.14, 0.03)	-0.04 (-0.18, 0.10)
<b>Covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	0.01 (-0.07, 0.09)	0.01 (-0.10, 0.12)
Age: 75+ years	0.38 (0.28, 0.47)	-0.10 (-0.24, 0.04)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	-0.02 (-0.09, 0.06)	0.47 (0.34, 0.60)
Duration 10+ years	0.18 (0.09, 0.27)	1.60 (1.47, 1.73)
Ethnicity, reference group: White		
South Asian	0.21 (0.04, 0.37)	-0.15 (-0.39, 0.08)
Other Ethnicity	0.31 (-0.08, 0.68)	0.52 (0.08, 0.95)
Male	0.24 (0.18, 0.31)	0.25 (0.16, 0.34)
Smoking status, reference: non-smoker		
Smoker	0.26 (0.17, 0.36)	-0.13 (-0.27, 0.00)
E*-smoker	0.07 (0.00, 0.14)	-0.12 (-0.22, -0.02)
Obesity category, reference group: under and normal weight		
Overweight	-0.01 (-0.11, 0.08)	-0.09 (-0.22, 0.04)
Obese	0.06 (-0.04, 0.15)	-0.10 (-0.24, 0.03)
eGFR		-0.01 (-0.01, -0.01)
Hypertensive	0.18 (0.12, 0.25)	0.33 (0.25, 0.42)
Cholesterol	0.03 (0.00, 0.06)	-0.01 (-0.05, 0.03)
HbA1c	0.09 (0.06, 0.11)	0.05 (0.02, 0.08)
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.11 (-0.43, 0.26)	0.41 (-1.62, 2.64)
High	-0.22 (-0.55, 0.15)	0.34 (-1.68, 2.58)
Diabetes treatment, reference group diet alone		
Metformin or sulphonylureas only	0.14 (0.04, 0.24)	0.40 (0.24, 0.57)
Combination, with no insulin	0.02 (-0.09, 0.13)	0.70 (0.53, 0.88)
Insulin only	0.18 (0.04, 0.32)	1.04 (0.86, 1.24)
Combination with insulin	0.21 (0.11, 0.31)	1.16 (0.96, 1.36)
BP treatments, reference group: No BP treatment		
ACE Inhibitors only	0.36 (0.26, 0.47)	0.32 (0.17, 0.46)
Combination with ACEI	0.53 (0.43, 0.62)	0.22 (0.09, 0.35)
Combination no ACEI	0.32 (0.23, 0.41)	0.13 (-0.01, 0.26)
Aspirin	0.08 (0.01, 0.14)	0.05 (-0.04, 0.14)
Lipid therapy	-0.05 (-0.12, 0.02)	-0.06 (-0.16, 0.04)
Shared care	-0.97 (-1.08, -0.85)	0.56 (0.42, 0.70)
Middlesbrough PCT	0.57 (0.27, 0.85)	-0.05 (-0.22, 0.13)
Visit year, reference group: 1999		
2000	-0.40 (-0.73, -0.07)	0.03 (-0.26, 0.33)
2001	-0.61 (-0.92, -0.29)	0.00 (-0.29, 0.30)
2002	-0.83 (-1.14, -0.52)	0.02 (-0.25, 0.32)
2003	-0.73 (-1.02, -0.42)	-0.09 (-0.37, 0.20)
2004	0.03 (-0.26, 0.33)	0.09 (-0.20, 0.38)
2005	0.21 (-0.08, 0.51)	0.36 (0.07, 0.65)
2006	0.59 (0.30, 0.90)	-0.67 (-0.97, -0.37)
2007	0.29 (-0.13, 0.72)	0.38 (0.10, 0.69)
<b>Interactions</b>		
Shared care, reference group: Low SES & Shared care		
Mid SES*Shared care	0.41 (0.23, 0.59)	-0.02 (-0.23, 0.19)

High SES*Shared care	-0.19 (-0.36, -0.01)	-0.07 (-0.27, 0.13)
<b>Cons</b>	-2.47 (-3.04, -1.84)	-3.45 (-5.75, -1.26)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.21 (0.13, 0.34)	0.05 (0.03, 0.10)
<b>Patient level</b>	0.01 (0.00, 0.03)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	25438.62	14530.78

## Primary care trust

In chapter 6, being managed under Middlesbrough PCT, compared to Redcar & Cleveland PCT, was a significant predictor of ICD but not with any other health outcome. The result showed patients managed by Middlesbrough PCT were significant more likely to have lower incidences of ICD (**Table 10**). This variable was not analysed graphically or modelled as an outcome variable as being managed by one PCT compared to the other, was not a consequence of patients' health or decisions in their care.

However, the results outlined here show there were statistically significant interactions between PCT and health outcomes by SES. Mid and high SES patients managed by Middlesbrough PCT were significantly more likely to have lower cholesterol levels compared to low SES patients in Middlesbrough PCT. In contrast, mid SES were significantly more likely to have higher microalbuminuria rates compared to low SES patients in Middlesbrough PCT.

Table 36: Saturated linear regression multilevel models examining HbA1c and cholesterol levels with interaction effect between SES and PCT by 1999 to 2007, conditional on relevant explanatory variables

	<b>HbA1c</b>	<b>Cholesterol</b>
<b>Social-economic status, reference group: low</b>		
Mid	-0.03 (-0.08, 0.01)	0.07 (0.04, 0.11)
High	-0.06 (-0.12, -0.01)	0.04 (0.00, 0.09)
<b>Socio-demographic, anthropometric, lifestyle and health</b>		
Age, reference group: <60 years		
Age: 60-74 years	-0.33 (-0.36, -0.29)	-0.20 (-0.23, -0.17)
Age: 75+ years	-0.41 (-0.45, -0.37)	-0.26 (-0.30, -0.23)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	0.06 (0.02, 0.09)	-0.09 (-0.11, -0.06)
Duration 10+ years	0.05 (0.01, 0.10)	-0.13 (-0.16, -0.10)
Ethnicity, reference group: White		
South Asian	0.46 (0.39, 0.54)	-0.08 (-0.14, -0.03)
Other Ethnicity	0.47 (0.31, 0.64)	0.08 (-0.05, 0.20)
Male	-0.06 (-0.09, -0.03)	-0.34 (-0.36, -0.32)
Smoking status, reference: non-smoker		
Smoker	0.23 (0.19, 0.27)	0.08 (0.04, 0.11)

Ex-smoker	0.05 (0.02, 0.08)	0.00 (-0.03, 0.02)
Obesity category, reference group: under and normal weight		
Overweight	0.02 (-0.02, 0.07)	0.03 (0.00, 0.07)
Obese	0.08 (0.04, 0.13)	0.03 (0.00, 0.07)
Creatinine > 300	-0.81 (-1.05, -0.56)	
Hypertensive	0.10 (0.07, 0.13)	0.14 (0.12, 0.16)
Ischaemic Cardiac	0.00 (-0.04, 0.03)	-0.13 (-0.15, -0.10)
Stroke or TIA	-0.06 (-0.10, -0.01)	-0.02 (-0.06, 0.01)
PVD	-0.06 (-0.12, -0.01)	0.01 (-0.03, 0.05)
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.13 (-0.16, -0.09)	-0.10 (-0.13, -0.08)
High	-0.15 (-0.19, -0.11)	-0.15 (-0.18, -0.11)
Diabetes treatment, reference group diet alone		
Metformin or sulphonylureas only	0.81 (0.77, 0.85)	
Combination, with no insulin	1.25 (1.20, 1.29)	
Insulin only	1.67 (1.61, 1.73)	
Combination with insulin	1.75 (1.69, 1.82)	
Aspirin		-0.09 (-0.11, -0.06)
Lipid therapy		-0.28 (-0.31, -0.26)
Middlesbrough PCT	0.12 (0.02, 0.22)	0.02 (-0.05, 0.08)
Shared care	0.17 (0.13, 0.21)	-0.07 (-0.10, -0.04)
Visit year, reference group: 1999		
2000	-0.32 (-0.40, -0.23)	-0.20 (-0.29, -0.11)
2001	-0.47 (-0.55, -0.38)	-0.21 (-0.30, -0.12)
2002	-0.55 (-0.63, -0.47)	-0.22 (-0.30, -0.14)
2003	-0.59 (-0.67, -0.52)	-0.37 (-0.45, -0.29)
2004	-0.63 (-0.71, -0.56)	-0.58 (-0.66, -0.50)
2005	-0.71 (-0.79, -0.64)	-0.73 (-0.82, -0.65)
2006	-1.18 (-1.26, -1.10)	-0.83 (-0.92, -0.75)
2007	-1.12 (-1.20, -1.04)	-0.93 (-1.02, -0.85)
<b>Interactions</b>		
Middlesbrough PCT, reference group: Low SES & Middlesbrough PCT		
Mid SES*Middlesbrough PCT	-0.02 (-0.10, 0.06)	-0.10 (-0.16, -0.03)
High SES*Middlesbrough PCT	-0.05 (-0.12, 0.03)	-0.08 (-0.14, -0.03)
<b>Cons</b>	7.58 (7.46, 7.71)	5.89 (5.78, 6.00)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.02 (0.01, 0.04)	0.01 (0.00, 0.01)
<b>Patient level</b>	0.00 (0.00, 0.02)	0.00 (0.00, 0.01)
<b>Visit year</b>	1.91 (1.88, 1.94)	1.14 (1.13, 1.16)
<b>Bayesian DIC</b>	133976.84	110328.06

Table 37: Saturated logistic regression multilevel models examining incidences of ICD, stroke or TIA and PVD with interaction effect between SES and PCT by 2000 to 2007, conditional on relevant explanatory variables

	ICD	Stroke or TIA	PVD
<b>Social-economic status, reference group: low</b>			
Mid	-0.19 (-0.38, -0.01)	0.05 (-0.20, 0.30)	-0.21 (-0.50, 0.08)
High	-0.11 (-0.31, 0.09)	0.08 (-0.19, 0.35)	-0.08 (-0.41, 0.23)
<b>Covariates</b>			
Age, reference group: <60 years			
Age: 60-74 years	0.33 (0.18, 0.48)	0.74 (0.51, 0.99)	0.62 (0.38, 0.88)
Age: 75+ years	0.60 (0.41, 0.78)	1.10 (0.83, 1.37)	0.82 (0.53, 1.12)
Duration of diabetes, reference group: 0-3 years			
Duration: 4-9 years	-0.59 (-0.73, -0.45)	-0.30 (-0.49, -0.11)	0.13 (-0.09, 0.35)
Duration 10+ years	-0.63 (-0.79, -0.46)	-0.18 (-0.39, 0.02)	0.38 (0.15, 0.62)
Ethnicity, reference group: White			
South Asian	0.08 (-0.24, 0.41)	0.10 (-0.35, 0.51)	-0.81 (-1.55, -0.16)
Other Ethnicity	-0.67 (-1.55, 0.12)	-1.16 (-3.02, 0.19)	-0.05 (-1.12, 0.85)
Male	0.32 (0.20, 0.44)	-0.11 (-0.27, 0.05)	0.40 (0.21, 0.58)
Smoking status, reference: non-smoker			
Smoker	0.15 (-0.02, 0.33)	0.27 (0.05, 0.50)	0.93 (0.68, 1.18)
Ex-smoker	0.31 (0.18, 0.44)	0.13 (-0.04, 0.31)	0.37 (0.16, 0.57)
Obesity category, reference group: under and normal weight			
Overweight	-0.02 (-0.19, 0.16)	-0.09 (-0.30, 0.13)	-0.20 (-0.44, 0.05)
Obese	0.12 (-0.05, 0.30)	-0.19 (-0.41, 0.03)	-0.25 (-0.49, 0.00)
eGFR	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)
Hypertensive	-0.27 (-0.39, -0.15)	0.15 (-0.01, 0.31)	0.13 (-0.04, 0.31)
Cholesterol	-0.23 (-0.29, -0.17)	-0.09 (-0.17, -0.01)	-0.05 (-0.14, 0.03)
HbA1c	0.07 (0.03, 0.11)	0.02 (-0.03, 0.08)	0.01 (-0.05, 0.07)
<b>Interventions</b>			
Quality of care, reference group: Low			
Mid	-0.31 (-0.45, -0.16)	-0.16 (-0.35, 0.04)	-0.18 (-0.41, 0.05)
High	-0.40 (-0.56, -0.24)	-0.02 (-0.24, 0.19)	0.18 (-0.06, 0.43)
Diabetes treatment, reference group diet alone			
Metformin or sulphonylureas only	-0.28 (-0.43, -0.14)	-0.18 (-0.38, 0.02)	-0.05 (-0.31, 0.21)
Combination, with no insulin	-0.40 (-0.59, -0.21)	-0.29 (-0.55, -0.04)	-0.11 (-0.43, 0.20)
Insulin only	0.12 (-0.13, 0.36)	-0.08 (-0.39, 0.22)	0.33 (0.00, 0.67)
Combination with insulin	-0.31 (-0.60, -0.03)	-0.28 (-0.66, 0.08)	0.33 (-0.04, 0.72)
BP treatment, reference group: no treatment			
ACE inhibitors only	0.26 (0.01, 0.52)	0.30 (0.00, 0.59)	0.47 (0.15, 0.80)
Combination, with ACEI	1.50 (1.31, 1.70)	0.16 (-0.08, 0.40)	0.42 (0.15, 0.70)
Combination, no ACEI	1.24 (1.05, 1.44)	0.18 (-0.05, 0.42)	0.37 (0.09, 0.66)
Aspirin	1.37 (1.26, 1.49)	1.06 (0.89, 1.23)	0.58 (0.40, 0.76)
Lipid therapy	0.61 (0.47, 0.74)	0.09 (-0.09, 0.27)	0.10 (-0.10, 0.30)
Shared care	0.28 (0.12, 0.43)	0.44 (0.24, 0.64)	0.84 (0.62, 1.06)
Middlesbrough PCT	-0.18 (-0.40, 0.04)	-0.04 (-0.35, 0.26)	-0.16 (-0.59, 0.27)
Visit year, reference group: 2000			
2001	-0.29 (-0.59, 0.00)	0.21 (-0.17, 0.60)	-0.23 (-0.65, 0.19)
2002	-0.31 (-0.58, -0.03)	0.03 (-0.33, 0.40)	-0.24 (-0.63, 0.13)
2003	-0.4 (-0.66, -0.13)	0.13 (-0.22, 0.49)	0.03 (-0.33, 0.39)
2004	-0.67 (-0.94, -0.40)	0.02 (-0.33, 0.38)	-0.01 (-0.37, 0.35)
2005	-1.25 (-1.52, -0.97)	-0.21 (-0.57, 0.17)	-0.31 (-0.69, 0.08)
2006	-1.68 (-1.97, -1.38)	-0.82 (-1.22, -0.42)	-0.79 (-1.21, -0.38)
2007	-1.82 (-2.12, -1.52)	-0.72 (-1.13, -0.32)	-1.14 (-1.60, -0.68)
<b>Interactions</b>			
Middlesbrough PCT, reference group: Low SES & Middlesbrough PCT			
Mid SES*Middlesbrough PCT	0.06 (-0.27, 0.37)	-0.13 (-0.57, 0.31)	0.20 (-0.28, 0.69)
High SES*Middlesbrough PCT	-0.09 (-0.37, 0.18)	-0.19 (-0.56, 0.20)	-0.24 (-0.68, 0.21)

<b>Cons</b>	-4.01 (-5.55, -2.83)	-5.04 (-6.14, -3.87)	-5.99 (-7.20, -4.80)
<b>Variance estimate at:</b>			
<b>Practice level</b>	0.05 (0.02, 0.11)	0.11 (0.04, 0.21)	0.28 (0.14, 0.49)
<b>Patient level</b>	2.27 (0.55, 7.71)	1.33 (0.34, 4.54)	1.43 (0.34, 4.88)
<b>Bayesian DIC</b>	9198.19	6135.54	5023.20

Table 38: Saturated logistic regression multilevel models examining microalbuminuria and retinopathy rates with interaction effect between SES and PCT by 1999 to 2007, conditional on relevant explanatory variables

	<b>Microalbuminuria</b>	<b>Retinopathy</b>
<b>Social-economic status, reference group: low</b>		
Mid	-0.18 (-0.29, -0.07)	-0.04 (-0.17, 0.10)
High	-0.18 (-0.30, -0.06)	0.00 (-0.16, 0.15)
<b>Covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	0.01 (-0.06, 0.09)	0.01 (-0.10, 0.12)
Age: 75+ years	0.38 (0.28, 0.47)	-0.10 (-0.24, 0.04)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	-0.01 (-0.09, 0.06)	0.47 (0.34, 0.59)
Duration 10+ years	0.18 (0.09, 0.27)	1.60 (1.47, 1.73)
Ethnicity, reference group: White		
South Asian	0.21 (0.05, 0.38)	-0.15 (-0.39, 0.08)
Other Ethnicity	0.30 (-0.07, 0.67)	0.52 (0.08, 0.94)
Male	0.24 (0.18, 0.31)	0.25 (0.16, 0.34)
Smoking status, reference: non-smoker		
Smoker	0.27 (0.18, 0.36)	-0.13 (-0.27, 0.00)
Ex-smoker	0.07 (0.00, 0.14)	-0.12 (-0.22, -0.02)
Obesity category, reference group: under and normal weight		
Overweight	-0.01 (-0.10, 0.09)	-0.09 (-0.22, 0.04)
Obese	0.06 (-0.03, 0.16)	-0.10 (-0.23, 0.03)
eGFR		-0.01 (-0.01, -0.01)
Hypertensive	0.18 (0.11, 0.24)	0.34 (0.25, 0.42)
Cholesterol	0.03 (0.00, 0.06)	-0.01 (-0.05, 0.03)
HbA1c	0.09 (0.06, 0.11)	0.05 (0.02, 0.08)
<b>Interventions</b>		
Quality of care, reference group: Low		
Mid	-0.08 (-0.46, 0.28)	0.85 (-0.79, 2.37)
High	-0.20 (-0.58, 0.17)	0.78 (-0.86, 2.32)
Diabetes treatment, reference group diet alone		
Metformin or sulphonylureas only	0.14 (0.05, 0.24)	0.40 (0.24, 0.56)
Combination, with no insulin	0.02 (-0.09, 0.14)	0.70 (0.52, 0.87)
Insulin only	0.18 (0.04, 0.32)	1.04 (0.85, 1.23)
Combination with insulin	0.21 (0.11, 0.30)	1.16 (0.96, 1.36)
BP treatments, reference group: No BP treatment		
ACE Inhibitors only	0.37 (0.26, 0.48)	0.31 (0.16, 0.46)
Combination with ACEI	0.53 (0.44, 0.62)	0.22 (0.09, 0.35)
Combination no ACEI	0.32 (0.23, 0.41)	0.13 (-0.01, 0.26)
Aspirin	0.08 (0.01, 0.14)	0.05 (-0.03, 0.15)
Lipid therapy	-0.05 (-0.12, 0.02)	-0.06 (-0.16, 0.03)
Shared care	-0.93 (-1.02, -0.84)	0.53 (0.43, 0.64)
Middlesbrough PCT	0.51 (0.21, 0.81)	0.01 (-0.19, 0.21)
Visit year, reference group: 1999		
2000	-0.40 (-0.73, -0.07)	0.03 (-0.27, 0.34)
2001	-0.61 (-0.92, -0.29)	0.00 (-0.30, 0.30)
2002	-0.84 (-1.15, -0.53)	0.02 (-0.27, 0.32)
2003	-0.73 (-1.03, -0.44)	-0.09 (-0.39, 0.21)

2004	0.02 (-0.27, 0.32)	0.09 (-0.20, 0.39)
2005	0.21 (-0.08, 0.51)	0.36 (0.05, 0.67)
2006	0.58 (0.29, 0.88)	-0.67 (-0.98, -0.36)
2007	0.27 (-0.15, 0.69)	0.39 (0.08, 0.69)
<b>Interactions</b>		
Middlesbrough PCT, reference group: Low SES & Middlesbrough PCT		
Mid SES*Middlesbrough PCT	0.20 (0.03, 0.37)	-0.08 (-0.33, 0.16)
High SES*Middlesbrough PCT	0.14 (-0.01, 0.30)	-0.13 (-0.35, 0.08)
<b>Cons</b>	-3.25 (-3.80, -2.71)	-3.91 (-5.48, -2.26)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.21 (0.13, 0.34)	0.05 (0.03, 0.10)
<b>Patient level</b>	0.01 (0.00, 0.03)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	25465.93	14530.17

## Principle findings

Overall, the findings from this chapter suggest that in most circumstances there were no differential association between type 2 diabetes interventions and health outcomes by SES. As such, little or no evidence of intervention generated inequalities. However, there were exceptions to this. Receiving high quality of care was found to be associated with higher levels of HbA1c but lower incidences of PVD for high SES patients compared to low SES patients. High quality of care was also found to be associated with higher incidences of ICD for mid SES patients compared to low SES patients. There was evidence suggesting being prescribed insulin only was associated with better health outcomes in terms of HbA1c and retinopathy but poorer rates of microalbuminuria for with higher SES patients than low SES patients. In addition, being prescribed insulin in combination with other diabetes treatments was associated with lower levels of HbA1c and ICD for high SES patients than low SES patients. There were a number of significant interactions between BP treatment regimens and SES, with association in lower rates of ICD and stroke or TIA but higher rates for PVD and retinopathy for mid and high SES patients than low SES patients. The results suggest that aspirin and lipid therapies are associated with lower cholesterol levels for higher SES patients than low SES patients. Interestingly, for high SES patients shared care was associated with lower HbA1c levels but lower incidences of stroke or TIA than low SES patients. PCT had significant differential impact on cholesterol and ICD rates; with contrasting relationships. From the limited conducted analyses in this chapter there appears to be no differential impact on long-term complications associated with timeliness of diagnosis by SES.

## Chapter 9: What impact do general practices have on inequalities by socio-economic status in diabetes care and health outcomes?

In the preceding results chapters, inequalities by SES have been examined in the intermediate and long-term health complications of type 2 diabetes, as well as the uptake and impact of type 2 diabetes interventions. This chapter examines the impact of general practices on inequalities by SES in terms of level of deprivation of the practice population, practice list size, diabetes prevalence and QOF diabetes indicators. In addition, the level of variation at general practice level when analysing socio-economic inequalities in the health outcomes and interventions in the MLM in the preceding chapters. This was because the large variations between the general practices could imply that the general practice at which patients are registered have an important influence on their health and care aside from the individual care they receive.

### Multilevel analyses

Table 39 shows the results from the saturated linear regression multilevel models for HbA1c and cholesterol with general practice data and interaction effects between SES and visit year. There were 22,056 and 22,135 available cases for HbA1c and cholesterol, respectively.

Table 39: Saturated linear regression multilevel models examining HbA1c and cholesterol by SES from 1999 to 2007, with general practice data and interaction effects between SES and visit year, conditional on explanatory variables

	HbA1c	Cholesterol
<b>Social-economic status, reference group: Low SES</b>		
Medium SES	-0.04 (-0.13, 0.06)	0.01 (-0.06, 0.09)
High SES	-0.07 (-0.16, 0.02)	-0.02 (-0.09, 0.05)
Visit year, reference group: 2004		
2005	-0.03 (-0.12, 0.05)	-0.10 (-0.16, -0.03)
2006	-0.40 (-0.50, -0.31)	-0.10 (-0.18, -0.03)
2007	-0.33 (-0.44, -0.22)	-0.17 (-0.25, -0.08)
SES x Visit year, reference group: Low SES x 2004		
Medium SES x 2005	-0.02 (-0.15, 0.11)	-0.03 (-0.14, 0.08)
Medium SES x 2006	0.00 (-0.13, 0.12)	0.07 (-0.03, 0.17)
Medium SES x 2007	0.04 (-0.10, 0.16)	0.02 (-0.09, 0.13)
High SES x 2005	-0.07 (-0.19, 0.05)	-0.01 (-0.11, 0.09)
High SES x 2006	-0.02 (-0.13, 0.10)	0.07 (-0.02, 0.16)

High SES x 2007	-0.03 (-0.15, 0.08)	0.00 (-0.10, 0.09)
<b>Covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	-0.36 (-0.40, -0.31)	-0.21 (-0.24, -0.17)
Age: 75+ years	-0.40 (-0.46, -0.35)	-0.26 (-0.30, -0.22)
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	0.03 (-0.02, 0.07)	-0.14 (-0.17, -0.11)
Duration 10+ years	0.03 (-0.02, 0.08)	-0.22 (-0.26, -0.18)
Ethnicity, reference group: White		
South Asian	0.46 (0.37, 0.55)	-0.05 (-0.12, 0.03)
Other Ethnicity	0.33 (0.13, 0.52)	0.13 (-0.03, 0.28)
Male	-0.02 (-0.06, 0.02)	-0.31 (-0.34, -0.28)
Smoking status, reference group: Non smoker		
Smoker	0.23 (0.17, 0.28)	0.11 (0.06, 0.15)
Ex-smoker	0.03 (-0.01, 0.07)	0.01 (-0.02, 0.04)
BMI status, reference group: Low & normal weight		
Overweight	-0.01 (-0.06, 0.05)	0.02 (-0.02, 0.07)
Obese	0.06 (0.01, 0.12)	0.01 (-0.03, 0.05)
Creatinine > 300	-0.78 (-1.08, -0.49)	
Hypertensive	0.12 (0.08, 0.16)	0.15 (0.12, 0.18)
Ischaemic Cardiac	0.01 (-0.03, 0.05)	-0.12 (-0.15, -0.08)
Stroke or TIA	-0.08 (-0.13, -0.02)	-0.03 (-0.07, 0.02)
PVD	-0.05 (-0.12, 0.01)	0.00 (-0.05, 0.05)
<b>Interventions, Patient level</b>		
Quality of Care level, reference group: Low quality		
Medium quality	-0.17 (-0.21, -0.12)	-0.14 (-0.18, -0.10)
High quality	-0.21 (-0.27, -0.16)	-0.24 (-0.28, -0.19)
Diabetes treatment, reference group diet alone		
Metformin or sulphonylureas only	0.74 (0.69, 0.79)	
Combination, no insulin	1.14 (1.09, 1.20)	
Insulin only	1.64 (1.56, 1.72)	
Combination with insulin	1.74 (1.67, 1.82)	
Aspirin		-0.08 (-0.11, -0.05)
Lipid therapy(s)		-0.37 (-0.40, -0.33)
Shared care	0.07 (0.02, 0.12)	-0.07 (-0.10, -0.03)
Middlesbrough PCT	0.08 (-0.04, 0.21)	-0.04 (-0.11, 0.03)
<b>Interventions, Practice level</b>		
Practice deprivation, reference group: High		
Mid	0.12 (0.00, 0.24)	0.02 (-0.05, 0.09)
Low	0.15 (-0.05, 0.36)	0.13 (0.02, 0.24)
Practice list size, reference: <7,000		
7,000 – 9,999	0.07 (-0.03, 0.18)	0.00 (-0.07, 0.06)
≥10,000	0.06 (-0.08, 0.21)	0.00 (-0.08, 0.09)
Diabetes prevalence	-0.09 (-0.18, 0.00)	0.01 (-0.04, 0.07)
BMI recording level	0.00 (-0.01, 0.01)	0.00 (-0.01, 0.01)
HbA1c ≤ 10%	-0.02 (-0.03, -0.01)	-0.01 (-0.02, 0.00)
Peripheral pulses recording level	0.00 (-0.01, 0.01)	0.00 (-0.01, 0.01)
Neuropathy test recording test	0.00 (-0.01, 0.01)	0.00 (-0.01, 0.00)
BP recording level	0.00 (-0.02, 0.02)	-0.01 (-0.02, 0.01)
BP ≤ 145/85 (mmHg) level (%)	0.00 (0.00, 0.00)	0.00 (0.00, 0.00)
Microalbuminuria recording level	0.00 (0.00, 0.01)	0.00 (0.00, 0.00)
Proteinuria/microalbuminuria treated with ACEI level	0.00 (0.00, 0.01)	0.00 (0.00, 0.00)
Cholesterol recording level	0.03 (0.01, 0.04)	0.03 (0.02, 0.04)
Cholesterol ≤ 5mmol/l (%)	-0.01 (-0.02, 0.00)	-0.02 (-0.02, -0.01)
Influenza immunisation level	0.00 (-0.01, 0.00)	0.00 (-0.01, 0.00)
<b>Cons</b>	6.79 (5.91, 7.66)	5.45 (4.80, 6.10)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.02 (0.01, 0.04)	0.01 (0.00, 0.01)
<b>Patient level</b>	0.01 (0.00, 0.05)	0.01 (0.00, 0.04)

<b>Visit year</b>	1.67 (1.64, 1.70)	1.11 (1.09, 1.13)
<b>Bayesian DIC</b>	73120.25	65260.66
<b>Available cases (N)</b>	22,056	22,135

The results in Table 39 show that there no significant differences in HbA1c and cholesterol, both overall and over time. These results were consistent across each step of the analyses for the cholesterol model. When HbA1c was modelled with the SES and visit year interaction only there were significant differences with higher SES associated with lower levels, however, this finding was not significant once socio-demographic, anthropometric, lifestyle and health status data were introduced into the model. As such, the results here suggest that general practices were not associated with differences by SES in terms of intermediate outcomes. However, there were statistically significant results suggesting that differences in general practice patients' health outcomes. As expected, the results indicated that general practices with patient level characteristics were associated with higher numbers of patients meeting care targets for HbA1c and cholesterol which were significantly associated with lower levels at the individual level. In contrast, however, higher cholesterol recording levels was significantly associated with higher levels in both outcomes. No other general practice indicator was statistically significant. However, this data did explain more of the variation in both models as measured by the Bayesian DIC statistics.

Table 40: Saturated linear regression multilevel models examining HbA1c and cholesterol by SES from 1999 to 2007, with general practice data and interaction effects between SES and visit year, conditional on explanatory variables

	<b>HbA1c</b>	<b>Cholesterol</b>
<b>Social-economic status, reference group: Low SES</b>		
Medium SES	-0.04 (-0.13, 0.06)	0.01 (-0.06, 0.09)
High SES	-0.07 (-0.16, 0.02)	-0.02 (-0.09, 0.05)
Visit year, reference group: 2004		
2005	-0.03 (-0.12, 0.05)	<b>-0.10 (-0.16, -0.03)</b>
2006	<b>-0.40 (-0.50, -0.31)</b>	<b>-0.10 (-0.18, -0.03)</b>
2007	<b>-0.33 (-0.44, -0.22)</b>	<b>-0.17 (-0.25, -0.08)</b>
SES x Visit year, reference group: Low SES x 2004		
Medium SES x 2005	-0.02 (-0.15, 0.11)	-0.03 (-0.14, 0.08)
Medium SES x 2006	0.00 (-0.13, 0.12)	0.07 (-0.03, 0.17)
Medium SES x 2007	0.04 (-0.10, 0.16)	0.02 (-0.09, 0.13)
High SES x 2005	-0.07 (-0.19, 0.05)	-0.01 (-0.11, 0.09)
High SES x 2006	-0.02 (-0.13, 0.10)	0.07 (-0.02, 0.16)
High SES x 2007	-0.03 (-0.15, 0.08)	0.00 (-0.10, 0.09)
<b>Covariates</b>		
Age, reference group: <60 years		
Age: 60-74 years	<b>-0.36 (-0.40, -0.31)</b>	<b>-0.21 (-0.24, -0.17)</b>
Age: 75+ years	<b>-0.40 (-0.46, -0.35)</b>	<b>-0.26 (-0.30, -0.22)</b>
Duration of diabetes, reference group: 0-3 years		
Duration: 4-9 years	0.03 (-0.02, 0.07)	<b>-0.14 (-0.17, -0.11)</b>

Duration 10+ years	0.03 (-0.02, 0.08)	-0.22 (-0.26, -0.18)
Ethnicity, reference group: White		
South Asian	0.46 (0.37, 0.55)	-0.05 (-0.12, 0.03)
Other Ethnicity	0.33 (0.13, 0.52)	0.13 (-0.03, 0.28)
Male	-0.02 (-0.06, 0.02)	-0.31 (-0.34, -0.28)
Smoking status, reference group: Non smoker		
Smoker	0.23 (0.17, 0.28)	0.11 (0.06, 0.15)
Ex-smoker	0.03 (-0.01, 0.07)	0.01 (-0.02, 0.04)
BMI status, reference group: Low & normal weight		
Overweight	-0.01 (-0.06, 0.05)	0.02 (-0.02, 0.07)
Obese	0.06 (0.01, 0.12)	0.01 (-0.03, 0.05)
Creatinine > 300	-0.78 (-1.08, -0.49)	
Hypertensive	0.12 (0.08, 0.16)	0.15 (0.12, 0.18)
Ischaemic Cardiac	0.01 (-0.03, 0.05)	-0.12 (-0.15, -0.08)
Stroke or TIA	-0.08 (-0.13, -0.02)	-0.03 (-0.07, 0.02)
PVD	-0.05 (-0.12, 0.01)	0.00 (-0.05, 0.05)
<b>Interventions, Patient level</b>		
Quality of Care level, reference group: Low quality		
Medium quality	-0.17 (-0.21, -0.12)	-0.14 (-0.18, -0.10)
High quality	-0.21 (-0.27, -0.16)	-0.24 (-0.28, -0.19)
Diabetes treatment, reference group diet alone		
Metformin or sulphonylureas only	0.74 (0.69, 0.79)	
Combination, no insulin	1.14 (1.09, 1.20)	
Insulin only	1.64 (1.56, 1.72)	
Combination with insulin	1.74 (1.67, 1.82)	
Aspirin		-0.08 (-0.11, -0.05)
Lipid therapy(s)		-0.37 (-0.40, -0.33)
Shared care	0.07 (0.02, 0.12)	-0.07 (-0.10, -0.03)
Middlesbrough PCT	0.08 (-0.04, 0.21)	-0.04 (-0.11, 0.03)
<b>Interventions, Practice level</b>		
Practice deprivation, reference group: High		
Mid	0.12 (0.00, 0.24)	0.02 (-0.05, 0.09)
Low	0.15 (-0.05, 0.36)	0.13 (0.02, 0.24)
Practice list size, reference: <7,000		
7,000 – 9,999	0.07 (-0.03, 0.18)	0.00 (-0.07, 0.06)
≥10,000	0.06 (-0.08, 0.21)	0.00 (-0.08, 0.09)
Diabetes prevalence	-0.09 (-0.18, 0.00)	0.01 (-0.04, 0.07)
BMI recording level	0.00 (-0.01, 0.01)	0.00 (-0.01, 0.01)
HbA1c ≤ 10%	-0.02 (-0.03, -0.01)	-0.01 (-0.02, 0.00)
Peripheral pulses recording level	0.00 (-0.01, 0.01)	0.00 (-0.01, 0.01)
Neuropathy test recording test	0.00 (-0.01, 0.01)	0.00 (-0.01, 0.00)
BP recording level	0.00 (-0.02, 0.02)	-0.01 (-0.02, 0.01)
BP ≤ 145/85 (mmHg) level (%)	0.00 (0.00, 0.00)	0.00 (0.00, 0.00)
Microalbuminuria recording level	0.00 (0.00, 0.01)	0.00 (0.00, 0.00)
Proteinuria/microalbuminuria treated with ACEI level	0.00 (0.00, 0.01)	0.00 (0.00, 0.00)
Cholesterol recording level	0.03 (0.01, 0.04)	0.03 (0.02, 0.04)
Cholesterol ≤ 5mmol/l (%)	-0.01 (-0.02, 0.00)	-0.02 (-0.02, -0.01)
Influenza immunisation level	0.00 (-0.01, 0.00)	0.00 (-0.01, 0.00)
<b>Cons</b>	6.79 (5.91, 7.66)	5.45 (4.80, 6.10)
<b>Variance estimate at:</b>		
<b>Practice level</b>	0.02 (0.01, 0.04)	0.01 (0.00, 0.01)
<b>Patient level</b>	0.01 (0.00, 0.05)	0.01 (0.00, 0.04)
<b>Visit year</b>	1.67 (1.64, 1.70)	1.11 (1.09, 1.13)
<b>Bayesian DIC</b>	73120.25	65260.66
<b>Available cases (N)</b>	22,056	22,135

## Practice level variation

This section reviews the intra class correlation coefficient (ICC) at practice level of the null multilevel models fitted for the research questions in the current and preceding results chapters. to establish the importance of patients' general practice on their health outcomes and care. These were summarised in Table 41.

Overall, the variance estimates and ICC results revealed that only a limited amount of the variance occurred at the general practice level. The vast majority of the multilevel models exhibited approximately 3% of variance or less of between practices. This potentially negates the need for multilevel analyses for many of these models. However, there were some notable exceptions. The modelling of intervention data as the outcome variable resulted in some cases where about 5% occurred between general practices. This was namely with diabetes being treated by diet alone (4.62%) and the most marked, receiving shared care (19.31%). This suggests that the practice a patient was registered with has some influence on the receipt of these interventions, particularly with shared care.

When modelling health outcomes, there was about 2% or less of the variance in intermediate outcomes and long term complications was accounted for at the general practice level. This suggests that differences in patients' health were explained by other factors and not the practice they were registered with.

Table 41: Intra class correlation coefficient results from the multilevel analyses ordered by research question and outcome variable

		Practice level	Patient level	Visit year
Are there socio-economic inequalities in intermediate outcomes and complications associated with type 2 diabetes over time? (Chapter 6)				
Intermediate health outcomes	Hba1c	0.04	0.01	99.95
	Cholesterol	0.01	0.00	99.99
Long-term complications	ICD	0.02	64.40	35.59
	Stroke or TIA	0.21	31.43	68.36
	PVD	1.58	21.54	76.88
	Micro-albuminuria	1.58	0.07	98.34
	Retinopathy	0.15	0.97	98.88
Are there socio-economic inequalities in interventions associated with type 2 diabetes over time? (Chapter 7)				
	Timeliness of diagnosis	0.14	76.06	23.84
	Quality of care	0.51	0.00	99.97

Diabetes treatments	Diet alone	4.62	0.35	95.03
	Metformin or sulphonylureas	0.15	0.25	99.61
	Combination, no insulin	0.19	0.00	99.81
	Insulin only	0.68	0.19	99.13
	Combination with insulin	0.19	0.08	99.73
BP treatments	No BP treatments	0.25	0.00	99.75
	ACEI only	0.37	0.00	99.63
	ACEI combination	0.05	0.00	99.95
	Other combination	0.08	0.00	99.92
Antithrombotic & lipid profile treatments	Lipid therapies	0.37	0.00	99.63
	Aspirin	0.19	0.00	99.80
	Shared care	19.31	0.48	80.21
Are there intervention-generated inequalities in type 2 diabetes care? (Chapter 8)				
	HbA1c	0.04	0.01	99.95
	Cholesterol	0.01	0.00	99.99
	ICD	0.02	64.40	35.59
	Stroke or TIA	0.21	31.43	68.36
	PVD	2.03	18.67	79.31
	Microalbuminuria	1.58	0.07	98.34
	Retinopathy	0.15	0.97	98.98
Timeliness of diagnosis	Retinopathy	0.43	0.59	98.98
	Microalbuminuria	6.04	0.05	93.91
What impact do general practices have on inequalities by socio-economic status in diabetes care and health outcomes? (Chapter 9)				
	HbA1c	0.01	0.00	99.98
	Cholesterol	0.01	0.01	99.98

## Principle findings

The analyses conducted here and in previous chapters, suggest that where a patient was registered for their primary care has little association with their health outcomes and interventions they receive. However, shared care was a notable exception with varied between practices.

## Chapter 10: Discussion

This chapter discusses the results from the secondary data analyses outlined in the preceding five chapters, drawing upon the findings from the systematic review. Firstly, the principle findings are summarised by research question. This was followed by a discussion the strengths and limitations of the study. Next, the meaning of the study and implications for practitioners and policy makers were discussed, including what contribution this study has made to the understanding of intervention generated inequalities. Finally, unanswered questions and future research were identified.

### Summary of results

#### **Are there socio-economic inequalities in intermediate outcomes and complications associated with type 2 diabetes over time?**

The multilevel analyses in chapter six found some, but inconsistent evidence of SES inequalities in intermediate outcomes. High SES patients were more likely to have lower HbA1c over time, but higher levels of cholesterol compared to low SES patients. In contrast, there were few differences in long-term complications by SES and no evidence of differences SES over time. However, in 2003 mid SES patients were more likely to have higher rates of ICD than low SES patients in 1999.

#### **Are there socio-economic inequalities in interventions associated with type 2 diabetes over time?**

In comparison to low SES patients in 1999, there was some evidence to suggest that mid SES patients received a more timely diagnosis, have their diabetes managed through diet alone and were less likely to be prescribed a monotherapy of metformin or sulphonlureas over time. Also, high SES patients were more likely to receive higher quality of care and shared care than low

SES patients in 1999. In addition, high SES patients were also more likely to be prescribed a monotherapy of metformin or sulphonylureas, insulin only and in combination with another diabetes treatment, any BP treatment and a combination of BP treatments with no ACEI than low SES patients in 1999.

### **Are there intervention-generated inequalities in type 2 diabetes care?**

The majority of the interaction results presented in chapter eight indicated that in most circumstances there were no differential association between type 2 diabetes interventions and health outcomes by SES. There were some exceptions indicating that there were intervention-generated inequalities in type 2 diabetes care as the association between interventions and health outcomes differed by SES.

High quality of care was associated with higher HbA1c level but lower incidences of PVD for high SES patients and higher incidences of ICD for mid SES patients compared to low SES patients receiving the same care. Prescriptions for the same treatments were associated with differences in health outcomes by SES. Prescriptions for insulin only were associated with lower HbA1c and retinopathy but with higher rates of microalbuminuria for high SES patients compared to low SES patients. Prescriptions for insulin in combination with other diabetes treatments were associated with lower HbA1c and lower incidences of ICD for high SES patients compared to low SES patients. BP treatments were associated with lower incidences of ICD and stroke or TIA and higher rates of PVD and retinopathy for mid and high SES patients compared to low SES patients. Aspirin and lipid therapies were associated with lower cholesterol levels for high SES patients than low SES patients. Finally, receiving shared care was associated with lower HbA1c levels but higher incidences of stroke or TIA for high SES patients than low SES patients. PCT had significant differential association with cholesterol and ICD incidences; with contrasting relationships.

## What impact do general practices have on inequalities by socio-economic status in diabetes care and health outcomes?

The results in chapter nine and the review of the analyses conducted in the preceding chapters indicated that characteristics of general practices in the South Tees area, which were included in the analyses, have no association with socio-economic inequalities in intermediate outcomes. In addition, the ICC results indicated that for patients in South Tees the choice of general practice at which they registered with had limited association in the variation in patients with health outcomes and diabetes care. However, notable proportion of the variation in receiving shared care was explained at the practice level.

### Principle findings

In most of the models featured in analyses in this thesis visit year was a statistically significant explanatory variable. The multilevel models, also supported by the graphical analyses, showed that quality of care, levels of HbA1c, cholesterol and ICD had improved over the study period. This could be partly attributed to policy level interventions such as the NSF Diabetes Strategy introduced in 2001 [200] and NICE clinical guidelines [46] introduced in 2002. However, the trend appears to have started prior to the publication of these policies therefore other factors are likely to be more important. For example, not surprisingly treatments were also associated with these health outcomes with statistical significance. The results in chapter seven showed statistically significant changes in all treatments over time. These may explain the improvements in health over time. However, it is unclear from these analyses whether treatments have driven the improvements or that there is an increase need for these treatments, that is patients health is deteriorating more over the study period.

One of the key findings from these analyses was the statistically significance differences in receiving no diabetes treatments over time by SES. The graphical analyses suggests that proportion of high SES patients receiving no diabetes treatments remain similar over time, there was a steady reduction to the proportion of low SES managing their diabetes through lifestyle changes alone. The stepwise models in appendix G shows the by including many of the explanatory variables mediates this relationship. However, there were still some statistically significant differences between medium and low SES patients. This potentially suggests that continued inequalities in type 2 diabetes patients health by SES may be generated by factors

that cannot be directly addressed by healthcare interventions alone. For example, health related behaviours, such as smoking, alcohol consumption, diet, and exercise, which have been shown to vary by area deprivation [201].

However, there was some evidence where diabetes care could be improved to reduce the differences in the receipt of care and the health outcomes they are associated with. Namely, quality of care and shared care. Both the graphical analyses and multilevel models indicated that whilst quality has in general improved overtime, patients from more advantaged backgrounds receive greater quality of care. The graphical analyses showed that the proportion of patients receiving shared care have decreased over time. This is likely to be explained partly by the governments drive to have more chronic conditions managed in primary care [58]. In addition, the capacity to managed patients in the hospital based diabetes clinic has remained steady over the study period [202], as such the reduction to the proportion of patients receiving such care is likely to be the increase in the number of diagnosed patients. In contrast to graphical analyses, the multilevel model showed when conditional on other factors high SES were more likely to receive shared care. This suggests access to a limited, specialist resource was not solely determined by patient need.

In addition, there statistical significant findings which indicated that when patients were receiving the same level of quality of care and shared care there were instances of differential health outcomes. However, the direction of these relationships need to be explored further.

## Strengths and limitations of the study

The major strength of this study was being able to investigate a wide range of aspects of the type 2 diabetes care pathway, from diagnosis to secondary and tertiary preventions, in the same population over a long period. Comparisons with QOF prevalence showed that patients with type 2 diabetes captured through the diabetes register were approximately at the expected level for practices in the South Tees area (see Appendix J). This was important as it has been hypothesised that intervention generated inequalities could be introduced at any stage of the intervention pathway [1, 27]. In doing so, this study was able to contribute to the existing evidence base which lacked analyses of data with repeat measurements and investigation into inequalities associated with particular interventions, namely: timeliness of diagnosis, quality of care and place of care.

The statistical techniques used for the secondary data analyses were also a particular strength of this study. By using multilevel regression techniques these analyses were able to control for and investigate the effect general practices have on the variation of patients' health outcomes and diabetes care. Whilst in the most part this analyses was found to unnecessary, this is in itself an important finding as it suggests that general practices, or interventions such QOF, operating at this level have limited impact and it appear to be the individual level factors which have the greatest impact on patients' health and the interventions they receive. However, these findings need to be supported by further investigation both outside South Tees and within as the impact of general practice on patients' health and care may only be the case with type 2 diabetes. Using interaction effects between SES and time, these analyses were also able to investigate trends over time in health outcomes and diabetes care, which were also lacking from the evidence base. There are many other more sophisticated techniques, such as time-lag and autoregressive using random coefficient multilevel models, which could have been employed. However, the choice of which technique is generally always taken on practical grounds rather than what is the most ideal, with the former approach taken for this thesis. It was not possible to use the more complex techniques due to the quality of the data available and the analyses failing to provide robust results. However, as with all such longitudinal analyses examining change there are problems with interpretation and, therefore, it is recommended that multiple sets of analyses using different techniques should be conducted in order to validate about the findings [194].

Like all secondary data analysis the quality and availability of the data limits the possibility of the study. As mentioned previously the completeness of the data and recording errors meant

that the quality of the data for multilevel modeling was poor. Whilst the robustness of the results was improved by using MCMC, a compromise was made regarding how much the analyses could have been strengthened against how much time was available. The results could have benefitted from using a greater number of iterations, however, because running each model took several hours this process was limited to ensure that as many relevant stepwise models as possible could be included in the final set of analyses. In addition, preliminary analyses indicated that missing data was significantly related to SES. As such, by using available cases only may bias the results as records with missing data may be patients who are consistently in worse health. For example, patients who do not engage on an annual basis with services, or at all, may also have worse health as such these results and may not to be a robust reflection of all type 2 diabetes patients.

As highlighted by the Bayesian DIC diagnostic statistics, another problematic feature of this study was the amount of unexplained variance in the multilevel models. This indicates there was a lot of information that was not accounted for in the models that could have potentially explained the variation in the outcomes. In particular, there were no individual level measures of SES. Using IMD data can only operate as a proxy indicator and therefore does not necessarily reflect a patients' individual circumstances. In addition, from the analyses conducted here it was not possible to determine whether there was reverse causation of IMD i.e. poor health results in increased deprivation rather than the other way around. Individual level measurement of SES could potentially overcome this. For example, using education level as this is likely to have been established prior to a diagnosis of type 2 diabetes. However, gaining this additional information would have been both very costly and time consuming. Such a process could also introduce potential bias into the analyses as patient consent would be required. If this is not equitable across the study population, and any subsequent analyses may have been biased.

Another issue regarding the availability data was surrounding comorbidities, which was discussed in Chapter 4. Comorbidities is an important area as it can greatly impact on the type and quality of care a patient receives and also their ability to cope and manage the level of, and potentially competing, health demands [148]. The research proposal submitted to the ethics committee had planned to measure comorbidity using HES data. Unfortunately, it was not until the latter stages of this study that it was discovered that whilst patient identifiable data, namely patient NHS numbers, could not be released to individuals who did not have prior access to them and a section 251 exemption having to be applied for. This was required for permission to be granted to enable one organisation to pass this data to another organisation. The section 251 was subsequently applied for and permission was granted. However, steps had to be taken to ensure that patients, whose data was to be used, were informed and allowed to dissent from the

study. Due to the time constraints that this imposed, as well as the length of time needed for the HES data to be extracted, cleaned and formatted, along with the potential bias that dissenting patient could introduce, this step had to be abandoned.

As well as the constraints that gaining access to this data imposed, there was always the risk that HES was not an appropriate data source to construct a comorbidity index. This is because it predominantly records inpatient data therefore only other health conditions which are sufficiently serious enough to result in a hospital admission or referral would be accounted for and therefore would miss other important information [153]. Conditions such as depression have been shown to have a particular detrimental impact on patients care and ability to cope with their condition [203]. Ideally, data from patients general practices would have been used to measure comorbidity, however, due to the large amounts of data to be extracted from all general practices in the South Tees area, which do not all use the same information systems, this would have made this a particularly laborious task.

As mentioned elsewhere in this thesis, this study was not able include details about patients' treatments such as the dose and delivery system and also whether patients adhered to their prescription instructions as these were not captured through the diabetes register proforma. The interpretation of the results related to patients' treatments was therefore limited to their access to treatment. In addition, it was not clear from this data who was offered but refused that treatment.

Additional general practice information was the other area of data collection that had been planned for in the research proposal. This included practice list size, number of patients with type 2 diabetes, staffing levels and skills per year from 1999 to 2007 that was not publically available elsewhere. Using a freedom of information request was advised as a method of data collection, however, this met with particular animosity from practice managers. This made voluntary contributions preferable so as not to alienate practices from taking part in future research in relation to this study. As such, practice managers were initially telephoned so that the background to the study and the importance of this information could be explained. Following that discussion, an agreement was made to send the data extraction form via email to either the practice manager or a nominated member of staff. However, whilst follow up calls were made, the majority of practices did not respond with the data. Some explained that too much staff time would be needed to fulfil this request. Furthermore, of those that did respond, there was considerable variation in how much data was returned, for example, with some completing the only few a years. Therefore, no data was used to the potential bias of the results.

There were also a number of social determinants of health, as outlined by Dahlgren, 1991 [141], which have been demonstrated elsewhere to impact upon patients' health outcomes associated with diabetes. For example, other constitutional factors such as birth weight [204], lifestyle such as physical activity and dietary behaviours [205-207], social and community networks such as (e.g. [208, 209]; living and working conditions including issues such as unemployment and work related stress (e.g. [210, 211]), and other general socio-economic, cultural and environmental conditions (e.g. [212]). At the other end of the scale, population level policies such as cigarette pricing, and Change 4 Life campaign, were not accounted for which could have a differential impact on patients health by SES [30].

In terms of contribution to the understanding of intervention generated inequalities an important weakness of the study was the observational nature of the study. Whilst significant associations have been found, causation cannot be determined as it was not clear whether the associations between health and an intervention by SES were evidence of the inverse care law, where care was not provided based on need. Alternatively, the findings indicate that the interventions in question have a differential efficacy for patients by SES. In either context, inequalities have occurred at some stage of the intervention process and further research is required to unpick the particular set of circumstances, to enable this to happen. The next sections draws upon research elsewhere to discuss to the possible circumstances which have enabled inequalities to occur at some stage of the intervention process.

## **Strengths and limitations in relation to other studies**

The results from the systematic review in chapter three showed that there were inconsistent findings in socio-economic inequalities, as measured by education, employment, income and composite measures, in intermediate and long-term complications [92, 95, 113, 114, 116, 118, 121]. The results presented here have added to this evidence base using repeat measurements with interaction effects between SES and time, methods that were not utilised by any of the studies in the review.

In comparison to the existing evidence, the results in chapter six add support to the cross-sectional analyses conducted in Germany, which found inequalities in HbA1c but not in long-term complications by SES, occupation and education. The German study did, however, find evidence socio-economic inequalities in retinopathy but in only one of the two sources of data used in the analyses [108]. The statistical significant inequalities in HbA1c levels by SES in the

German study was also not accounted for by differences in age, sex, duration, obesity, diabetes medication and physical activity. The results in chapter six showed, with the exception of one incidence of socio-economic inequalities in ICD rates, there were no inequalities in long-term complications. This was in contrast to the one study, included in the review, which utilised longitudinal data to measure time to first CV event for patients in New Zealand. This study used a cox proportional hazards model and found significant inequalities by SES [116]. This highlights one of the drawbacks of the analyses outlined whereby vascular disease was recorded as in the diabetes register as history of vascular disease but it was not clear when this event occurred. Another limitation was the relatively low prevalence of long-term complications, coupled with the poor recording rates of microalbuminuria and retinopathy, which made determining inequalities by SES difficult.

Only one other study in the systematic review examined patients' health status at diagnosis and found no inequalities by SES [168]. The study from the review and the analyses outlined in this thesis both used data collected by a NHS Trust. The contrasting results may be due to the outcome variables used being different. Retinopathy develops after prolonged uncontrolled HbA1c. The data extraction process for this analysis meant that it included all data during patients first year of diagnosis which could arguably be interpreted as an analysis of inequalities of HbA1c as a consequence of patients first year of care.

Three studies in the systematic review examined inequalities in prescriptions of treatments by indicator(s) of SES. Two of these studies which focused on diabetes treatments using univariate analyses, found no inequalities in being managed by diet alone [92] and prescriptions of diabetes medication by SES [108]. In contrast, cross-sectional multiple regression analyses conducted in New Zealand found the most deprived patients were significantly less likely to be prescribed a combination therapy use, that is, a statin or other lipid lowering medication and an ACEI or other anti-hypertensive treatment [101]. The analyses here, therefore, have contributed to an under researched area and were able to examine trends over time and importantly controlled for patients health status.

One study in the systematic review examined inequalities by SES across interventions broadly categorised as services. The study conducted in Spain found that the average number of consultations per year were higher amongst lower status patients [113], something which could not being examined here due to the method of data extraction from the register. No other studies in the review examined inequalities in quality of care or place of care by SES.

There were limited comparisons to be made with studies included in the systematic review with analyses examining the impact of general practices on inequalities by SES. No other study that

met the systematic review criteria used QOF data. This is likely because the QOF diabetes domain targets and measures type 1 and type 2 patients together, therefore, it makes sense to investigate the impact of this policy intervention on both groups of patients together.

Two Australian studies used multilevel analyses to adjust for clustering of management processes [110] and cardiovascular risk factors [120] at patient, practice and division levels. Both studies found that the results were greatly affected by clustering at the practice level. These findings contrast with the results in this chapter but this may be due to the differences in the number of practices included in the samples. The Australian study used the same population covering 250 practices in 16 Divisions. This study featured patients from 43 different practices. Conducting multilevel analyses of a greater number of general practices in England would help to establish, with more certainty, the impact of general practices on diabetes care and patients outcomes.

A systematic review examining whether the introduction of QOF has improved the management of diabetes in the UK found evidence to suggest that the policy has accelerated improvements but it is difficult to distinguish this from other national initiatives [213]. While the analyses conducted in chapter nine suggests that there has been an improvement in HbA1c and cholesterol since 2004 the limited significant QOF variables suggest that these had not contributed to this trend. In addition, the graphical analyses of HbA1c, cholesterol and quality of care in chapters five all indicated that trends towards better outcomes started several years before QOF was introduced.

## **Meaning of the study and implications for practitioners and policy makers**

Overall, the results from the secondary data analyses in this thesis indicate that patients' health has improved over time. These were likely to reflect the changes in diabetes care, which were also evidenced over the same period. However, there were a number of examples where inequalities associated with the management of type 2 diabetes where further work may be required to improve care for all patient groups.

Whilst there were socio-economic inequalities in intermediate outcomes, these results are potentially reassuring to diabetes practitioners and policy makers as there were no significant inequalities in long-term complications over time. However, further analyses should be conducted to support and confirm these results as the systematic review, and the wider

evidence base, indicates that these findings remain variable. For example, a review of studies across Europe over a similar time period found that higher mortality rates in more disadvantaged patients as a consequence of type 2 diabetes [44]. The findings from this review suggests that patients would have poorer control and long-term complications thereby resulting in differences in mortality rates.

One explanation for the discrepancy between intermediate and long-term complications could be survivor bias, with higher mortality rates from long-term complications amongst low SES patients. Another study conducted using the South Tees diabetes register found that excess mortality was significantly worsen with increase deprivation [159]. This data examined the 5 year period before the data used here and examined both type 1 and type 2, however, the results would support the theory that excess mortality may bias the examination of inequalities in long-term complications. Other studies examining both type 1 and type 2, that did not meet the systematic review criteria and are not directly comparable, have found evidence of socio-economic status being associated with increased prevalence of long-term complications [214-217]. As type 2 diabetes account for the majority of these samples the discrepancy between the results is unlikely to be due to the inclusion of type 1 diabetes patients in these analyses.

One explanation for inequalities in HbA1c level by SES in Germany put forward by the authors was that more disadvantaged patients were receiving a level of care inappropriate to their needs. The current analyses were able to control for this and the stepwise analyses indicated that they diabetes care only partially attenuated inequalities in HbA1c levels. As such, these findings could potentially suggest that diabetes care was not delivered equitably in order to overcome the inequalities that occur in intermediate outcomes.

The theory that more disadvantage patients were receiving a level of care inappropriate to their needs was evidenced throughout the secondary analyses. The descriptive analyses showed that high SES patients had a higher mean number of care process recorded compared to low patients, though they were less likely to receive shared care. Interestingly, high SES patients were significantly more likely to receive shared care than mid SES patients. These trends were also seen over time in the graphical analyses and multilevel analyses where quality of care and shared care were included as explanatory variables and where they were modelled as dependent variables.

In terms of explanatory variables, few were statistically significant in the model that had quality of care as the dependent variable (**Table 13**). Though age was significant, it supported the NDA findings that younger patients receive poor quality of care. However, patients aged 75 years and over were more likely to receive poorer care compared to those aged less than 60 years [218].

The significance of shared care in this model has suggested that patients were likely to receive greater quality of care in the secondary care compared to primary care. This may be because diabetes specialist practitioners are more responsive of the clinical guidelines and experienced at administering the nine recommended care processes compared to general practitioners. It could also be due to the use of the diabetes register proforma, and interest in maintaining the dataset being higher in the diabetes care centre where the register is hosted. Using a proforma may be a successful intervention in itself for improving care [219, 220].

Contrary to this argument and the inverse care law, which suggests care inversely related to patient need [221], shared care may be acting as a proxy indicator of patients who have poorer health and therefore needing more specialist care and greater quality of care. That is, the receipt of care was based on the complexity of patients' disease management. The receipt of shared care and higher quality of care were significantly associated with prescriptions of diabetes treatments regimes. There was also evidence which suggested that interventions during patients' first year of diagnosis were found to mediate inequalities in HbA1c by SES at diagnosis. This could indicate that care administered during patients' first year of diagnosis was, at least, administered appropriately. Whilst the relationship with shared care was expected, patients with more complex needs are more likely to be referred for specialist care. The relationship with quality of care was not expected to occur which could be regarded as appropriate with more care being based on greater need. However, without undertaking all care processes, early detection of complications could be missed and appropriate action not taken in order to prevent or treat the problem.

In addition, there was also evidence relating to the uptake and health association by SES with shared care in chapter eight. These findings showed that shared care was associated with more favourable HbA1c and rates of microalbuminuria for high SES patients compared to low SES patients. In contrast, mid SES patients were more likely to have microalbuminuria compared to low SES patients when both using shared care. Furthermore, there were significant interactions between quality of care and SES. The results suggest that high SES had poorer HbA1c and mid SES had poorer rates of PVD compared to low SES receiving shared care. This may be because higher SES patients only receive increased quality of care when their health deteriorates. It could also indicate a potential source of reducing inequalities, that is, by increasing the quality of care in low SES patients it could improve their HbA1c and reduce the significant differences, compared to high SES who were found to receive higher quality of care and healthier HbA1c levels overall and over time.

There has been a wide range of research investigating socio-economic inequalities associated with specialist services (e.g. [222-224]). The differences in shared care use may be due to lower SES patients having more barriers to accessing specialist services, such as transport costs, inflexible service delivery and cultural barriers such as language [223]. In addition, high SES may be more assertive in requesting specialist care compared to low SES [222]. One study also suggested that general practitioners in deprived areas may be generally less likely to refer overall rather than more socially disadvantaged patients being less likely to be referred than more advantaged patients within the same practice [224]. Yet, the results from chapter seven showed that patients in Middlesbrough PCT were more likely to receive shared care and the descriptive analyses indicated that practices in this PCT served more deprived populations overall compared to Redcar & Cleveland PCT. For this study population the differences in referral rates may be related to the diabetes care centre being located in Middlesbrough and factors such as travel times becoming a major contributor as more practices in Middlesbrough are located in urban places compared to Redcar & Cleveland, which is a much larger, mainly rural area. The cultural barriers and high SES patients' assertiveness may also explain the differences in the association with HbA1c levels and shared care by SES as there may be more effective communication between these patients and specialists. The contrasting results in the interaction effects between PCT with cholesterol level and microalbuminuria by SES are hard to explain and require further investigation. However, it suggests that patients interact with factors measured at the PCT level differently.

Patients' age may also be an important explanatory factor of where there were inequalities in the receipt of long-term complications and level of care. In the analysis of the NDA, being younger at the onset of diabetes was found to be associated social deprivation and the social gradient in type 2 diabetes prevalence was more common in patients aged less than 55 years compared to older patients. In addition, younger patients were also more likely to receive poorer quality of care [218]. The secondary analyses here supported the NDA findings, with older patients significantly more likely to receive share care. The relationship between younger patients and care may also be explained by younger patients experiencing greater barriers in engaging with health services, such as work demands, compared to older patients who are more likely to be of retirement age and may already be more engaged with health services.

Another important factor was ethnicity. Having a South Asian ethnicity is also a known risk factor of type 2 diabetes [176]. The results from these analyses show that being South Asian was significantly associated with a poorer HbA1c level at diagnosis. The finding suggesting this group, who along with younger patients, were overrepresented in the lower SES groups in this study population and were being provide with lower quality of care. The significant relationship

between poorer HbA1c at diagnosis and South Asian ethnicity may be a result of ineffective communication between patients and providers and differences in cultural values, health behaviours and healthcare preferences compared to the white population [225]. These explanations were given in the possible mechanisms in the inequalities in diagnostic delays found by age, ethnicity and social class in six common cancers. In addition, different levels of knowledge of symptoms and access to services were also discussed as possible mechanisms [226]. However, in contrast, South Asian patients were significantly more likely to receive greater quality of care compared to white patients. In addition, South Asian patients were also significantly likely to have both poor and more favourable health outcomes, therefore, these explanations are not consistent across all diabetes care.

By being able to control for patients health status and other relevant factors these analyses were able to determine whether treatments were being administered systematically. The results in chapter seven showed that there were significant inequalities over time in the prescription of all treatments except for combination of diabetes treatments with no insulin, ACEI only and in combination with other BP treatments, lipid therapies and aspirin following adjustment for patients' health status and other factors. The statistically significant trends in prescription rates in the descriptive analyses were not simply a reflection of inequalities in health status. In addition to the inequalities in prescription of appropriate treatments, the results from chapter eight indicated that there were significant differences by SES in the association with health outcomes with particular treatments. Namely, being prescribed insulin, solely and in combination, ACEI only, BP treatments with no ACEI, aspirin and lipid therapies.

The results in chapter seven indicated that there were no differences in the prescriptions in lipid therapies and aspirin by SES but the findings in chapter eight showed that more favourable levels of cholesterol levels for higher SES patients being prescribed these treatments compared to low SES patients. These results may be a consequence of delays in the initiation of these treatments and/or non-adherence for low SES patients. Mid SES patients prescribed lipid therapies were also more likely to have better microalbuminuria rates compared to low SES patients. These findings were in contrast to a UK wide study which analysed socioeconomic trends in CHD mortality. The authors found that medical treatments accounted for a 50% decline in rates over 2007 to 2007 and the effect was equitable across SES patient groups [199].

Delays in the initiation and non-adherence may also explain the significant inequalities in four out of five diabetes treatments over time. Discrepancies between guidelines and treatment practices have been found worldwide [227, 228]. Though these studies have not examined whether these discrepancies in the adherence to treatment guidelines were socially stratified.

Factors such as patients' attitudes to their condition and healthcare, lack of resources and co-morbidities were suggested as contributing to these differences and it possible that these are related to SES.

Discrepancies between guidelines and treatment practice may also contribute to the differences in the associated between insulin, both on its own and in combination with other therapies, and HbA1c and incidences of ICD, microalbuminuria and retinopathy by SES. Higher SES patients were more likely to have more favourable outcomes on these treatments than low SES.

Interestingly, mid SES patients were more likely to have poorer microalbuminuria rates when prescribed insulin only compared to low SES patients. Using insulin requires timely recommendation and effective implementation of the treatment, which then must be adhered to by the patient and should then be continually reviewed and if necessary intensified. This requires the patient to adopt a new treatment regimen. A recent international survey examined groups of patients and practitioners and their attitudes to insulin use and found that explanations given for insulin non-adherence were similar from both groups. These included skipping meals, logistical problems such as being busy and travelling, psychosocial problems such as stress, emotions and embarrassment. Patients also suggested forgetting was another issue. Using insulin was also perceived negatively with groups seeing the treatment regimen as restrictive and wanted it to be more flexible in accordance with daily activities [228]. Whilst the authors of this study did not analyse its findings by SES and these problems are likely to be experienced across the social spectrum. However, high SES patients may have more resources to draw upon to overcome these barriers compared to low SES patients. A finding, which is of particular relevance to practitioners and policy makers, was the amount of variation at the general practice level of patients being managed by diet alone. It was not clear from these analyses whether this indicates that some practices are better at delaying patients' progression to treatments or whether these delays were appropriate or not and should be investigated further.

As general practice spending is such a political issue at present [13], the results relating to QOF data were disappointing as the data yielded little statistical significance. The evidence presented in this thesis, in terms of using QOF data and the adjustment for clustering at the practice level, suggested that interventions targeted at the practice level have limited impact on the improvement of diabetes patients' care and outcomes. However, there are a number of criticisms of QOF in its current inception, including the lack of targets set to improve patients communication, engagement and empowerment [213] and the notable differences between NICE and QOF treatment targets. Initiatives, such as QOF, for diabetes care in their current form, are likely to have a limited role in improving care and reducing inequalities by SES.

The NICE Type 2 diabetes care guideline [46], NSF for Diabetes [49, 52], QOF [47], NDA [50] and the NHS Diabetic Eye Screening Programme [48] are all designed to improve and systematically deliver equitable care for all diabetes patients. However, whilst these policies and programmes may have played a role in improving care and health outcomes for patients overall during the study period, the results presented here have shown that there were socio-economic inequalities associated with outcomes and interventions. This is particularly evident in the intermediate health outcomes and the receipt of quality of care, combinations of BP treatments and shared care over time and the different association with health outcomes by SES in patients using insulin and lipid treatments, shared care and being managed by PCT.

The evidence presented in this thesis arguably supports many of the previous theories regarding intervention generated inequalities. Gaining effective diabetes control requires long-term engagement with health services as well as making substantial lifestyle changes and adapting to changes in treatments. Whilst practitioners are there to support and ensure patients receive the appropriate care in a timely manner, interventions such as attending retinal screening services, attending annual reviews and adhering to treatment regimens can be described as ‘agentic’ interventions as they rely upon individuals sustaining behaviour change [229]. However, evidence published elsewhere suggests that these types of interventions actually increase inequalities as they do not address the exposure to the risk factors [28]. In the case of type 2 diabetes, interventions investigated here do not tackle social environment and circumstances, which lead them to develop the condition and are more reactionary as patients’ health deteriorate. That is, they are not ‘structural strategies’ which have been found to have a more equitable impact on population health [28, 229] [1].

There was also evidence to support Ali’s [33] argument that the way health systems operate and the personalisation of the NHS could exacerbate inequalities [33]. In particular, the receipt of shared care and its association with health outcomes varies by SES. This highlights that the current arrangements of this care may not be meeting all patients’ needs equally. Addressing this disparity may require examining the referral practices of GPs, who act as gate-keepers to this care, or if the service could be redesigned to ensure that patients from low SES groups are able to overcome the personal and structural barriers which prevent them benefitting from this care.

A recent review examining interventions which improve care in socially disadvantaged diabetes patients found that culturally tailoring, community educators, one-to-one interventions, treatment algorithms, focusing on behaviour-related tasks, patient feedback and high intensity interventions were found to have consistent impact on reducing inequalities [230]. Many of

these interventions would address some of the explanations given as to why there were inequalities found in these analyses. There are many potential opportunities for diabetes practitioners and policy makers to improve patients' health and address where there were socio-economic inequalities in patients health outcomes and receipt of care. Many of these interventions would require long-term planning and investment. In the short term, practitioners should ensure that all patients receive the recommended care processes and that treatments algorithms implemented appropriately, as outlined in NICE guidelines. Not following these recommendations for all patients have been shown to be possible sources of intervention generated inequalities.

## Unanswered questions and future research

The findings from this thesis indicates that in most circumstances there were no evidence of inequalities associated with type 2 diabetes health and care in the South Tees area. However, there were notable exceptions and further research is required to fully understand how this arises. Due to the observational nature of the analyses there are many areas which could be expanded upon to determine causation. However, what is particularly important, where there was evidence of intervention generated inequalities, is to unpick whether these were a result of interventions not being appropriately accessed and/or administered based on need or if the efficacy of these interventions differed by SES.

If inequalities arise due to efficacy of the interventions by SES, future research should be conducted to determine which strategies could be implement to avoid this. The work by Glazier *et al* [230] has found that there are interventions which could implemented to improve the care of the most disadvantaged populations. Particularly, the incorporation of treatment algorithms, providing intense care over long periods and interventions based on health behaviours. These could be implemented locally and evaluated to establish if they work for the type 2 diabetes population in South Tees.

Equitable access by patients need is particularly important in context of the new health landscape as general practitioners in the form of Clinical Commissioning Groups (CCGs) are now in control of the majority of the health care budget. This means that they decide what healthcare is commissioned and who can access these services. General Practitioners and CCGs therefore are dominant gatekeepers to health care services [13, 15, 16]. The new reforms may ensure access to specialist diabetes care may even become more equitable across South Tees as the 49

general practices in South Tees now make up the South Tees Clinical Commissioning Group [231]. This Group aims to work closely with South Tees Hospitals NHS Foundation Trust as well as the constituent general practices working closely to reduce health inequalities and improve health and wellbeing of their patients [231]. These findings could potentially inform the priorities of this clinical commissioning group as well as encouraging other CCGs and specialist services to reflect upon whether they are providing equitable care and health outcomes for their patients.

## Appendix A. The terms and strategies used to search each database in the systematic review

### Pubmed (U.S. National Library of Medicine National Institutes of Health):

- #1 (diabetes[Title/Abstract] or diabetic[Title/Abstract])
- #2 ("type 2" OR "type II" OR "type two" OR "non\*insulin\*dependent" OR NIDDM)
- #3 ((sex[Title/Abstract] or gender[Title/Abstract] or ethnicity[Title/Abstract] or ethnic[Title/Abstract]) and (inequalit\*[Title/Abstract] or inequit\*[Title/Abstract] or disparit\*[Title/Abstract] or equit\*[Title/Abstract] or bias[Title/Abstract]))
- #4 (deprived[Title/Abstract] or deprivation[Title/Abstract] or income[Title/Abstract] or poverty[Title/Abstract] or education\*[Title/Abstract] or "social class\*" [Title/Abstract] or "socio\*economic class\*" [Title/Abstract] or "socio\*economic status" [Title/Abstract] or "socio\*economic position" [Title/Abstract] or "socio\*economic factor\*" [Title/Abstract] or (urban[Title/Abstract] AND rural[Title/Abstract]))
- #5 #3 OR #4
- #6 ("Spain" or "Portugal" or "Greece" or "Italy" or "Great Britain" or "United Kingdom" or "Scotland" or "Wales" or "Northern Ireland" or England or "Ireland" or "France" or "Germany" or "Austria" or "Belgium" or "Netherlands" or "Holland" or "Denmark" or "Finland" or "Norway" or "Sweden" OR Swedish or "Canada" or "Japan" or "Australia" or "New Zealand" or "South Korea" or "Luxembourg" or "Iceland")
- #7 #1 AND #2 AND #5 AND #6
- #8 "Animals"[Mesh]
- #9 "Humans"[Mesh]
- #10 #8 NOT #9
- #11 #7 NOT #10
- #12 #11 Limits English language, 1998 onwards

### Embase (OvidSP, Wolters Kluwer Health):

- 1 (diabetes or diabetic).ti,ab.
- 2 limit 1 to (english language and yr="1998 -Current")
- 3 ("type 2" or "type II" or "type two" or "non\*insulin\*dependent" or NIDDM).af.

- 4 limit 3 to (english language and yr="1998 -Current")  
5 ((sex or gender or ethnicity or ethnic) and (inequalit\* or inequit\* or disparit\* or equit\*  
or bias)).ti,ab.  
6 limit 5 to (english language and yr="1998 -Current")  
7 (deprived or deprivation or income or poverty or education\* or social class\* or  
socio\*economic class\* or socio\*economic status or socio\*economic position or  
socio\*economic factor\* or (urban and rural)).ti,ab.  
8 limit 7 to (english language and yr="1998 -Current")  
9 6 or 8  
10 ("Spain" or "Portugal" or "Greece" or "Italy" or "Great Britain" or "United Kingdom" or  
"Scotland" or "Wales" or "Northern Ireland" or England or "Ireland" or "France" or  
"Germany" or "Austria" or "Belgium" or "Netherlands" or "Holland" or "Denmark" or  
"Finland" or "Norway" or "Sweden" or Swedish or "Canada" or "Japan" or "Australia" or  
"New Zealand" or "South Korea" or "Luxembourg" or "Iceland").af.  
11 limit 10 to (english language and yr="1998 -Current")  
12 2 and 4 and 9 and 11  
13 exp humans/  
14 exp animals/  
15 14 not 13  
16 12 not 15

### CINALH (Ebscohost):

- S1 TI ( (diabetes or diabetic) ) or AB ( (diabetes or diabetic) ) Search modes -  
Boolean/Phrase  
S2 ("type 2" or "type II" or "type two" or "non\*insulin\*dependent" or NIDDM) Search  
modes - Boolean/Phrase  
S3 TI ( ((sex or gender or ethnicity or ethnic) and (inequalit\* or inequit\* or disparit\* or  
equit\* or bias)) ) or AB ( ((sex or gender or ethnicity or ethnic) and (inequalit\* or  
inequit\* or disparit\* or equit\* or bias)) ) Search modes - Boolean/Phrase  
S4 TI ( (deprived or deprivation or income or poverty or education\* or social class\* or  
socio\*economic class\* or socio\*economic status or socio\*economic position or  
socio\*economic factor\* or (urban and rural)) ) or AB ( (deprived or deprivation or  
income or poverty or education\* or social class\* or socio\*economic class\* or

- socio\*economic status or socio\*economic position or socio\*economic factor\* or (urban and rural)) ) Search modes - Boolean/Phrase
- S5 S2 or S4 Search modes - Boolean/Phrase
- S6 ("Spain" or "Portugal" or "Greece" or "Italy" or "Great Britain" or "United Kingdom" or "Scotland" or "Wales" or "Northern Ireland" or England or "Ireland" or "France" or "Germany" or "Austria" or "Belgium" or "Netherlands" or "Holland" or "Denmark" or "Finland" or "Norway" or "Sweden" or Swedish or "Canada" or "Japan" or "Australia" or "New Zealand" or "South Korea" or "Luxembourg" or "Iceland") Search modes - Boolean/Phrase
- S7 S1 and S2 and S5 and S6 Search modes - Boolean/Phrase
- S8 S7 Limiters - Published Date from: 19980101-20121231; English Language

### ASSIA search:

- #1 TI=(diabetes or diabetic) or AB=(diabetes or diabetic)
- #2 "type 2" or "type II" or "type two" or "non\*insulin\*dependent" or NIDDM
- #3 TI=((sex or gender or ethnicity or ethnic) and (inequalit\* or inequit\* or disparit\* or equit\* or bias)) or AB=((sex or gender or ethnicity or ethnic) and (inequalit\* or inequit\* or disparit\* or equit\* or bias))
- #4 AB=(deprived or deprivation or income or poverty or education\* or social class\* or socio\*economic class\* or socio\*economic status or socio\*economic position or socio\*economic factor\* or (urban and rural)) or TI=(deprived or deprivation or income or poverty or education\* or social class\* or socio\*economic class\* or socio\*economic status or socio\*economic position or socio\*economic factor\* or (urban and rural))
- #5 #3 OR #4
- #6 "Spain" or "Portugal" or "Greece" or "Italy" or "Great Britain" or "United Kingdom" or "Scotland" or "Wales" or "Northern Ireland" or England or "Ireland" or "France" or "Germany" or "Austria" or "Belgium" or "Netherlands" or "Holland" or "Denmark" or "Finland" or "Norway" or "Sweden" or Swedish or "Canada" or "Japan" or "Australia" or "New Zealand" or "South Korea" or "Luxembourg" or "Iceland"
- #7 #1 AND #2 AND #5 AND #6

## Appendix B: Copies of access and ethical approval letters

- School of Medicine and Health Ethics Committee, Durham University  
Ref: ESC2/2010/12
- County Durham & Tees Valley Research Ethics Committee, National Research Ethics Service  
Ref: 10/H0908/63
- Research & Development / Academic Division, South Tees Hospitals NHS Foundation Trust



Wolfson Research Institute  
Improving health and well-being

**Rebecca Perrett**

Research and Development Manager, Wolfson Research Institute  
Chair, School of Medicine and Health Ethics Committee

Tel: 0191 334 0425

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**Anna Christie**  
PhD Student  
North East Public Health Observatory  
The Wolfson Research Institute  
Durham University Queen's Campus  
Stockton-on-Tees  
TS17 6BH  
United Kingdom

6<sup>th</sup> October 2010

Dear Anna,

**RE: Do interventions generate inequalities? An Analysis of inequalities in the access to, uptake of and impact of Type 2 diabetes health interventions in South Tees**

**Ref: ESC2/2010/12**

Thank you for sending your revisions to the above application to the School of Medicine and Health ethics committee. These have now been reviewed and I am satisfied that all of the points raised by the committee have been addressed. I can therefore grant you SMH ethics approval to conduct the study.

Please do not hesitate to contact me should you have any questions. I hope that the study goes well.

With best wishes

A handwritten signature in black ink that reads "R Perrett".

Rebecca Perrett



**National Research Ethics Service**  
County Durham & Tees Valley Research Ethics Committee

Room 002  
TEDCO Business Centre  
Viking Industrial Park  
Rolling Mill Road  
Jarrow  
Tyne & Wear  
NE32 30T

Telephone: 0191 428 3556  
Facsimile: 0191 428 3432

05 November 2010

Ms Anna Christie  
PhD Student  
University of Durham  
NEPHO, Wolfson Research Institute  
Durham University Queen's Campus  
Stockton-on-Tees  
TS17 6BH



Dear Ms Christie

**Study Title:** Do interventions generate inequalities? An analysis of inequalities in the access to, uptake of and impact of Type 2 diabetes health interventions in South Tees.  
**REC reference:** 10/H0908/03  
**Protocol number:** T2001

The Proportionate Review Sub-committee of the County Durham & Tees Valley Research Ethics Committee reviewed the above application at the meeting held on 05 November 2010, via email correspondence.

#### Ethical opinion

Members noted the following issue:

It was queried whether there should be a letter of agreement from the Diabetes Register Manager regarding the search and anonymisation of data as this could be time consuming. However, upon further inspection, it was elicited from the application form that one of the key collaborators was, in fact, the Chief of Service for the department concerned - Members agreed it could be assumed therefore that approval for this was already in place.

Members confirmed that the application contained no material ethical issues.

The members of the Committee present gave a favourable ethical opinion of the above research on the basis described in the application form, protocol and supporting documentation, subject to the conditions specified below.

#### Ethical review of research sites

The favourable opinion applies to all NHS sites taking part in the study, subject to management permission being obtained from the NHS/HSC R&D office prior to the start of the study (see "Conditions of the favourable opinion" below).

### Conditions of the favourable opinion

The favourable opinion is subject to the following conditions being met prior to the start of the study.

Management permission or approval must be obtained from each host organisation prior to the start of the study at the site concerned.

*Management permission ("R&D approval") should be sought from all NHS organisation(s) involved in the study in accordance with NHS research governance arrangements. Guidance on applying for NHS permission for research is available in the Integrated Research Application System (IRAS) or at <http://www.rdforum.nhs.uk>*

*Where a NHS organisation's role in the study is limited to identifying and referring potential participants to research sites ("participant identification centre"), guidance should be sought from the R&D office on the information it requires to give permission for this activity.*

*For non-NHS sites, site management permission should be obtained in accordance with the procedures of the relevant host organization.*

*Sponsors are not required to notify the Committee of approvals from host organizations.*

**It is the responsibility of the sponsor to ensure that all the conditions are complied with before the start of the study or its initiation at a particular site (as applicable).**

### Approved documents

The documents reviewed and approved at the meeting were:

Document	Version	Date
Investigator CV – Anna Christie	1	22 October 2010
Protocol	T2DD1	22 October 2010
Cv Professor J Wilkinson	1	25 October 2010
REC application	IRAS 3.0	25 October 2010
Covering Letter	1	25 October 2010
Letter from Sponsor	1	06 October 2010
Email correspondence from NIGB	1	03 August 2010
Evidence of insurance or indemnity	1	02 July 2010
Letter from Statistician	1	22 October 2010

### Membership of the Proportionate Review Sub-Committee

The members of the Sub-Committee who were present at the meeting are listed on the attached sheet.

### Statement of compliance

The Committee is constituted in accordance with the Governance Arrangements for Research Ethics Committees (July 2001) and complies fully with the Standard Operating Procedures for Research Ethics Committees in the UK.

### After ethical review

Now that you have completed the application process please visit the National Research Ethics Service website > After Review

You are invited to give your view of the service that you have received from the National Research Ethics Service and the application procedure. If you wish to make your views known please use the feedback form available on the website.

The attached document "After ethical review – guidance for researchers" gives detailed guidance on reporting requirements for studies with a favourable opinion, including:

- Notifying substantial amendments
- Adding new sites and investigators
- Progress and safety reports
- Notifying the end of the study

The NRES website also provides guidance on these topics, which is updated in the light of changes in reporting requirements or procedures.

We would also like to inform you that we consult regularly with stakeholders to improve our service. If you would like to join our Reference Group please email [referencegroup@nres.npsa.nhs.uk](mailto:referencegroup@nres.npsa.nhs.uk).

10/H0908/63	Please quote this number on all correspondence
-------------	--

With the Committee's best wishes for the success of this project

Yours sincerely



 Dr John Drury  
Chair

Email: [leigh.pollard@nhs.net](mailto:leigh.pollard@nhs.net)

*Enclosures: List of names and professions of members who were present at the meeting and those who submitted written comments*

*"After ethical review – guidance for researchers"*

*Copy to: Professor D Petley, Deputy Head of Faculty (Research), Durham University, Durham, DH1 3YG  
Professor J Wilkinson, Wolfson Research Institute, Durham University, Queen's Campus, Stockton-on-Tees, TS17 6BH  
R&D Office, James Cook University Hospital, Middlesbrough, TS4 3BW*

**County Durham & Tees Valley Research Ethics Committee**

**Attendance at PRS Sub-Committee of the REC meeting on 05 November 2010 via  
email correspondence**

**Committee Members:**

<i>Name</i>	<i>Profession</i>	<i>Present</i>	<i>Notes</i>
Mrs S Brooks	Retired Health Visitor	Yes	Alternate Vice Chair – Lay Member
Dr John Drury	Consultant Pathologist	Yes	Chair – Expert Member
Mrs F Hutchison	Principal Teacher	Yes	Lay Member
Dr EM Scott	Research Advisor	Yes	Expert Member

South Tees Hospitals   
NHS Foundation Trust

Research & Development / Academic Division  
Academic Centre  
The James Cook University Hospital  
Marton Road  
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[www.southtees.nhs.uk](http://www.southtees.nhs.uk)

Tel: 01642 282585

Email: [julie.rowbotham@stees.nhs.uk](mailto:julie.rowbotham@stees.nhs.uk)

17 May 2013

Ms Anna Christie  
PhD Student  
University of Durham  
NEPHO Wolfson Research Institute, Queens Campus  
Stockton on Tees  
TS17 6BH

Dear Ms Anna Christie

**Re: 2010105 - Do interventions generate inequalities? An analysis of inequalities in the access to, uptake of and impact of Type 2 diabetes health interventions in South Tees.**

Thank you for submitting your project to Research & Development. After review we concluded that your work falls into the category of Service Development and poses no unacceptable governance or ethics issues. We will register your project with Research and Development and wish you well with your study.

Kind regards.

**Mr A Owens**  
Chairman of Research Approval Board  
GMC 3485934

## Appendix C: Data source and variable construction

This appendix outlines the name of each variable, where it was sourced, what it measures and any additional formatting.

Table 42: Socio-demographic, socio-economic, anthropometric and lifestyle data

Variable name	Source	Measurement/derivation/formatting
Visit year	Diabetes register	Refers to the year in which the patients visit was recorded. Derived from 'Date of Visit' which was the last visit the patient had during a calendar year. Each variable included was the latest recording of that variable in the calendar year recorded.
Age		Subjects' age in years at the end of 2007 was part of the original data extraction, derived from patients' date of birth by the register data manager. 'Age' was constructed to establish patients' age at the end of the year in which the visit was recorded and calculated as follows: [Age end 2007]-(2007-[Visit year])
SES	ONS, Diabetes register	Patients' socio-economic status was measured using Index of Multiple Deprivation 2004. A table of postcodes in the South Tees area and the lower super output area (LSOA) the majority each postcode falls into was linked by Database Manager using to register extract via patients address. The addresses were then removed.  The national rank of all England LSOA according its Index of Multiple Deprivation 2004 score was extracted from ONS data. These ranks were divided into quintiles with 1 indicating the lowest socio-economic status group. These were then assigned to the register extract via patients LSOA for that year.  Quintiles were used for the descriptive analyses, however, the quintiles three, four and five (the three highest SES groups) were then recoded into one group for the subsequent analyses.
Age at diagnosis	Diabetes register	The year which patients were diagnosed was extracted from the diabetes register. The age in years at the end of the year the

Duration of diabetes	<p>patient was diagnosed and calculated as follows:  <math display="block">[\text{Age end 2007}] - (2007 - [\text{Year of Diagnosis}])</math> This variable measures the years since diagnosis to current year of visit. Calculated as follows: <math>[\text{Age}] - [\text{Age at diagnosis}]</math>. Recording of '0' indicates that the visit was recorded the same year the patient was diagnosed.</p> <p>The year the patient was diagnosed as recorded in the register. 112 records (85 patients) had the year of diagnosis as a year following the recorded visit. These values were removed.</p>
Sex	<p>This is recorded in the register as 1 = Male, 2 = Female. One patient did not have their sex recorded so all their data was removed.</p>
Ethnicity	<p>In the register patients have their ethnic origin recorded as one of four categories: Europid, South Asian, Afro-Caribbean and Other. 46 patients were recorded as Afro-Caribbean and 74 patients as 'Other' therefore to give more power to the analysis these two categories were grouped into one category. As such Ethnicity has three categories coded as follows: 1 = White, 2 = South Asian and 3 = 'Other'</p>
Weight	<p>Subjects weight in kilograms (kg).</p> <p>Further formatting: On the recommendations by the register staff values outside the range 0 to 220 were considered to be a result of inaccurate recording and were removed.</p>
BMI	<p>Body mass index is calculated from height and weight: <math>(\text{kg}/\text{m}^2)</math> by the data input team. BMI and Height was not extracted from the register directly, however, it calculated from weight and BMI fields as follows: <math>\sqrt{(\text{Weight}/\text{BMI})}</math>. This was done to check the validity of the weight and BMI fields which were extracted from the register. Following recommendations height was limited to values greater than or equal to 0.8 and less than or equal to 2.1 metres, with values outside this range were removed. By producing a cross tabulated table of subjects' height per year it was clear from eye-balling the data that approximately 33% of the study population's height ranged by over 10cms. In order to reduce the bias resulting from measurement error subjects' median height was calculated for subjects with three or more</p>

Smoking status	<p>height recordings over the study period. By using median instead of mean reduces the influence that extreme values have on the final statistic.</p> <p>Due to the nature of diabetes, many patients can experience sudden weight gain and loss over a short period of time. It would be impossible to judge whether any large variation in subjects' BMI was a result of recording and/or measurement errors or an accurate reflection of their body mass due to changes in health and/or result of treatments. To ensure that it is not a recording or measurement error of subjects' height or weight BMI was recalculated once the extreme values of these indicators were removed and using the median height of each subject. The new values which were greater than 100 were removed.</p> <p>Patients' smoking status was recorded in the diabetes register using the following categories: 0 = No, 1 = Yes, 2 = Ex. All three categories should be offered to subjects as options for the self report of their smoking status. However, from eye balling the data it was clear that some patients have been categorised as non-smokers even though they have been recorded as a smoker in previous years. As such any recording of '0' following a recording of '1' in an earlier year was changed to a recording of '2' to reflect the previous and current smoking status per patient. This was based on the assumption that an ex-smoker was more likely to be recorded as a non smoker than an inaccurate recording of being a smoker previously.</p>
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Table 43: Intermediate outcomes and long-term complications

Variable name	Source	Measurement/derivation/formatting
sBP	Diabetes register	Systolic blood pressure measured in mmHg.
		Further formatting: as recommended by the register staff all values which were equal to or less than the patient's diastolic blood pressure values were removed. No specific limits were given.
dBp		Diastolic blood pressure measured in mmHg.

	<p>Further formatting: As recommended by the register staff all values which were equal to or less than its corresponding systolic blood pressure values were removed. No specific limits were given.</p>
Hypertensive	<p>Patients were classified as being hypertensive if their blood pressure was above the NICE recommended target of 130/80 mmHg: 1 = if both patient <math>DBP \leq 80</math> And their <math>sBP \leq 130</math>; 0 = if not.</p>
HbA1c	<p>Glycosylated haemoglobin is measured as a percentage.</p> <p>Further formatting: As recommended by the register staff values were limited to those greater than or equal to 2.5 and less than or equal to 23. Values outside this range were removed.</p>
Cholesterol	<p>Total cholesterol measured in mmol/l.</p> <p>Further formatting: As recommended by the register staff values were limited to those greater than or equal to 1.5 and less than to or equal to 40. Values outside this range were removed.</p>
Creatinine	<p>Creatinine measured in <math>\mu\text{mol/l}</math>.</p> <p>Further formatting: As recommended by register staff values were limited to those greater than or equal to 20 and less than or equal to 1400. Values outside this range were removed.</p>
CreatinineGreater300	<p>This variable derived from Creatinine to indicate records with creatinine measurements greater than 300 <math>\mu\text{mol/l}</math> as '1'; and '0' if not. This was necessary covariate to be controlled for when analysing HbA1c outcomes, as recommended by register staff.</p>
ICD	<p>Recorded as '1' if a patient has ever had a history of ischaemic cardiac disease; and '0' if not.</p> <p>Further formatting: Due to how these indicators are recorded, values of '0' if a subject had a recording of '1' in a previous year.</p>
Stroke or TIA	<p>Recorded as '1' if a patient has ever had a history of stroke or transient ischaemic attack; and '0' if not.</p> <p>Further formatting: Due to how these indicators are recorded, values of '0' if a subject had a recording of '1' in a previous year.</p>
PVD	<p>Recorded as '1' if a patient has ever had a history of peripheral vascular disease; and '0' if not.</p> <p>Further formatting: Due to how these indicators are recorded, values of '0' if a subject had a recording of '1' in a previous year.</p>
Micro-	<p>This is based on a patients' albumin/creatinine ratio (ACR,</p>

albuminuria		mg/mmol). Patients were classified as having microalbuminuria and recorded as '1' if ACR≥3 for men, ACR≥3.5 for women; and '0' if not.
eGFR		This was calculated using abbreviated Modification of Diet in Renal Disease equation as recommended by NICE, SIGN, and Renal Association: $eGFR_{ml/min/1.73m^2} = 186 \times (Creatinine/88.4)^{-1.154} \times (Age)^{-0.203} \times (0.742 \text{ if female}) \times (1.210 \text{ if black}).$ This was kept as a continuous variable, however, the lower the number indicates worse kidney function [66].
Retinopathy	Diabetes register, screening programme	Retinopathy is currently (2010) recorded in the diabetes register as follows: 0 = None, 1 = Background, 2 = Pre-Proliferative, 3 = Proliferative. However, during the study period the way retinopathy has been recorded has changed several times as such the database manager recoded the data as follows: 0 = None, 1 = Background, 2 = Advanced; where advanced retinopathy is anything more serious than background retinopathy. Further formatting: The data from the diabetic retinal screening programme for 2006 and 2007 was recorded as follows: R0M0 = No diabetic retinopathy, no maculopathy; R1M0 = Background diabetic retinopathy, no maculopathy; R1M1 = Background diabetic retinopathy, maculopathy; R2M0 = Pre-proliferative diabetic retinopathy, no maculopathy; R2M1 = Pre-proliferative diabetic retinopathy, maculopathy; R3M0 = Proliferative diabetic retinopathy, maculopathy; R3M1 = Proliferative diabetic retinopathy, maculopathy. However, the prevalence of each these grades were very low, particular at the severe end of the scale. As such this data was recorded into three above categories. A new indicator was created combining these two sources of retinopathy data. In a 180 cases the values between the sources conflicted. In these cases the values from the retinal screening programme were favoured as this a more direct source.

Table 44: Diabetes interventions data

Variable name	Source	Measurement/derivation/formatting
Diagnosis at HbA1c	Diabetes register	This is the HbA1c value measured during the year patient was diagnosed. This variable acts a proxy measurement to the severity of patients' condition at diabetes.
Diabetes treatments		<p>This variable recoded the following diabetes treatments, which are recorded as '1' receiving that treatment and '0' not receiving the treatment, into one categorical variable: diet alone, metformin, sulphonylureas, metformin, acarbose, glitazone and insulin.</p> <p>The new variable were coded based on the NICE type 2 diabetes guidelines as the following:  1 = Diet alone, 2 = Metformin or sulphonylureas only, 3 = Diabetes treatment combination excluding insulin, 4 = Insulin only and 5 = Diabetes treatment combination including insulin.</p>
BP treatments		<p>This variable recoded the following BP treatments, which are recorded as '1' receiving that treatment and '0' not receiving the treatment, into one categorical variable: diuretics, beta blockers, alpha blockers, ACE inhibitors and calcium antagonists The new variable were coded based on the NICE type 2 diabetes guidelines as the following:  1 = No BP treatment, 2 = ACEIs only, 3 = ACEIs plus any combination of other BP treatments and 4 = other treatment(s).</p>
Aspirin		Recorded as '1' if patient was being treated with aspirin, '0' if not.
Lipid therapies		<p>The diabetes register records what lipid therapies a patient is receiving within one indicator as follows:  0 = None, 1 = Statin only, 2 = Fibrate only, 3 = Other only, 4 = Multiple lipid therapies. However, the prevalence of the use of fibrates, other and multiple lipid therapies was very low therefore this was recoded into one binary variable:  '1' if the patient was in receipt of one or more lipid therapies, '0' if not.</p>

Quality of care		<p>This variable was constructed in stages. Firstly, new variables were coded on the basis of a recording of any value for each of the following indicators which should be recorded on an annual basis according to the NICE type 2 diabetes: BMI, HbA1c, BP, albumin, creatinine, cholesterol, smoking status and retinopathy. This was done prior to the omission of extreme values on the assumption that the practitioner who noted the recording would act on the primary data at the time of measurement rather the secondary data from the register. 0 = Not recorded, 1 = Recorded. Next, this was added into one overall score, which potentially ranged from 0 to 8. However, as only available cases were used, this ranged from 4 to 8 depending on which set of variables were included in the model. The overall score, therefore, was recoded into a categorical variable: 1 = less than 7, 2 = 7 and 3 = 8 care processes received. These are described in the results section as poor, medium or high quality of care.</p>
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Table 45: Provider data

Variable name	Source	Measurement/derivation/formatting
Practice deprivation	PHO	<p>The PHOs of England calculated the overall deprivation of general practice populations using the Index of Multiple Deprivation (IMD) 2007 applied proportionally to the Attribution Dataset practice populations, 2010 (see below for more information regarding IMD). Based on this score all practices in England were ranked with 1 representing the most deprived practice population. In order to identify non-linear trends these ranks were divided into quartiles with '1' indicating the most deprived 25% of practices. None of the practices in the South Tees area were in the 25% least deprived practice populations as such the three included categories were coded as: '1' high, '2' mid, and '3' low deprivation. These variables were assigned to each patient record based on the general practice they were registered with for that year.</p>

Practice list size	QOF	This continuous variable was extracted from the QOF dataset and is the number of patients on the clinical register for each general practice. To establish non-linear trends it was recoded '1' if than 7,000, '2' if there between 7,000 and 9,999 inclusively, and '3' if there were 10,000 or more patients registered with the practice. These variables were assigned to each patient record based on the general practice they were registered with for that year.
Diabetes prevalence		This was extracted from the QOF dataset and is the prevalence of diabetes patients aged 17 years old and over, calculated as follows: (Diabetes register/Practice List Size)*100. These variables were assigned to each patient record based on the general practice they were registered with for that year.
BMI recording Level		The percentage of patients with diabetes whose notes record BMI in the previous 15 months.
HbA1c ≤ 10%		The percentage of patients with diabetes in whom the last HbA1c is 10 or less (or equivalent test/reference range depending on local laboratory) in last 15 months.
Peripheral pulses Recording level		The percentage of patients with diabetes with a record of the presence or absence of peripheral pulses in the previous 15 months.
Neuropathy test recording level		The percentage of patients with diabetes with a record of neuropathy testing in the previous 15 months.
BP recording level		The percentage of patients with diabetes who have a record of the blood pressure in the previous 15 months.
BP ≤ 145/85 level		The percentage of patients with diabetes in whom the last blood pressure is 145/85 or less.
Microalbuminuria recording level		The percentage of patients with diabetes who have a record of micro-albuminuria testing in the previous 15 months (exception reporting for patients with proteinuria).
Proteinuria/microalbuminuria treated		The percentage of patients with diabetes with a diagnosis of proteinuria or micro-albuminuria who are treated with ACE inhibitors (or A2 antagonists).

with ACEI level		
Total cholesterol Recording level		The percentage of patients with diabetes who have a record of total cholesterol in the previous 15 months.
Total cholesterol $\leq$ 5 mmol/l level		The percentage of patients with diabetes whose last measured total cholesterol within the previous 15 months is 5mmol/l or less.
Influenza immunisation level		The percentage of patients with diabetes who have had influenza immunisation in the preceding 1 September to 31 March.

## Appendix D: Missing data per variable over time

The following tables indicate what percentage of each indicator is complete by year following the data cleaning, which is outlined in chapter 4. The colour is graded from red to yellow with the latter indicating the most complete fields.

**Table 46** shows that, overall, the completeness of population data for the study period is high. Completeness of the anthropometric and lifestyle data has steadily improved over the study but there was a decline in 2007. Similarly **Table 47** shows a steady increase in the recording of intermediate outcomes from 1999, however, this seems to peak around 2004 and then begin to drop off again. This may be because from this year onwards primary practitioners also have to input data onto the QOF system, which is separate from the practice systems and the paper proforma of the diabetes register. As the QOF system determines a significant part of GPs pay this work is likely to be prioritised above populating the diabetes register. The redness in this table also highlights the poor recording of patients lipid profiles with LDL cholesterol which was only introduced as a separate field in 2007. Due to this poor level of recording only total cholesterol out the lipid profile was used in the analyses. In addition, estimated cardiovascular risk was not examined either.

The recording of patients' history of vascular disease is relatively high with recording levels of over 80% for each indicator per year. The calculation for patients' eGFR level are based upon their creatinine levels. Both these indicators reflect the trend described above: a steady increase until 2004 with levels beginning to fall after this year. There are poor recording levels for proteinuria, microalbuminuria and retinopathy. These results are particularly worrying as the recording of patients' microalbuminuria and retinopathy are two of the nine key care processes recommended by NICE [46]. The retinopathy levels are falling far short of the target set as part of the NHS Diabetes Retinal Screening programme [48]. Estimated GFR, proteinuria and microalbuminuria are all indicators of kidney function, as eGFR was the most complete over the study variable was used as a covariate in the analyses.

The recording of diabetes treatments are very high with 90% complete for each indicator per year. Similarly with blood pressure and lipid treatments which have achieved the same high levels since 2002. The conspicuous lack of data is the recording of patients' lipid therapies in 1999 which achieved only about 30% completeness. Table 15 highlights the introduction of QOF over the study period and also when particular indicators were introduced and/or suspended.

Table 46: The percentage of population, anthropometric and lifestyle data complete by study year

	1999	2000	2001	2002	2003	2004	2005	2006	2007	Total
LSOA	100	100	100	100	100	100	100	100	100	100
Sex	100	100	100	100	100	100	100	100	100	100
Ethnicity	100	100	100	100	100	100	100	100	100	100
Age (years)	100	100	100	100	100	100	100	100	100	100
Age at diagnosis (years)	99.3	99.8	99.2	99.6	99.5	99.7	99.5	99.3	98.9	99.4
Duration of diabetes (years)	99.3	99.8	99.2	99.6	99.5	99.7	99.5	99.3	98.9	99.4
BMI (kg/m <sup>2</sup> )	64.1	66.3	68.7	72.9	77.7	83.2	84.4	83.9	76.4	76.6
Weight (kg)	75.3	75.1	76.9	79.1	80.4	84.7	87.2	86.3	76.5	80.8
Smoking Status	67.9	72.3	78.6	86.4	86.7	91.2	91.4	90.7	84.6	84.7

Table 47: The percentage of intermediate health outcomes data complete by study year

	1999	2000	2001	2002	2003	2004	2005	2006	2007	Total
sBP	80.9	83.1	85.2	86.2	90.0	93.1	93.8	93.9	90.0	89.3
dBP	76.6	78.1	77.0	78.4	89.9	93.0	93.9	93.9	89.9	87.0
Hypertension	76.6	78.0	77.0	78.4	89.9	93.0	93.8	93.9	89.9	86.9
HbA1c	73.3	78.4	82.9	92.9	93.6	94.6	90.9	90.5	87.0	88.1
Cholesterol	70.0	70.3	74.7	92.4	95.1	96.2	90.6	91.9	86.2	86.8
HDL	3.2	9.1	17.2	52.5	62.8	77.0	69.5	71.9	69.7	53.2
LDL	0.0	0.0	0.0	0.0	0.0	0.0	0.0	0.0	60.1	8.3
Triglycerides	7.0	12.1	25.7	53.8	62.6	75.8	67.3	71.0	70.0	54.2
Creatinine	69.5	73.4	77.5	92.6	94.3	96.2	90.9	91.2	88.2	87.4

Table 48: Percentage of long term complications complete by study year

	1999	2000	2001	2002	2003	2004	2005	2006	2007	Total
eGFR	68.6	71.5	74.8	88.9	89.8	90.9	85.6	85.4	82.5	83.1
Ischaemic cardiac Stroke or TIA)	82.9	83.8	87.0	85.6	89.9	89.9	88.7	90.3	85.5	87.4
PVD	82.3	82.2	85.9	84.5	87.4	87.5	87.2	88.9	84.2	85.9
Microalbuminuria	23.9	27.4	39.0	47.1	51.2	68.6	66.8	65.0	3.6	45.1
Proteinuria	54.2	57.8	60.2	64.3	61.9	68.7	72.1	67.8	44.7	61.7
Retinopathy	41.8	47.1	45.1	45.4	43.6	38.2	27.1	52.2	58.3	44.5

Table 49: Percentage of study population with HbA1c at diagnosis recorded in dataset

	1999	2000	2001	2002	2003	2004	2005	2006	2007	Total
HbA1c at Diagnosis	6.5	12.7	18.7	26.2	32.8	39.2	43.9	47.0	49.1	33.6

Table 50: Percentage of diabetes treatments complete by study year

	1999	2000	2001	2002	2003	2004	2005	2006	2007	Total
Diet alone	97.1	97.7	96.4	97.2	97.7	98.0	99.0	99.1	95.5	97.6
Insulin	97.1	97.7	96.4	97.2	97.1	98.0	99.0	99.1	94.6	97.4
Sulphonylurea	97.1	97.7	96.4	97.2	97.4	98.0	98.9	99.1	95.0	97.5
Metformin	97.1	97.7	96.4	97.2	97.5	98.0	98.9	99.1	95.2	97.5
Acarbose	97.1	97.7	96.4	97.2	97.1	98.0	98.9	99.1	94.6	97.4
Glitazone	97.1	97.7	96.4	97.2	97.1	98.0	98.9	99.1	94.7	97.4

Table 51: Percentage of blood pressure and lipid treatments complete by study year

	1999	2000	2001	2002	2003	2004	2005	2006	2007	Total
Diuretics	80.7	82.3	87.7	92.7	93.8	95.1	95.8	96.3	93.0	91.8
Beta Blockers	79.1	81.2	87.2	92.5	93.1	94.5	94.2	94.9	92.5	90.9
Alpha Blockers	76.5	78.4	85.2	92.1	92.0	93.8	93.5	94.0	91.6	89.7
ACE Inhibitor	80.2	82.6	88.1	92.9	94.4	95.9	95.3	95.8	93.4	91.9
AT2 Blockers	66.7	78.3	85.3	92.2	92.1	93.7	93.9	94.4	92.1	89.1
Calcium Antagonist	79.3	81.1	87.3	92.6	93.4	94.7	94.4	95.1	92.5	91.0
Aspirin	79.7	83.0	88.0	92.8	93.9	95.6	95.4	95.9	93.9	91.9
Lipid Therapy	28.8	76.6	85.6	92.0	94.3	95.9	95.8	96.8	94.7	87.5

Table 52: Percentage of practice level data complete by study year

	1999	2000	2001	2002	2003	2004	2005	2006	2007	Total
Practice deprivation score	100	100	100	100	100	100	100	100	100	100
Practice list size	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Diabetes register	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
DM QOF Prevalence	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Exception reporting level of DM indicators	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
BMI recording level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Smoking recording level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	0.0	0.0	22.5
HbA1c recording level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Patients achieving HbA1c ≤ 7.4%	0.0	0.0	0.0	0.0	0.0	88.5	88.7	0.0	0.0	22.5
Patients achieving HbA1c ≤ 10%	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Retinal screening level (1)	0.0	0.0	0.0	0.0	0.0	88.5	88.7	0.0	0.0	22.5
Peripheral pulses recording level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Neuropathy test recording level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
BP recording level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2

Patients achieving BP $\leq$ 145/85 (mmHg)	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Microalbuminuria recording level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Serum creatinine recording level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	0.0	0.0	22.5
Proteinuria/microalbuminuria treated with ACE inhibitors level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Total cholesterol recording level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Patients achieving total cholesterol $\leq$ 5mmol/l (%)	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Influenza immunisation level	0.0	0.0	0.0	0.0	0.0	88.5	88.7	100	100	50.2
Patients achieving HbA1c $\leq$ 7.5%	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100	100	27.7
Retinal screening level (2)	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100	100	27.7
eGFR or serum creatinine recording	0.0	0.0	0.0	0.0	0.0	0.0	0.0	100	100	27.7

## Appendix E: Analyses of missing data mechanisms

Table 53 shows that there were statistically significant association between deprivation level and the odds of missing data on the outcome variables. The least deprived were likely less to have missing data on the intermediate outcomes, eGFR and retinopathy. However, this association was more complex with the vascular disease variables. There were statistically significant association between missing data on the intermediate outcomes, eGFR and retinopathy with patients from white and south Asian backgrounds less likely to have missing data. The statistically significant odds ratio for age and gender shows that these have a small effect on likelihood of missing data.

Table 54 shows that the least deprived groups were less likely to have missing data on all the diabetes treatments, with statistical significance. Table 55 shows a statistically significant relationship with the least deprived quintile being about half as likely to have missing data on blood pressure treatment than patients from the most deprived quintile. The ethnicity, gender and age of patients do not predict missing data on diabetes and blood pressure treatments. Table four shows that male patients have statistically significant reduced odds of having missing data on aspirin treatment than women but no other demographic predict odds of having missing data on lipid treatments.

Table 53: Logistic regression analyses of missing data on outcome variables allowing for demographic variables

	Hba1c missing	Cholesterol missing	sBP missing	eGFR missing	Ischiac Cardiac missing	Stroke or TIA missing	PVD missing	Retinopathy missing
Ref. group: Most deprived								
Q2	0.83 (0.78, 0.88) 0.000	0.80 (0.76, 0.85) 0.000	0.82 (0.77, 0.87) 0.000	0.86 (0.81, 0.9) 0.000	1.28 (1.21, 1.35) 0.000	1.26 (1.19, 1.33) 0.000	1.25 (1.19, 1.32) 0.000	0.82 (0.79, 0.85) 0.000
Q3	0.83 (0.78, 0.89) 0.000	0.83 (0.78, 0.89) 0.000	0.81 (0.75, 0.87) 0.000	0.88 (0.83, 0.94) 0.000	0.93 (0.87, 1.00) 0.044	0.93 (0.87, 0.99) 0.032	0.93 (0.87, 0.99) 0.028	0.89 (0.85, 0.93) 0.000
Q4	0.84 (0.77, 0.91) 0.000	0.86 (0.80, 0.93) 0.000	0.85 (0.78, 0.92) 0.000	0.90 (0.84, 0.96) 0.001	1.3 (1.21, 1.40) 0.000	1.28 (1.20, 1.37) 0.000	1.25 (1.17, 1.34) 0.000	0.86 (0.82, 0.90) 0.000
Q5: Least deprived	0.54 (0.46, 0.65) 0.000	0.71 (0.62, 0.83) 0.000	0.53 (0.44, 0.64) 0.000	0.82 (0.72, 0.93) 0.002	0.46 (0.38, 0.56) 0.000	0.44 (0.37, 0.53) 0.000	0.46 (0.38, 0.55) 0.000	0.84 (0.77, 0.92) 0.000
White	0.54 (0.41, 0.71) 0.000	0.65 (0.49, 0.86) 0.002	0.56 (0.42, 0.74) 0.000	0.57 (0.45, 0.73) 0.000	1.07 (0.76, 1.52) 0.687	1.17 (0.82, 1.66) 0.386	1.11 (0.79, 1.55) 0.56	0.94 (0.75, 1.17) 0.586
South Asian	0.78 (0.58, 1.04) 0.086	0.95 (0.71, 1.26) 0.702	0.73 (0.54, 0.99) 0.042	0.75 (0.58, 0.97) 0.029	1.04 (0.72, 1.5) 0.825	1.09 (0.76, 1.58) 0.636	1.06 (0.74, 1.50) 0.759	1.31 (1.04, 1.65) 0.022
Afro Caribbean	0.8 (0.52, 1.24) 0.318	0.89 (0.58, 1.37) 0.603	1.01 (0.66, 1.56) 0.957	0.70 (0.47, 1.04) 0.076	1.11 (0.66, 1.86) 0.696	1.17 (0.7, 1.97) 0.541	1.19 (0.73, 1.94) 0.492	1.11 (0.79, 1.55) 0.561
Age	1.00 (1.00, 1.00) 0.251	1.00 (0.99, 1.00) 0.000	1.00 (1.00, 1.00) 0.312	0.99 (0.99, 0.99) 0.000	1.00 (1.00, 1.00) 0.002	1.00 (1.00, 1.01) 0.000	1.01 (1.00, 1.01) 0.000	1.00 (1.00, 1.00) 0.000
Male	0.94 (0.90, 0.99) 0.013	0.93 (0.89, 0.98) 0.002	0.91 (0.87, 0.96) 0.000	0.95 (0.91, 0.99) 0.014	0.96 (0.91, 1.00) 0.047	0.99 (0.94, 1.03) 0.538	1.00 (0.96, 1.04) 0.910	0.96 (0.93, 0.99) 0.006

Table 54: Logistic regression analyses of missing data on diabetes treatments allowing for demographic variables

	Diet alone missing	Insulin missing	Sulphonylurea missing	Metformin missing	Acarbose missing	Glitazone missing
Ref. group: Most deprived						
Q2	0.82 (0.73, 0.93) 0.002	0.87 (0.78, 0.98) 0.022	0.86 (0.76, 0.97) 0.013	0.83 (0.74, 0.94) 0.003	0.87 (0.77, 0.97) 0.015	0.86 (0.76, 0.97) 0.012
Q3	0.78 (0.67, 0.90) 0.001	0.78 (0.68, 0.90) 0.001	0.78 (0.67, 0.90) 0.001	0.79 (0.68, 0.92) 0.002	0.78 (0.67, 0.90) 0.001	0.78 (0.67, 0.90) 0.001
Q4	0.74 (0.62, 0.87) 0.000	0.84 (0.72, 0.98) 0.029	0.76 (0.64, 0.89) 0.001	0.77 (0.65, 0.91) 0.002	0.82 (0.70, 0.96) 0.014	0.81 (0.69, 0.95) 0.011
Q5: Least deprived	0.37 (0.24, 0.58) 0.000	0.39 (0.25, 0.59) 0.000	0.36 (0.23, 0.56) 0.000	0.38 (0.25, 0.59) 0.000	0.38 (0.25, 0.59) 0.000	0.39 (0.25, 0.59) 0.000
White	0.90 (0.45, 1.83) 0.78	0.96 (0.47, 1.94) 0.906	0.94 (0.46, 1.90) 0.865	0.92 (0.46, 1.87) 0.828	0.96 (0.48, 1.95) 0.920	0.96 (0.47, 1.93) 0.900
South Asian	0.92 (0.44, 1.92) 0.814	0.95 (0.45, 1.99) 0.893	0.94 (0.45, 1.97) 0.873	0.94 (0.45, 1.97) 0.870	0.96 (0.46, 2.02) 0.924	0.96 (0.46, 2.02) 0.923
Afro Caribbean	1.27 (0.47, 3.44) 0.637	1.60 (0.62, 4.11) 0.333	1.45 (0.55, 3.81) 0.455	1.27 (0.47, 3.43) 0.639	1.44 (0.55, 3.79) 0.463	1.43 (0.54, 3.77) 0.468
Age	1.02 (1.01, 1.02) 0.000	1.02 (1.01, 1.02) 0.000	1.02 (1.01, 1.02) 0.000	1.02 (1.01, 1.02) 0.000	1.02 (1.01, 1.02) 0.000	1.02 (1.01, 1.02) 0.000
Male	0.97 (0.88, 1.07) 0.520	0.97 (0.88, 1.07) 0.525	0.95 (0.86, 1.04) 0.278	0.97 (0.88, 1.06) 0.473	0.96 (0.87, 1.05) 0.380	0.97 (0.88, 1.06) 0.504

Table 55: Logistic regression analyses of missing data on blood pressure and lipid treatments allowing for demographic variables

	Diuretics missing	Beta blockers missing	Alpha blockers missing	ACE inhibitors missing	AT2 blockers missing	Calcium anatagonists missing	Aspirin missing	Lipid therapy missing
Ref. group: Most deprived								
Q2	0.96 (0.90, 1.03) 0.291	0.90 (0.84, 0.96) 0.002	0.92 (0.87, 0.98) 0.012	0.94 (0.88, 1.01) 0.086	0.91 (0.85, 0.96) 0.002	0.93 (0.87, 0.99) 0.030	0.97 (0.90, 1.03) 0.32	0.93 (0.88, 0.98) 0.011
Q3	0.98 (0.90, 1.06) 0.616	0.93 (0.86, 1.00) 0.062	0.96 (0.89, 1.03) 0.255	0.97 (0.90, 1.06) 0.53	0.95 (0.88, 1.02) 0.141	0.95 (0.88, 1.03) 0.219	0.98 (0.91, 1.07) 0.695	0.92 (0.86, 0.99) 0.017
Q4	1.08 (0.99, 1.18) 0.077	1.00 (0.92, 1.09) 0.938	1.05 (0.97, 1.13) 0.261	0.99 (0.90, 1.08) 0.825	1.01 (0.94, 1.10) 0.725	1.04 (0.96, 1.14) 0.344	1.03 (0.94, 1.12) 0.579	0.98 (0.91, 1.06) 0.667
Q5: Least deprived	0.52 (0.42, 0.64) 0.000	0.52 (0.43, 0.64) 0.000	0.49 (0.40, 0.60) 0.000	0.52 (0.42, 0.65) 0.000	0.55 (0.45, 0.66) 0.000	0.49 (0.39, 0.60) 0.000	0.55 (0.45, 0.68) 0.000	0.55 (0.46, 0.65) 0.000
White	0.82 (0.57, 1.17) 0.272	0.70 (0.50, 0.97) 0.032	0.84 (0.60, 1.17) 0.301	0.73 (0.51, 1.04) 0.083	0.95 (0.68, 1.35) 0.787	0.88 (0.61, 1.26) 0.488	0.74 (0.52, 1.05) 0.095	1.01 (0.72, 1.41) 0.963
South Asian	1.03 (0.71, 1.50) 0.872	0.82 (0.58, 1.16) 0.272	1.000 (0.70, 1.42) 0.993	0.92 (0.64, 1.33) 0.66	1.09 (0.76, 1.56) 0.638	1.06 (0.73, 1.55) 0.744	0.82 (0.57, 1.19) 0.293	1.14 (0.81, 1.61) 0.456
Afro Caribbean	1.24 (0.73, 2.10) 0.424	1.20 (0.74, 1.95) 0.454	1.22 (0.75, 1.99) 0.425	1.13 (0.67, 1.91) 0.636	1.55 (0.96, 2.51) 0.071	1.15 (0.68, 1.97) 0.598	1.17 (0.70, 1.96) 0.548	1.46 (0.91, 2.34) 0.112
Age	0.99 (0.99, 1.00) 0.000	1.00 (1.00, 1.00) 0.939	1.00 (1.00, 1.00) 0.339	1.00 (1.00, 1.00) 0.304	1.00 (1.00, 1.00) 0.835	1.00 (1.00, 1.00) 0.12	0.99 (0.99, 1.00) 0.000	1.00 (1.00, 1.00) 0.042
Male	1.07 (1.01, 1.13) 0.023	0.94 (0.89, 0.99) 0.015	0.95 (0.90, 1.00) 0.044	0.90 (0.86, 0.95) 0.000	0.99 (0.95, 1.04) 0.803	0.94 (0.9, 1.00) 0.034	0.88 (0.84, 0.93) 0.000	0.98 (0.94, 1.03) 0.408

## Appendix F. Stepwise models for intermediate outcomes and long-term complications with interaction between visit year and socio-economic status

### Intermediate health outcomes

Table 56: Stepwise linear regression multilevel models examining HbA1c by SES from 1999 to 2007, with interaction effect between SES and visit year and conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status &amp; visit year</b>				
Social-economic status, reference group: Low SES				
Medium SES		-0.04 (-0.21, 0.14)	-0.05 (-0.22, 0.12)	-0.09 (-0.26, 0.06)
High SES		0.02 (-0.14, 0.18)	0.08 (-0.08, 0.23)	0.04 (-0.10, 0.19)
Visit year, reference group: 1999				
2000		-0.17 (-0.31, -0.04)	-0.18 (-0.31, -0.05)	-0.24 (-0.37, -0.12)
2001		-0.45 (-0.59, -0.32)	-0.43 (-0.56, -0.30)	-0.47 (-0.59, -0.35)
2002		-0.52 (-0.64, -0.39)	-0.51 (-0.62, -0.39)	-0.49 (-0.60, -0.38)
2003		-0.55 (-0.67, -0.43)	-0.54 (-0.65, -0.42)	-0.53 (-0.64, -0.43)
2004		-0.62 (-0.74, -0.50)	-0.62 (-0.74, -0.51)	-0.61 (-0.71, -0.50)
2005		-0.69 (-0.81, -0.58)	-0.70 (-0.81, -0.59)	-0.68 (-0.79, -0.58)
2006		-1.22 (-1.34, -1.10)	-1.21 (-1.33, -1.10)	-1.16 (-1.27, -1.06)
2007		-1.08 (-1.19, -0.96)	-1.09 (-1.21, -0.98)	-1.11 (-1.22, -1.00)
SES x Visit year, reference group: Low SES x 1999				
Medium SES x 2000		-0.13 (-0.37, 0.11)	-0.08 (-0.31, 0.16)	0.01 (-0.20, 0.23)
Medium SES x 2001		-0.01 (-0.24, 0.22)	0.05 (-0.18, 0.27)	0.14 (-0.07, 0.35)
Medium SES x 2002		-0.17 (-0.38, 0.05)	-0.11 (-0.31, 0.10)	-0.04 (-0.23, 0.15)
Medium SES x 2003		-0.15 (-0.35, 0.07)	-0.08 (-0.28, 0.12)	0.01 (-0.18, 0.20)
Medium SES x 2004		-0.10 (-0.31, 0.10)	-0.02 (-0.22, 0.18)	0.03 (-0.15, 0.22)
Medium SES x 2005		-0.09 (-0.29, 0.11)	-0.01 (-0.21, 0.19)	0.05 (-0.13, 0.24)
Medium SES x 2006		-0.03 (-0.23, 0.17)	0.04 (-0.16, 0.23)	0.08 (-0.11, 0.26)
Medium SES x 2007		-0.02 (-0.22, 0.19)	0.06 (-0.15, 0.26)	0.11 (-0.07, 0.30)
High SES x 2000		-0.32 (-0.54, -0.10)	-0.31 (-0.52, -0.10)	-0.26 (-0.46, -0.06)
High SES x 2001		-0.15 (-0.36, 0.07)	-0.17 (-0.37, 0.04)	-0.09 (-0.28, 0.10)
High SES x 2002		-0.21 (-0.40, -0.01)	-0.18 (-0.37, 0.02)	-0.16 (-0.34, 0.02)
High SES x 2003		-0.25 (-0.44, -	-0.24 (-0.43, -	-0.20 (-0.37, -

High SES x 2004		0.06 -0.16 (-0.34, 0.02)	0.06 -0.12 (-0.30, 0.06)	0.02 -0.11 (-0.28, 0.06)
High SES x 2005		-0.23 (-0.41, -0.04)	-0.18 (-0.36, 0.00)	-0.14 (-0.31, 0.03)
High SES x 2006		-0.19 (-0.37, 0.00)	-0.16 (-0.34, 0.01)	-0.11 (-0.28, 0.05)
High SES x 2007		-0.24 (-0.42, -0.06)	-0.19 (-0.37, -0.01)	-0.13 (-0.30, 0.04)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			-0.46 (-0.50, -0.42)	-0.33 (-0.36, -0.29)
Age: 75+ years			-0.69 (-0.73, -0.64)	-0.41 (-0.46, -0.37)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.38 (0.35, 0.41)	0.06 (0.02, 0.09)
Duration 10+ years			0.70 (0.66, 0.74)	0.05 (0.01, 0.10)
Ethnicity, reference group: White				
South Asian			0.47 (0.39, 0.55)	0.46 (0.39, 0.54)
Other Ethnicity			0.66 (0.48, 0.83)	0.47 (0.31, 0.63)
Male			-0.12 (-0.15, -0.09)	-0.06 (-0.09, -0.03)
Smoking status, reference group: Non smoker				
Smoker			0.25 (0.21, 0.30)	0.23 (0.19, 0.27)
Ex-smoker			0.07 (0.04, 0.11)	0.05 (0.02, 0.08)
BMI status, reference group: Low & normal weight				
Overweight			0.06 (0.01, 0.10)	0.02 (-0.02, 0.07)
Obese			0.19 (0.15, 0.24)	0.08 (0.04, 0.13)
Creatinine > 300			-0.85 (-1.10, -0.59)	-0.81 (-1.06, -0.56)
Hypertensive			0.14 (0.11, 0.17)	0.10 (0.07, 0.13)
Ischaemic Cardiac Stroke or TIA			0.05 (0.01, 0.08)	0.00 (-0.04, 0.03)
			-0.01 (-0.06, 0.04)	-0.06 (-0.10, -0.01)
PVD			0.09 (0.04, 0.15)	-0.06 (-0.12, -0.01)
<b>Interventions</b>				
Quality of Care level, reference group: Low quality				
Medium quality				-0.13 (-0.16, -0.09)
High quality				-0.15 (-0.19, -0.11)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				0.81 (0.77, 0.85)
Combination with no insulin				1.25 (1.20, 1.29)
Insulin only				1.67 (1.61, 1.73)
Combination with insulin				1.75 (1.69, 1.82)
Shared care				0.17 (0.13, 0.21)
Middlesbrough PCT				0.10 (0.00, 0.20)
<b>Cons</b>	7.62 (7.49, 7.74)	8.50 (8.37, 8.62)	8.25 (8.13, 8.38)	7.56 (7.42, 7.70)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.05 (0.03, 0.08)	0.04 (0.03, 0.07)	0.03 (0.02, 0.05)	0.02 (0.01, 0.04)
<b>Patient level</b>	0.02 (0.01, 0.06)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.00 (0.00, 0.02)
<b>Visit year</b>	2.47 (2.43, 2.50)	2.36 (2.32, 2.39)	2.20 (2.17, 2.23)	1.91 (1.88, 1.94)
<b>Bayesian DIC</b>	145158.08	143556.00	140856.59	133988.98

N = 38,413

Table 57: Stepwise linear regression multilevel models examining cholesterol by SES from 1999 to 2007, with interaction effect between SES and visit year and conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status &amp; visit year</b>				
Social-economic status, reference group: Lowest SES				
Medium SES		0.05 (-0.15, 0.25)	0.07 (-0.13, 0.27)	0.06 (-0.13, 0.26)
High SES		-0.24 (-0.42, -0.06)	-0.21 (-0.38, -0.03)	-0.22 (-0.40, -0.05)
Visit year, reference group: 1999				
2000		-0.27 (-0.41, -0.14)	-0.25 (-0.38, -0.12)	-0.25 (-0.38, -0.12)
2001		-0.32 (-0.45, -0.18)	-0.29 (-0.42, -0.16)	-0.28 (-0.41, -0.16)
2002		-0.35 (-0.48, -0.22)	-0.31 (-0.43, -0.19)	-0.29 (-0.41, -0.16)
2003		-0.55 (-0.68, -0.43)	-0.50 (-0.62, -0.38)	-0.43 (-0.55, -0.31)
2004		-0.80 (-0.93, -0.68)	-0.75 (-0.87, -0.63)	-0.64 (-0.76, -0.52)
2005		-0.98 (-1.10, -0.85)	-0.91 (-1.03, -0.79)	-0.79 (-0.91, -0.67)
2006		-1.14 (-1.27, -1.02)	-1.06 (-1.18, -0.95)	-0.92 (-1.04, -0.80)
2007		-1.18 (-1.31, -1.06)	-1.1 (-1.22, -0.98)	-0.99 (-1.11, -0.87)
SES x Visit year, reference group: Low SES x 1999				
Medium SES x 2000		0.01 (-0.23, 0.25)	-0.01 (-0.25, 0.22)	-0.01 (-0.24, 0.22)
Medium SES x 2001		-0.01 (-0.24, 0.23)	-0.02 (-0.25, 0.20)	-0.02 (-0.24, 0.20)
Medium SES x 2002		-0.03 (-0.26, 0.19)	-0.03 (-0.25, 0.19)	-0.02 (-0.23, 0.20)
Medium SES x 2003		-0.04 (-0.26, 0.18)	-0.04 (-0.26, 0.18)	-0.04 (-0.25, 0.17)
Medium SES x 2004		-0.07 (-0.29, 0.14)	-0.07 (-0.29, 0.14)	-0.06 (-0.27, 0.15)
Medium SES x 2005		-0.09 (-0.3, 0.13)	-0.09 (-0.30, 0.12)	-0.08 (-0.29, 0.13)
Medium SES x 2006		0.02 (-0.20, 0.23)	0.01 (-0.20, 0.22)	0.02 (-0.18, 0.23)
Medium SES x 2007		-0.05 (-0.26, 0.17)	-0.04 (-0.26, 0.17)	-0.03 (-0.24, 0.18)
High SES x 2000		0.17 (-0.04, 0.39)	0.16 (-0.05, 0.36)	0.16 (-0.05, 0.37)
High SES x 2001		0.27 (0.07, 0.49)	0.26 (0.05, 0.46)	0.26 (0.06, 0.46)
High SES x 2002		0.20 (0.00, 0.40)	0.20 (0.00, 0.39)	0.22 (0.02, 0.41)
High SES x 2003		0.19 (0.00, 0.39)	0.19 (0.00, 0.38)	0.20 (0.01, 0.39)
High SES x 2004		0.20 (0.00, 0.39)	0.20 (0.01, 0.38)	0.22 (0.04, 0.41)
High SES x 2005		0.20 (0.02, 0.40)	0.20 (0.02, 0.39)	0.22 (0.04, 0.41)
High SES x 2006		0.24 (0.05, 0.43)	0.24 (0.05, 0.43)	0.27 (0.08, 0.45)
High SES x 2007		0.19 (0.00, 0.39)	0.19 (0.01, 0.38)	0.21 (0.02, 0.39)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			-0.21 (-0.23, -0.18)	-0.20 (-0.23, -0.17)
Age: 75+ years			-0.23 (-0.26, -0.20)	-0.26 (-0.30, -0.23)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.11 (-0.13, -0.08)	-0.09 (-0.11, -0.06)
Duration 10+ years			-0.16 (-0.19, -0.13)	-0.13 (-0.16, -0.10)
Ethnicity, reference group: White				
South Asian			-0.06 (-0.12, -0.00)	-0.08 (-0.14, -0.02)

Other Ethnicity			0.01 0.11 (-0.02, 0.24)	0.02 0.08 (-0.05, 0.20)
Male			-0.33 (-0.35, - 0.31)	-0.34 (-0.36, - 0.32)
Smoking status, reference group: Non smoker				
Smoker			0.08 (0.05, 0.11)	0.08 (0.05, 0.11)
Ex-smoker			-0.02 (-0.04, - 0.01)	0.00 (-0.03, 0.02)
BMI, reference group: Under or normal weight				
Overweight			0.00 (-0.03, 0.04)	0.03 (0.00, 0.07)
Obese			0.00 (-0.04, 0.03)	0.03 (0.00, 0.07)
Hypertensive			0.14 (0.12, 0.16)	0.14 (0.12, 0.16)
Ischaemic Cardiac			-0.21 (-0.24, - 0.19)	-0.13 (-0.15, - 0.10)
Stroke or TIA			-0.05 (-0.09, - 0.02)	-0.02 (-0.06, 0.01)
PVD			-0.02 (-0.06, 0.03)	0.01 (-0.03, 0.05)
<b>Interventions</b>				
Quality of Care level, reference group: Low quality				
Medium quality				-0.10 (-0.13, - 0.07)
High quality				-0.15 (-0.18, - 0.11)
Aspirin				-0.09 (-0.11, - 0.06)
Lipid therapy				-0.28 (-0.31, - 0.26)
M. PCT				-0.03 (-0.09, 0.03)
Shared care				-0.07 (-0.10, - 0.04)
<b>Cons</b>	4.65 (4.60, 4.70)	5.40 (5.26, 5.54)	5.79 (5.66, 5.92)	5.98 (5.85, 6.12)
<b>Variance estimates (Standard Error):</b>				
<b>Practice level</b>	0.01 (0.01, 0.02)	0.01 (0.01, 0.02)	0.01 (0.01, 0.02)	0.01 (0.01, 0.01)
<b>Patient level</b>	0.00 (0.00, 0.01)	0.01 (0.00, 0.03)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)
<b>Visit year</b>	1.35 (1.33, 1.37)	1.24 (1.22, 1.25)	1.17 (1.15, 1.18)	1.14 (1.13, 1.16)
<b>Bayesian DIC</b>	116373.88	113194.24	111042.23	110350.13
N = 37,085				

## Long-term complications

Table 58: Stepwise logistic regression multilevel models examining incidences of ICD by SES 2000 to 2007, with interaction effect between SES and visit year and conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status &amp; visit year</b>				
Social-economic status, reference group: Low				
Medium		-0.55 (-1.05, - 0.07)	-0.66 (-1.17, - 0.17)	-0.73 (-1.31, - 0.15)
High		-0.40 (-0.85, 0.03)	-0.36 (-0.85, 0.11)	-0.30 (-0.83, 0.23)
Visit year, reference group: 2000				
2001		-0.28 (-0.65, 0.08)	-0.42 (-0.80, - 0.04)	-0.55 (-0.97, - 0.10)

2002		-0.13 (-0.45, 0.18)	-0.23 (-0.55, 0.11)	-0.36 (-0.73, 0.03)
2003		-0.18 (-0.49, 0.11)	-0.32 (-0.63, 0.00)	-0.68 (-1.04, -0.30)
2004		-0.22 (-0.52, 0.07)	-0.43 (-0.75, -0.11)	-0.84 (-1.20, -0.45)
2005		-0.60 (-0.92, -0.30)	-0.89 (-1.22, -0.55)	-1.34 (-1.72, -0.94)
2006		-1.15 (-1.48, -0.82)	-1.47 (-1.82, -1.12)	-1.89 (-2.27, -1.46)
2007		-1.01 (-1.35, -0.67)	-1.34 (-1.70, -0.98)	-1.95 (-2.35, -1.52)
SES x Visit year, reference group: Low SES x 2000				
Medium SES x 2001		0.45 (-0.20, 1.10)	0.53 (-0.15, 1.20)	0.54 (-0.26, 1.31)
Medium SES x 2002		0.37 (-0.21, 0.95)	0.43 (-0.17, 1.05)	0.41 (-0.28, 1.09)
Medium SES x 2003		0.72 (0.16, 1.30)	0.80 (0.21, 1.38)	0.91 (0.25, 1.58)
Medium SES x 2004		0.56 (0.00, 1.14)	0.59 (0.01, 1.17)	0.60 (-0.09, 1.24)
Medium SES x 2005		0.36 (-0.22, 0.96)	0.40 (-0.23, 1.00)	0.41 (-0.28, 1.08)
Medium SES x 2006		0.53 (-0.09, 1.15)	0.63 (0.00, 1.26)	0.69 (-0.02, 1.37)
Medium SES x 2007		0.37 (-0.25, 0.99)	0.40 (-0.24, 1.05)	0.37 (-0.37, 1.08)
High SES x 2001		0.37 (-0.24, 0.99)	0.34 (-0.30, 0.98)	0.49 (-0.23, 1.21)
High SES x 2002		0.18 (-0.37, 0.73)	0.04 (-0.54, 0.63)	-0.11 (-0.76, 0.53)
High SES x 2003		0.26 (-0.25, 0.79)	0.20 (-0.34, 0.75)	0.30 (-0.32, 0.92)
High SES x 2004		0.33 (-0.19, 0.85)	0.21 (-0.33, 0.78)	0.13 (-0.48, 0.73)
High SES x 2005		0.17 (-0.37, 0.72)	0.08 (-0.50, 0.69)	0.02 (-0.60, 0.63)
High SES x 2006		0.40 (-0.15, 0.97)	0.33 (-0.25, 0.94)	0.20 (-0.45, 0.84)
High SES x 2007		0.30 (-0.26, 0.87)	0.22 (-0.37, 0.82)	0.18 (-0.47, 0.83)

#### Socio-demographic, anthropometric, lifestyle and health covariates

Age, reference group: <60 years				
Age: 60-74 years			0.65 (0.51, 0.79)	0.33 (0.18, 0.48)
Age: 75+ years			0.83 (0.66, 1.01)	0.60 (0.41, 0.78)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.63 (-0.75, -0.50)	-0.59 (-0.74, -0.45)
Duration 10+ years			-0.56 (-0.70, -0.42)	-0.63 (-0.80, -0.47)
Ethnicity, reference group: White				
South Asian			-0.11 (-0.42, 0.18)	0.08 (-0.25, 0.40)
Other Ethnicity			-0.63 (-1.44, 0.08)	-0.68 (-1.55, 0.09)
Male			0.36 (0.24, 0.47)	0.33 (0.21, 0.45)
Smoking status, reference group: non smoker				
Smoker			0.16 (0.00, 0.32)	0.15 (-0.02, 0.32)
Ex-smoker			0.34 (0.22, 0.46)	0.31 (0.18, 0.44)
Obesity category, reference group: under & normal weight				
Overweight			0.10 (-0.06, 0.27)	-0.02 (-0.20, 0.15)
Obese			0.35 (0.19, 0.51)	0.12 (-0.06, 0.29)
HbA1c			0.06 (0.02, 0.09)	0.07 (0.03, 0.11)
Hypertensive			-0.19 (-0.30, -0.08)	-0.28 (-0.40, -0.16)
Cholesterol			-0.33 (-0.38, -0.27)	-0.23 (-0.29, -0.17)
eGFR			-0.02 (-0.02, -0.01)	-0.01 (-0.01, 0.00)

#### Interventions

Quality of care level, reference group: Low quality				
Medium quality				-0.31 (-0.44, -0.17)
High quality				-0.40 (-0.57, -0.24)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				-0.28 (-0.42, -

Combination, no insulin				0.13 -0.40 (-0.60, -0.20)
Insulin only				0.12 (-0.12, 0.37)
Combination with insulin				-0.29 (-0.58, -0.01)
Blood pressure treatment, reference group: No treatments				
ACE inhibitors only				0.26 (0.00, 0.52)
ACEI + other(s)				1.50 (1.31, 1.70)
Combination/other				1.25 (1.05, 1.44)
Aspirin				1.38 (1.26, 1.50)
Lipid therapy				0.61 (0.48, 0.74)
M. PCT				-0.20 (-0.39, -0.01)
Shared care				0.27 (0.11, 0.43)
<b>Cons</b>	-3.98 (-5.22, -2.58)	-3.60 (-4.76, -2.58)	-1.89 (-3.91, -0.24)	-3.50 (-4.69, -2.18)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.04 (0.02, 0.09)	0.04 (0.01, 0.08)	0.03 (0.01, 0.07)	0.05 (0.02, 0.11)
<b>Patient level</b>	2.44 (0.59, 8.18)	2.48 (0.65, 7.79)	2.70 (0.61, 9.53)	2.15 (0.53, 7.50)
<b>Bayesian DIC</b>	11620.93	11425.04	10735.28	9208.63

N = 24,004

Table 59: Stepwise logistic regression multilevel models examining incidences of stroke or TIA by SES 2000 to 2007 with interaction effect between SES and visit year, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status &amp; visit year</b>				
Social-economic status, reference group: Low SES				
Medium		0.05 (-0.67, 0.72)	0.06 (-0.68, 0.78)	0.20 (-0.50, 0.93)
High		-0.10 (-0.82, 0.61)	-0.12 (-0.87, 0.58)	0.05 (-0.69, 0.80)
Visit year, reference group: 2000				
2001		0.21 (-0.34, 0.76)	0.16 (-0.40, 0.71)	0.32 (-0.25, 0.92)
2002		0.11 (-0.38, 0.61)	0.06 (-0.45, 0.58)	0.21 (-0.32, 0.78)
2003		0.25 (-0.22, 0.75)	0.22 (-0.27, 0.71)	0.31 (-0.19, 0.85)
2004		0.00 (-0.46, 0.49)	-0.05 (-0.53, 0.44)	0.09 (-0.41, 0.64)
2005		-0.31 (-0.80, 0.19)	-0.35 (-0.86, 0.15)	-0.20 (-0.71, 0.39)
2006		-0.98 (-1.52, -0.42)	-1.04 (-1.59, -0.49)	-0.87 (-1.45, -0.24)
2007		-0.62 (-1.13, -0.09)	-0.67 (-1.21, -0.14)	-0.53 (-1.11, 0.09)
SES x Visit year, reference group: Low SES x 2000				
Medium SES x 2001		-0.17 (-1.07, 0.75)	-0.18 (-1.12, 0.77)	-0.25 (-1.20, 0.67)
Medium SES x 2002		-0.04 (-0.85, 0.82)	-0.09 (-0.96, 0.79)	-0.24 (-1.12, 0.62)
Medium SES x 2003		0.02 (-0.76, 0.86)	0.00 (-0.84, 0.85)	-0.05 (-0.89, 0.77)
Medium SES x 2004		-0.21 (-1.03, 0.64)	-0.27 (-1.12, 0.59)	-0.41 (-1.25, 0.41)
Medium SES x 2005		0.07 (-0.74, 0.92)	-0.03 (-0.88, 0.86)	-0.20 (-1.07, 0.65)
Medium SES x 2006		0.39 (-0.47, 1.29)	0.35 (-0.53, 1.27)	0.16 (-0.75, 1.04)
Medium SES x 2007		-0.37 (-1.28, 0.56)	-0.46 (-1.42, 0.49)	-0.70 (-1.67, 0.26)
High SES x 2001		-0.11 (-1.05, 0.83)	-0.08 (-1.03, 0.89)	-0.13 (-1.09, 0.82)
High SES x 2002		-0.25 (-1.15, 0.64)	-0.27 (-1.16, 0.67)	-0.40 (-1.32, 0.50)
High SES x 2003		-0.47 (-1.36, 0.37)	-0.49 (-1.33, 0.39)	-0.56 (-1.44, 0.29)
High SES x 2004		0.32 (-0.48, 1.12)	0.29 (-0.50, 1.13)	0.16 (-0.67, 0.98)
High SES x 2005		0.39 (-0.42, 1.23)	0.32 (-0.50, 1.19)	0.17 (-0.71, 1.02)
High SES x 2006		0.30 (-0.59, 1.22)	0.28 (-0.62, 1.21)	0.06 (-0.88, 0.99)
High SES x 2007		0.12 (-0.75, 0.99)	0.09 (-0.77, 1.00)	-0.07 (-1.00, 0.83)
Age, reference group: <60 years				
Age: 60-74 years			0.86 (0.63, 1.09)	0.74 (0.50, 0.99)
Age: 75+ years			1.21 (0.95, 1.48)	1.10 (0.82, 1.38)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.31 (-0.50, -0.13)	-0.31 (-0.49, -0.12)
Duration 10+ years			-0.04 (-0.22, 0.15)	-0.18 (-0.39, 0.03)
Ethnicity, reference group: White				
South Asian			0.04 (-0.39, 0.43)	0.11 (-0.34, 0.53)
Other Ethnicity			-1.20 (-3.04, 0.13)	-1.17 (-3.07, 0.17)
Male			0.01 (-0.15, 0.17)	-0.11 (-0.28, 0.06)
Smoking status, reference group: non smoker				
Smoker			0.31 (0.08, 0.53)	0.28 (0.05, 0.51)
Ex-smoker			0.21 (0.04, 0.38)	0.14 (-0.04, 0.31)

Obesity category, reference group: under & normal weight				
Overweight			-0.06 (-0.27, 0.15)	-0.08 (-0.29, 0.13)
Obese			-0.09 (-0.30, 0.11)	-0.19 (-0.41, 0.03)
HbA1c			0.02 (-0.03, 0.07)	0.02 (-0.03, 0.07)
Hypertensive			0.15 (0.00, 0.31)	0.14 (-0.01, 0.30)
Cholesterol			-0.11 (-0.19, -0.04)	-0.09 (-0.17, -0.02)
eGFR			-0.01 (-0.02, -0.01)	-0.01 (-0.01, 0.00)
<b>Interventions</b>				
Quality of care level, reference group: Low quality				
Medium quality				-0.17 (-0.36, 0.03)
High quality				-0.03 (-0.24, 0.19)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				-0.18 (-0.38, 0.02)
Combo., no insulin				-0.30 (-0.56, -0.04)
Insulin only				-0.08 (-0.39, 0.23)
Combo., with insulin				-0.29 (-0.68, 0.08)
Blood pressure treatment, reference group: No treatments				
ACE inhibitors only				0.31 (0.03, 0.61)
ACE + other(s)				0.16 (-0.08, 0.40)
Combination/other				0.18 (-0.05, 0.43)
Aspirin				1.06 (0.89, 1.23)
Lipid therapy				0.08 (-0.09, 0.26)
M. PCT				-0.12 (-0.39, 0.14)
Shared care				0.45 (0.24, 0.65)
<b>Cons</b>	-4.63 (-5.38, -3.86)	-4.56 (-5.92, -3.61)	-4.2 (-5.32, -3.17)	-5.14 (-6.50, -4.01)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.10 (0.04, 0.21)	0.08 (0.03, 0.18)	0.07 (0.01, 0.16)	0.11 (0.04, 0.22)
<b>Patient level</b>	1.23 (0.31, 4.03)	1.45 (0.34, 5.10)	1.33 (0.33, 4.41)	1.36 (0.33, 4.57)
<b>Bayesian DIC</b>	6705.64	6651.42	6444.78	6143.66
N = 29,800				

Table 60: Stepwise logistic regression multilevel models examining incidences of PVD by SES 2000 to 2007 with interaction effect between SES and visit year, conditional on relevant explanatory variables

	<b>Null</b>	<b>Model 1</b>	<b>Model 2</b>	<b>Model 3</b>
<b>Social-economic status &amp; visit year</b>				
Social-economic status, reference group: Low				
Medium		-0.35 (-1.06, 0.33)	-0.33 (-1.08, 0.39)	-0.19 (-0.98, 0.53)
High		-0.26 (-0.95, 0.39)	-0.18 (-0.85, 0.50)	-0.03 (-0.77, 0.65)
Visit year, reference group: 2000				
2001		-0.56 (-1.15, 0.02)	-0.54 (-1.16, 0.09)	-0.38 (-1.00, 0.23)
2002		-0.34 (-0.84, 0.15)	-0.34 (-0.85, 0.18)	-0.17 (-0.71, 0.37)
2003		-0.15 (-0.61, 0.31)	-0.09 (-0.55, 0.37)	0.11 (-0.39, 0.63)

2004		0.31) -0.20 (-0.64, 0.27)	0.39) -0.12 (-0.57, 0.37)	0.16 (-0.34, 0.67)
2005		-0.80 (-1.29, -0.31)	-0.74 (-1.24, -0.22)	-0.40 (-0.94, 0.14)
2006		-1.01 (-1.51, -0.52)	-0.88 (-1.39, -0.38)	-0.62 (-1.18, -0.06)
2007		-1.55 (-2.15, -0.97)	-1.47 (-2.08, -0.87)	-1.09 (-1.76, -0.45)
SES x Visit year, reference group: Low SES x 2000				
Medium SES x 2001		0.65 (-0.31, 1.64)	0.63 (-0.38, 1.63)	0.47 (-0.52, 1.49)
Medium SES x 2002		0.47 (-0.37, 1.35)	0.49 (-0.40, 1.40)	0.39 (-0.49, 1.32)
Medium SES x 2003		-0.07 (-0.91, 0.77)	-0.06 (-0.97, 0.86)	-0.12 (-1.04, 0.80)
Medium SES x 2004		0.03 (-0.78, 0.86)	0.00 (-0.86, 0.89)	-0.18 (-1.04, 0.73)
Medium SES x 2005		0.28 (-0.60, 1.15)	0.24 (-0.68, 1.16)	-0.05 (-0.99, 0.93)
Medium SES x 2006		0.00 (-0.94, 0.91)	-0.06 (-1.03, 0.90)	-0.23 (-1.21, 0.75)
Medium SES x 2007		0.24 (-0.83, 1.27)	0.15 (-0.93, 1.26)	-0.02 (-1.10, 1.10)
High SES x 2001		0.30 (-0.68, 1.26)	0.20 (-0.80, 1.17)	0.13 (-0.87, 1.13)
High SES x 2002		-0.62 (-1.57, 0.35)	-0.63 (-1.62, 0.30)	-0.81 (-1.81, 0.19)
High SES x 2003		0.06 (-0.72, 0.87)	-0.05 (-0.86, 0.75)	-0.17 (-0.98, 0.68)
High SES x 2004		-0.08 (-0.86, 0.73)	-0.20 (-1.01, 0.61)	-0.42 (-1.25, 0.44)
High SES x 2005		0.61 (-0.18, 1.45)	0.57 (-0.25, 1.38)	0.35 (-0.48, 1.22)
High SES x 2006		-0.22 (-1.13, 0.70)	-0.33 (-1.26, 0.58)	-0.46 (-1.40, 0.49)
High SES x 2007		0.04 (-0.98, 1.05)	-0.02 (-1.06, 0.98)	-0.14 (-1.21, 0.91)

#### Socio-demographic, anthropometric, lifestyle and health covariates

Age, reference group: <60 years				
Age: 60-74 years			0.67 (0.42, 0.91)	0.63 (0.38, 0.89)
Age: 75+ years			0.72 (0.42, 1.01)	0.83 (0.52, 1.14)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.25 (0.04, 0.45)	0.13 (-0.10, 0.35)
Duration 10+ years			0.74 (0.53, 0.94)	0.38 (0.14, 0.62)
Ethnicity, reference group: White				
South Asian			-0.88 (-1.60, -0.25)	-0.79 (-1.52, -0.16)
Other Ethnicity			0.02 (-1.02, 0.89)	-0.04 (-1.10, 0.84)
Male			0.40 (0.22, 0.58)	0.39 (0.20, 0.58)
Smoking status, reference group: non smoker				
Smoker			0.90 (0.66, 1.14)	0.94 (0.68, 1.19)
Ex-smoker			0.42 (0.22, 0.63)	0.38 (0.17, 0.59)
Obesity category, reference group: under & normal weight				
Overweight			-0.13 (-0.36, 0.11)	-0.20 (-0.46, 0.05)
Obese			-0.06 (-0.29, 0.17)	-0.25 (-0.50, 0.00)
HbA1c			0.07 (0.01, 0.12)	0.01 (-0.05, 0.07)
Hypertensive			0.17 (0.00, 0.35)	0.13 (-0.05, 0.31)
Cholesterol			-0.13 (-0.21, -0.05)	-0.05 (-0.13, 0.03)
eGFR			-0.01 (-0.02, -0.01)	-0.01 (-0.01, 0.00)

#### Interventions

Quality of care level, reference group: Low quality				
Medium quality				-0.18 (-0.42, 0.06)

High quality Diabetes treatment, reference group diet alone Metformin/sulphonylureas only				0.19 (-0.06, 0.43)
Combo., no insulin				-0.05 (-0.31, 0.21)
Insulin only Combo., with insulin				-0.12 (-0.43, 0.20)
Blood pressure treatment, reference group: No treatments				0.33 (0.00, 0.67)
ACE inhibitors only				0.33 (-0.05, 0.71)
Combination, with ACEI				0.50 (0.17, 0.83)
Combination, no ACEI				0.43 (0.15, 0.72)
Aspirin				0.38 (0.10, 0.67)
Lipid therapy				0.57 (0.40, 0.76)
M. PCT				0.10 (-0.09, 0.30)
Shared care				-0.17 (-0.54, 0.21)
<b>Cons</b>	-4.80 (-5.55, -4.12)	-4.17 (-4.99, -3.32)	-4.68 (-5.89, -3.36)	0.85 (0.63, 1.07)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.26 (0.13, 0.45)	0.26 (0.14, 0.47)	0.26 (0.14, 0.47)	0.28 (0.14, 0.48)
<b>Patient level</b>	0.96 (0.26, 2.98)	0.99 (0.27, 3.08)	1.14 (0.31, 3.52)	1.43 (0.34, 4.92)
<b>Bayesian DIC</b>	5910.02	5576.48	5325.55	5033.64
N = 30,053				

Table 61: Stepwise logistic regression multilevel models examining incidences of microalbuminuria by SES 2000 to 2007 with interaction effect between SES and visit year, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status &amp; visit year</b>				
Social-economic status, reference group: Lowest SES				
Medium SES		0.38 (-0.31, 1.04)	0.33 (-0.32, 1.11)	0.52 (-0.20, 1.29)
High SES		0.35 (-0.25, 0.86)	0.22 (-0.42, 0.78)	0.36 (-0.27, 1.07)
Visit year, reference group: 1999				
2000		-0.02 (-0.43, 0.41)	-0.08 (-0.57, 0.39)	-0.11 (-0.60, 0.41)
2001		-0.16 (-0.56, 0.26)	-0.21 (-0.68, 0.24)	-0.35 (-0.83, 0.16)
2002		-0.37 (-0.75, 0.04)	-0.43 (-0.89, 0.01)	-0.75 (-1.20, -0.25)
2003		0.05 (-0.30, 0.44)	0.00 (-0.44, 0.42)	-0.44 (-0.89, 0.05)
2004		0.78 (0.43, 1.15)	0.75 (0.31, 1.17)	0.30 (-0.14, 0.78)
2005		1.05 (0.71, 1.42)	1.03 (0.59, 1.44)	0.51 (0.07, 1.00)
2006		1.36 (1.02, 1.75)	1.38 (0.94, 1.80)	0.90 (0.46, 1.38)
2007		0.91 (0.26, 1.55)	0.90 (0.22, 1.57)	0.56 (-0.15, 1.27)
SES x Visit year, reference group: Low SES x 1999				
Medium SES x 2000		-0.35 (-1.14, 0.47)	-0.32 (-1.20, 0.46)	-0.49 (-1.38, 0.36)
Medium SES x 2001		-0.20 (-0.94, 0.56)	-0.14 (-0.99, 0.61)	-0.45 (-1.28, 0.38)
Medium SES x 2002		-0.05 (-0.77, 0.69)	0.03 (-0.79, 0.74)	-0.20 (-1.01, 0.57)
Medium SES x 2003		-0.41 (-1.11, 0.31)	-0.34 (-1.14, 0.35)	-0.53 (-1.32, 0.23)
Medium SES x 2004		-0.54 (-1.21, 0.18)	-0.47 (-1.26, 0.19)	-0.66 (-1.43, 0.08)
Medium SES x 2005		-0.62 (-1.30, 0.08)	-0.56 (-1.35, 0.11)	-0.69 (-1.47, 0.04)

Medium SES x 2006		-0.58 (-1.25, 0.12)	-0.53 (-1.32, 0.13)	-0.69 (-1.46, 0.03)
Medium SES x 2007		-0.38 (-1.31, 0.55)	-0.32 (-1.29, 0.62)	-0.63 (-1.60, 0.35)
High SES x 2000		-0.85 (-1.56, -0.11)	-0.67 (-1.43, 0.10)	-0.67 (-1.50, 0.11)
High SES x 2001		-0.48 (-1.11, 0.21)	-0.33 (-1.00, 0.37)	-0.52 (-1.29, 0.21)
High SES x 2002		-0.24 (-0.82, 0.43)	-0.08 (-0.70, 0.60)	-0.18 (-0.93, 0.50)
High SES x 2003		-0.62 (-1.18, 0.02)	-0.47 (-1.07, 0.19)	-0.57 (-1.31, 0.10)
High SES x 2004		-0.42 (-0.95, 0.21)	-0.27 (-0.85, 0.38)	-0.39 (-1.10, 0.25)
High SES x 2005		-0.52 (-1.05, 0.10)	-0.36 (-0.94, 0.30)	-0.46 (-1.18, 0.18)
High SES x 2006		-0.55 (-1.09, 0.06)	-0.41 (-0.99, 0.25)	-0.51 (-1.23, 0.13)
High SES x 2007		-0.55 (-1.82, 0.67)	-0.38 (-1.66, 0.87)	-0.45 (-1.78, 0.82)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.13 (0.05, 0.20)	0.01 (-0.06, 0.09)
Age: 75+ years			0.53 (0.44, 0.62)	0.38 (0.28, 0.47)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.01 (-0.08, 0.06)	-0.01 (-0.09, 0.06)
Duration 10+ years			0.08 (0.00, 0.16)	0.18 (0.09, 0.27)
Ethnicity, reference group: White				
South Asian			0.16 (0.00, 0.32)	0.22 (0.05, 0.38)
Other Ethnicity			0.19 (-0.18, 0.55)	0.30 (-0.07, 0.67)
Male			0.23 (0.17, 0.30)	0.24 (0.18, 0.31)
Smoking status, reference group: Non smoker				
Smoker			0.28 (0.19, 0.37)	0.26 (0.17, 0.36)
Ex-smoker			0.08 (0.01, 0.15)	0.07 (0.00, 0.14)
BMI, reference group: Under or normal weight				
Overweight			0.03 (-0.06, 0.13)	-0.01 (-0.11, 0.08)
Obese			0.12 (0.03, 0.21)	0.06 (-0.04, 0.16)
Hypertensive			0.14 (0.07, 0.20)	0.18 (0.11, 0.24)
Cholesterol			0.03 (0.00, 0.05)	0.03 (0.00, 0.06)
HbA1c			0.06 (0.04, 0.08)	0.09 (0.06, 0.11)
<b>Interventions</b>				
Quality of Care level, reference group: Low quality				
Medium quality				-0.13 (-0.54, 0.25)
High quality				-0.25 (-0.66, 0.14)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				0.14 (0.04, 0.24)
Combination, no insulin				0.02 (-0.10, 0.13)
Insulin only				0.18 (0.04, 0.32)
Combination with insulin				0.20 (0.10, 0.30)
Blood pressure treatment, reference group: No treatments				
ACE inhibitors only				0.36 (0.25, 0.47)
Combination with ACEI				0.52 (0.43, 0.61)
Combination, no ACEI				0.32 (0.22, 0.41)
Aspirin				0.08 (0.01, 0.14)
Lipid therapy				-0.05 (-0.12, 0.02)
Middlesbrough PCT				0.58 (0.29, 0.86)
Shared care				-0.92 (-1.01, -0.83)
<b>Cons</b>	-1.06 (-1.33, -	-1.61 (-2.01, -	-2.69 (-3.21, -	-2.73 (-3.43, -

	0.84)	1.25)	2.11)	2.11)
<b>Variance estimates (Standard Error):</b>				
<b>Practice level</b>	0.23 (0.15, 0.37)	0.24 (0.15, 0.39)	0.24 (0.15, 0.38)	0.21 (0.13, 0.34)
<b>Patient level</b>	0.05 (0.01, 0.21)	0.01 (0.00, 0.04)	0.01 (0.00, 0.04)	0.01 (0.00, 0.03)
<b>Bayesian DIC</b>	27573.79	26339.88	26095.32	25480.02

N = 23,304

Table 62: Stepwise logistic regression multilevel models examining incidences of retinopathy by SES 2000 to 2007 with interaction effect between SES and visit year, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status &amp; visit year</b>				
Social-economic status, reference group: Low				
Medium		-0.11 (-0.69, 0.41)	-0.26 (-0.94, 0.37)	-0.37 (-1.07, 0.29)
High		-0.14 (-0.68, 0.37)	-0.42 (-1.00, 0.17)	-0.45 (-1.09, 0.11)
Visit year, reference group: 1999				
2000		-0.23 (-0.59, 0.14)	-0.22 (-0.63, 0.19)	-0.17 (-0.60, 0.26)
2001		-0.46 (-0.82, -0.10)	-0.34 (-0.73, 0.06)	-0.25 (-0.67, 0.17)
2002		-0.43 (-0.77, -0.09)	-0.25 (-0.63, 0.15)	-0.10 (-0.51, 0.30)
2003		-0.53 (-0.88, -0.19)	-0.30 (-0.69, 0.08)	-0.17 (-0.56, 0.23)
2004		-0.57 (-0.91, -0.23)	-0.27 (-0.65, 0.12)	-0.06 (-0.46, 0.34)
2005		-0.42 (-0.77, -0.08)	-0.10 (-0.50, 0.31)	0.11 (-0.31, 0.53)
2006		-1.72 (-2.08, -1.36)	-1.14 (-1.53, -0.73)	-0.94 (-1.36, -0.50)
2007		-0.63 (-0.95, -0.30)	-0.02 (-0.39, 0.37)	0.23 (-0.18, 0.64)
SES x Visit year, reference group: Low SES x 1999				
Medium SES x 2000		0.35 (-0.28, 1.03)	0.39 (-0.37, 1.17)	0.46 (-0.29, 1.25)
Medium SES x 2001		0.33 (-0.29, 0.98)	0.34 (-0.39, 1.09)	0.48 (-0.26, 1.25)
Medium SES x 2002		-0.07 (-0.66, 0.57)	0.01 (-0.70, 0.76)	0.10 (-0.62, 0.85)
Medium SES x 2003		-0.38 (-0.98, 0.25)	-0.21 (-0.91, 0.52)	-0.05 (-0.77, 0.71)
Medium SES x 2004		-0.05 (-0.62, 0.58)	0.13 (-0.56, 0.87)	0.24 (-0.47, 0.98)
Medium SES x 2005		-0.08 (-0.66, 0.54)	0.17 (-0.54, 0.90)	0.31 (-0.41, 1.05)
Medium SES x 2006		0.26 (-0.34, 0.89)	0.39 (-0.31, 1.13)	0.54 (-0.16, 1.29)
Medium SES x 2007		0.05 (-0.50, 0.65)	0.27 (-0.41, 0.98)	0.37 (-0.31, 1.10)
High SES x 2000		0.15 (-0.47, 0.78)	0.40 (-0.29, 1.09)	0.36 (-0.32, 1.11)
High SES x 2001		0.16 (-0.44, 0.78)	0.40 (-0.27, 1.06)	0.48 (-0.18, 1.20)
High SES x 2002		0.16 (-0.41, 0.76)	0.42 (-0.23, 1.05)	0.39 (-0.23, 1.10)
High SES x 2003		0.08 (-0.49, 0.68)	0.34 (-0.29, 0.98)	0.35 (-0.26, 1.04)
High SES x 2004		0.03 (-0.52, 0.63)	0.32 (-0.33, 0.94)	0.35 (-0.27, 1.05)
High SES x 2005		0.16 (-0.42, 0.75)	0.55 (-0.10, 1.19)	0.62 (-0.01, 1.32)
High SES x 2006		0.08 (-0.51, 0.68)	0.37 (-0.28, 1.02)	0.45 (-0.18, 1.15)
High SES x 2007		-0.13 (-0.67, 0.44)	0.20 (-0.44, 0.81)	0.22 (-0.38, 0.89)
<b>Socio-demographic, anthropometric, lifestyle and health covariates</b>				
Age: 60-74 years			-0.05 (-0.16, 0.05)	0.00 (-0.11, 0.11)

Age: 75+ years			-0.29 (-0.42, -0.15)	-0.11 (-0.25, 0.03)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.64 (0.51, 0.77)	0.47 (0.34, 0.60)
Duration 10+ years			2.00 (1.88, 2.13)	1.60 (1.47, 1.73)
Ethnicity, reference group: White				
South Asian			-0.23 (-0.47, 0.00)	-0.16 (-0.39, 0.08)
Other Ethnicity			0.64 (0.21, 1.07)	0.53 (0.08, 0.95)
Male			0.25 (0.16, 0.34)	0.25 (0.16, 0.34)
Smoking status, reference group: non smoker				
Smoker			-0.14 (-0.27, -0.01)	-0.13 (-0.27, 0.00)
Ex-smoker			-0.10 (-0.19, -0.01)	-0.12 (-0.22, -0.03)
Obesity status, reference group: under and normal weight				
Overweight			-0.05 (-0.19, 0.08)	-0.09 (-0.22, 0.04)
Obese			0.02 (-0.11, 0.15)	-0.10 (-0.23, 0.03)
HbA1c			0.16 (0.13, 0.19)	0.05 (0.02, 0.08)
Hypertensive			0.38 (0.29, 0.47)	0.33 (0.25, 0.42)
Cholesterol			-0.05 (-0.09, -0.01)	-0.02 (-0.06, 0.03)
eGFR			-0.01 (-0.02, -0.01)	-0.01 (-0.01, -0.01)

#### Interventions

Quality of care level, reference group: Mid quality				
High quality				-0.07 (-0.17, 0.04)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				0.40 (0.24, 0.56)
Combination, no insulin				0.70 (0.52, 0.87)
Insulin only				1.05 (0.86, 1.23)
Combination with insulin				1.16 (0.96, 1.36)
Blood pressure treatment, reference group: No treatments				
ACE inhibitors only				0.32 (0.17, 0.46)
Combination with ACEI				0.22 (0.10, 0.35)
Combination, no ACEI				0.13 (0.00, 0.26)
Aspirin				0.05 (-0.04, 0.15)
Lipid therapy				-0.06 (-0.16, 0.04)
Middlesbrough PCT				-0.05 (-0.22, 0.13)
Shared care				0.52 (0.42, 0.63)
<b>Cons</b>	-1.19 (-1.50, -0.88)	-0.39 (-0.87, 0.11)	-2.44 (-3.00, -1.89)	-2.83 (-3.48, -2.19)

#### Variance estimate at:

<b>Practice level</b>	0.07 (0.04, 0.12)	0.08 (0.04, 0.13)	0.06 (0.03, 0.10)	0.05 (0.03, 0.10)
<b>Patient level</b>	0.18 (0.05, 0.55)	0.34 (0.10, 1.03)	0.02 (0.00, 0.07)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	17531.30	17098.58	15146.30	14536.53

N= 18,665

## Appendix G: Stepwise models of interventions with interaction between visit year and socio-economic status

### Timeliness of diagnosis

Table 63: Stepwise linear regression multilevel models examining timeliness of diagnosis with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		0.83 (-0.16, 1.78)	0.71 (-0.23, 1.64)	0.69 (-0.20, 1.55)
High		0.38 (-0.46, 1.21)	0.35 (-0.46, 1.16)	0.27 (-0.49, 1.03)
Visit year, reference group: 1999				
2000		-0.03 (-0.73, 0.68)	-0.02 (-0.72, 0.68)	-0.24 (-0.90, 0.39)
2001		-0.82 (-1.47, -0.18)	-0.80 (-1.43, -0.16)	-0.76 (-1.37, -0.18)
2002		-0.25 (-0.84, 0.34)	-0.23 (-0.82, 0.35)	-0.12 (-0.68, 0.42)
2003		-0.67 (-1.26, -0.09)	-0.64 (-1.22, -0.07)	-0.42 (-0.95, 0.12)
2004		-0.58 (-1.16, -0.02)	-0.52 (-1.09, 0.03)	-0.42 (-0.97, 0.11)
2005		-0.81 (-1.39, -0.24)	-0.77 (-1.35, -0.21)	-0.59 (-1.14, -0.05)
2006		-1.19 (-1.76, -0.62)	-1.15 (-1.73, -0.57)	-0.99 (-1.54, -0.44)
2007		-1.30 (-1.89, -0.71)	-1.24 (-1.84, -0.65)	-1.14 (-1.73, -0.57)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		-0.79 (-1.99, 0.43)	-0.56 (-1.74, 0.64)	-0.40 (-1.50, 0.71)
Mid. SES*2001		0.02 (-1.13, 1.16)	0.17 (-0.93, 1.29)	0.25 (-0.80, 1.31)
Mid. SES*2002		-1.35 (-2.42, -0.28)	-1.23 (-2.28, -0.19)	-1.11 (-2.08, -0.12)
Mid. SES*2003		-0.76 (-1.80, 0.31)	-0.68 (-1.70, 0.34)	-0.64 (-1.60, 0.32)
Mid. SES*2004		-1.21 (-2.23, -0.18)	-1.05 (-2.03, -0.04)	-0.91 (-1.84, 0.04)
Mid. SES*2005		-0.76 (-1.79, 0.31)	-0.58 (-1.58, 0.46)	-0.41 (-1.35, 0.54)
Mid. SES*2006		-0.54 (-1.58, 0.52)	-0.37 (-1.40, 0.66)	-0.37 (-1.33, 0.60)
Mid. SES*2007		-0.44 (-1.48, 0.65)	-0.33 (-1.37, 0.72)	-0.45 (-1.41, 0.55)
High SES*2000		-1.14 (-2.22, -0.06)	-1.13 (-2.18, -0.07)	-0.76 (-1.75, 0.23)
High SES*2001		-0.22 (-1.24, 0.79)	-0.13 (-1.13, 0.88)	-0.03 (-0.96, 0.90)
High SES*2002		-1.01 (-1.91, -0.08)	-0.88 (-1.77, 0.05)	-0.70 (-1.53, 0.16)
High SES*2003		-0.77 (-1.69, 0.15)	-0.64 (-1.54, 0.27)	-0.50 (-1.33, 0.34)
High SES*2004		-0.59 (-1.49, 0.31)	-0.51 (-1.38, 0.36)	-0.33 (-1.14, 0.49)
High SES*2005		-0.72 (-1.62, 0.19)	-0.61 (-1.50, 0.31)	-0.44 (-1.28, 0.39)
High SES*2006		-0.84 (-1.76, 0.08)	-0.69 (-1.59, 0.19)	-0.43 (-1.26, 0.42)
High SES*2007		-0.57 (-1.50, 0.37)	-0.43 (-1.32, 0.49)	-0.24 (-1.08, 0.61)
<b>Covariates</b>				
Age at diagnosis, reference group: <60				
60-74			-0.27 (-0.42, -0.11)	-0.08 (-0.22, 0.07)
75+			-0.36 (-0.58, -0.14)	-0.12 (-0.33, 0.08)
Ethnicity, reference group: white				
South Asian			0.63 (0.31, 0.94)	0.47 (0.17, 0.77)
Other Ethnicity			0.55 (-0.14, 1.25)	0.32 (-0.32, 0.98)
Male			0.01 (-0.12, 0.14)	0.05 (-0.08, 0.17)
Smoking status, reference group: non-smoker				
Smoker			0.31 (0.14, 0.48)	0.21 (0.05, 0.36)
Ex-smoker			0.04 (-0.11, 0.18)	0.03 (-0.11, 0.16)
Obesity status, reference group: Under and normal weight				
Overweight			0.05 (-0.16, 0.25)	0.16 (-0.03, 0.35)
Obese			-0.07 (-0.27, 0.13)	0.05 (-0.14, 0.24)
Hypertensive			0.02 (-0.11, 0.15)	0.04 (-0.08, 0.17)
Cholesterol			0.15 (0.10, 0.19)	0.16 (0.12, 0.20)
Creatinine > 300			0.21 (-2.24, 2.64)	0.53 (-1.78, 2.75)
eGFR			0.00 (0.00, 0.01)	0.00 (0.00, 0.01)
Ischaemic Cardiac			-0.01 (-0.17, 0.16)	0.09 (-0.09, 0.26)

Stroke or TIA			-0.16 (-0.45, 0.13)	-0.10 (-0.37, 0.18)
PVD			-0.01 (-0.44, 0.42)	-0.01 (-0.41, 0.39)
<b>Interventions</b>				
Care level, reference group: <7				
Care level: 7				-0.20 (-0.34, -0.05)
Care level: 8				-0.11 (-0.29, 0.08)
Diabetes treatment, reference group diet alone				
Metformin only				1.01 (0.87, 1.15)
Sulphonylurea only				1.03 (0.82, 1.25)
Insulin only				2.00 (1.65, 2.35)
Combination/other				1.64 (1.44, 1.84)
BP treatment, reference group no treatments				
ACE inhibitors only				-0.32 (-0.54, -0.10)
ACE & other(s)				-0.25 (-0.42, -0.07)
Other BP				-0.13 (-0.29, 0.03)
Aspirin				0.04 (-0.11, 0.19)
Lipid therapy				-0.11 (-0.24, 0.03)
Shared care				0.20 (0.01, 0.38)
M. PCT				0.14 (-0.09, 0.38)
<b>Cons</b>	7.71 (7.59, 7.85)	8.54 (8.00, 9.07)	7.51 (6.79, 8.26)	6.79 (6.06, 7.50)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.12 (0.06, 0.22)	0.10 (0.05, 0.19)	0.09 (0.04, 0.17)	0.10 (0.05, 0.18)
<b>Patient level</b>	3.24 (3.08, 3.41)	3.10 (2.95, 3.26)	2.98 (2.83, 3.14)	2.57 (2.45, 2.71)
<b>Bayesian DIC</b>	12356.02	12244.78	12139.62	11700.65

N = 3,071

## Quality of care

Table 64: Stepwise linear regression multilevel models examining quality of care with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		-0.02 (-0.15, 0.11)	-0.03 (-0.15, 0.10)	-0.04 (-0.16, 0.09)
High		-0.18 (-0.30, -0.07)	-0.20 (-0.31, -0.08)	-0.16 (-0.28, -0.05)
Visit year, reference group: 1999				
2000		0.04 (-0.05, 0.13)	0.03 (-0.05, 0.12)	0.05 (-0.03, 0.14)
2001		0.06 (-0.02, 0.14)	0.05 (-0.03, 0.14)	0.11 (0.02, 0.19)
2002		-0.01 (-0.09, 0.07)	-0.02 (-0.10, 0.06)	0.06 (-0.02, 0.14)
2003		-0.01 (-0.09, 0.07)	-0.03 (-0.10, 0.05)	0.06 (-0.01, 0.14)
2004		0.08 (0.00, 0.15)	0.05 (-0.03, 0.13)	0.15 (0.07, 0.23)
2005		-0.06 (-0.14, 0.01)	-0.10 (-0.18, -0.02)	0.02 (-0.06, 0.10)
2006		0.19 (0.11, 0.27)	0.15 (0.07, 0.22)	0.27 (0.19, 0.34)
2007		-0.47 (-0.55, -0.39)	-0.51 (-0.59, -0.43)	-0.40 (-0.47, -0.32)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		-0.07 (-0.22, 0.08)	-0.06 (-0.21, 0.09)	-0.03 (-0.18, 0.12)
Mid. SES*2001		-0.05 (-0.20, 0.10)	-0.05 (-0.19, 0.10)	-0.01 (-0.16, 0.13)
Mid. SES*2002		0.09 (-0.05, 0.23)	0.09 (-0.05, 0.23)	0.11 (-0.03, 0.24)
Mid. SES*2003		0.05 (-0.09, 0.19)	0.06 (-0.08, 0.19)	0.07 (-0.07, 0.21)
Mid. SES*2004		-0.02 (-0.15, 0.12)	-0.01 (-0.15, 0.12)	-0.01 (-0.14, 0.13)
Mid. SES*2005		0.03 (-0.11, 0.17)	0.03 (-0.10, 0.17)	0.03 (-0.10, 0.17)
Mid. SES*2006		0.06 (-0.07, 0.20)	0.07 (-0.07, 0.20)	0.06 (-0.07, 0.20)
Mid. SES*2007		0.09 (-0.05, 0.23)	0.09 (-0.05, 0.22)	0.08 (-0.05, 0.22)
High SES*2000		0.10 (-0.04, 0.24)	0.11 (-0.03, 0.24)	0.09 (-0.05, 0.22)
High SES*2001		0.12 (-0.01, 0.25)	0.13 (0.00, 0.26)	0.11 (-0.02, 0.24)
High SES*2002		0.24 (0.11, 0.36)	0.25 (0.12, 0.37)	0.21 (0.09, 0.33)

High SES*2003		0.20 (0.08, 0.32)	0.21 (0.09, 0.33)	0.17 (0.05, 0.29)
High SES*2004		0.22 (0.09, 0.34)	0.22 (0.10, 0.34)	0.18 (0.06, 0.30)
High SES*2005		0.23 (0.11, 0.35)	0.24 (0.12, 0.36)	0.19 (0.07, 0.31)
High SES*2006		0.24 (0.12, 0.36)	0.25 (0.13, 0.37)	0.21 (0.09, 0.32)
High SES*2007		0.19 (0.07, 0.31)	0.19 (0.07, 0.31)	0.16 (0.03, 0.28)
<b>Covariates</b>				
Age, reference group: <60				
60-74			0.02 (0.00, 0.04)	0.04 (0.02, 0.06)
75+			-0.05 (-0.07, -0.02)	-0.01 (-0.03, 0.02)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.08 (0.06, 0.09)	0.06 (0.04, 0.07)
Duration 10+ years			0.07 (0.05, 0.09)	0.01 (-0.01, 0.03)
Ethnicity, reference group: white				
South Asian			-0.08 (-0.12, -0.05)	-0.08 (-0.12, -0.04)
Other Ethnicity			-0.06 (-0.14, 0.03)	-0.08 (-0.16, 0.00)
Male			-0.02 (-0.03, 0.00)	-0.01 (-0.03, 0.00)
Smoking status, reference group: non-smoker				
Smoker			-0.08 (-0.10, -0.06)	-0.06 (-0.09, -0.04)
Ex-smoker			0.02 (0.00, 0.04)	0.02 (0.00, 0.04)
Obesity status, reference group: Under and normal weight				
Overweight			0.04 (0.02, 0.06)	0.03 (0.01, 0.05)
Obese			0.02 (0.00, 0.05)	0.00 (-0.02, 0.02)
Hypertensive			-0.01 (-0.02, 0.01)	-0.03 (-0.04, -0.01)
HbA1c			0.00 (0.00, 0.01)	-0.01 (-0.02, -0.01)
Cholesterol			-0.03 (-0.04, -0.02)	-0.02 (-0.03, -0.02)
Creatinine > 300			-0.07 (-0.20, 0.06)	-0.07 (-0.19, 0.06)
eGFR			0.00 (0.00, 0.00)	0.00 (0.00, 0.00)
Ischaemic Cardiac Stroke or TIA			-0.02 (-0.03, 0.00)	-0.03 (-0.05, -0.01)
PVD			0.00 (-0.02, 0.03)	-0.01 (-0.03, 0.02)
			0.06 (0.03, 0.09)	0.02 (-0.01, 0.04)
<b>Interventions</b>				
Diabetes treatment, reference group diet alone				
Sulphonylures / metformin only				0.05 (0.03, 0.08)
OHA comb.				0.02 (-0.01, 0.04)
Insulin only				0.02 (-0.01, 0.05)
Insulin & OHAs				0.04 (0.02, 0.07)
BP treatment, reference group no treatments				
ACE inhibitors only				0.04 (0.01, 0.06)
ACE & other(s)				0.02 (0.00, 0.05)
Other BP				0.02 (0.00, 0.04)
Aspirin				0.00 (-0.02, 0.01)
Lipid therapy				0.02 (0.00, 0.03)
Shared care				0.27 (0.25, 0.29)
M. PCT				-0.07 (-0.18, 0.03)
<b>Cons</b>	7.12 (7.05, 7.19)	7.13 (7.03, 7.23)	7.18 (7.06, 7.29)	7.06 (6.93, 7.19)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.03 (0.02, 0.05)	0.03 (0.02, 0.04)	0.03 (0.02, 0.04)	0.03 (0.02, 0.05)
<b>Patient level</b>	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)
<b>Visit year level</b>	0.47 (0.47, 0.48)	0.43 (0.43, 0.44)	0.43 (0.42, 0.44)	0.42 (0.41, 0.43)
<b>Bayesian DIC</b>	69260.18	66475.44	66137.73	65285.29

## Diabetes treatments

Table 65: Stepwise logistic regression multilevel model examining no blood glucose treatments with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		-0.34 (-0.64, -0.03)	-0.36 (-0.70, -0.03)	-0.31 (-0.65, 0.03)
High		-0.04 (-0.31, 0.23)	-0.03 (-0.32, 0.29)	-0.05 (-0.34, 0.25)
Visit year, reference group: 1999				
2000		-0.25 (-0.49, 0.00)	-0.46 (-0.73, -0.17)	-0.45 (-0.71, -0.17)
2001		-0.15 (-0.38, 0.09)	-0.57 (-0.85, -0.30)	-0.69 (-0.96, -0.43)
2002		-0.09 (-0.31, 0.13)	-0.43 (-0.67, -0.17)	-0.62 (-0.87, -0.37)
2003		-0.20 (-0.40, 0.02)	-0.51 (-0.75, -0.27)	-0.74 (-0.97, -0.51)
2004		-0.32 (-0.51, -0.10)	-0.53 (-0.77, -0.29)	-0.81 (-1.04, -0.58)
2005		-0.39 (-0.60, -0.18)	-0.60 (-0.84, -0.35)	-0.95 (-1.18, -0.72)
2006		-0.26 (-0.47, -0.06)	-0.79 (-1.03, -0.55)	-1.15 (-1.37, -0.92)
2007		-0.46 (-0.67, -0.25)	-0.83 (-1.07, -0.58)	-1.21 (-1.45, -0.98)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		0.30 (-0.12, 0.73)	0.27 (-0.19, 0.72)	0.10 (-0.38, 0.57)
Mid. SES*2001		0.39 (-0.01, 0.80)	0.44 (0.00, 0.89)	0.27 (-0.17, 0.72)
Mid. SES*2002		0.34 (-0.03, 0.70)	0.28 (-0.13, 0.68)	0.17 (-0.26, 0.57)
Mid. SES*2003		0.47 (0.11, 0.84)	0.45 (0.06, 0.85)	0.37 (-0.04, 0.77)
Mid. SES*2004		0.47 (0.11, 0.82)	0.35 (-0.04, 0.74)	0.29 (-0.11, 0.67)
Mid. SES*2005		0.61 (0.25, 0.96)	0.56 (0.17, 0.94)	0.54 (0.15, 0.94)
Mid. SES*2006		0.43 (0.08, 0.78)	0.40 (0.02, 0.78)	0.39 (0.00, 0.76)
Mid. SES*2007		0.50 (0.14, 0.85)	0.47 (0.08, 0.85)	0.47 (0.08, 0.87)
High SES*2000		0.32 (-0.05, 0.69)	0.15 (-0.28, 0.57)	0.10 (-0.33, 0.53)
High SES*2001		0.14 (-0.21, 0.50)	0.15 (-0.27, 0.55)	0.09 (-0.32, 0.49)
High SES*2002		0.10 (-0.24, 0.43)	-0.05 (-0.43, 0.32)	0.01 (-0.37, 0.38)
High SES*2003		0.16 (-0.17, 0.48)	0.01 (-0.36, 0.36)	0.05 (-0.31, 0.40)
High SES*2004		0.21 (-0.11, 0.52)	0.00 (-0.36, 0.35)	0.05 (-0.30, 0.39)
High SES*2005		0.26 (-0.05, 0.57)	0.09 (-0.27, 0.43)	0.15 (-0.20, 0.48)
High SES*2006		0.21 (-0.10, 0.51)	0.07 (-0.29, 0.40)	0.14 (-0.20, 0.47)
High SES*2007		0.38 (0.06, 0.69)	0.21 (-0.16, 0.56)	0.25 (-0.10, 0.59)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.29 (0.21, 0.37)	0.19 (0.11, 0.27)
Age: 75+ years			0.60 (0.50, 0.70)	0.44 (0.34, 0.54)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 yrs			-1.09 (-1.16, -1.02)	-1.07 (-1.14, -1.00)
Duration 10+ yrs			-1.78 (-1.87, -1.69)	-1.64 (-1.74, -1.55)
Ethnicity, reference group: white				
South Asian			0.12 (-0.06, 0.30)	0.13 (-0.06, 0.32)
Other Ethnicity			-0.06 (-0.47, 0.33)	0.10 (-0.33, 0.51)
Male			0.27 (0.20, 0.33)	0.28 (0.22, 0.35)
Smoking status, reference group: non-smoker				
Smoker			-0.02 (-0.11, 0.07)	-0.07 (-0.16, 0.03)
Ex-smoker			-0.09 (-0.16, -0.02)	-0.09 (-0.16, -0.02)
Obesity categories, reference group: under and normal weight				
Overweight			-0.23 (-0.32, -0.14)	-0.23 (-0.32, -0.14)
Obese			-0.44 (-0.53, -0.35)	-0.43 (-0.52, -0.34)
HbA1c			-0.85 (-0.88, -0.81)	-0.82 (-0.85, -0.79)
Hypertensive			-0.03 (-0.09, 0.04)	0.03 (-0.04, 0.09)
Cholesterol			0.25 (0.22, 0.28)	0.24 (0.21, 0.27)
Creatinine > 300			-0.24 (-0.77, 0.27)	-0.18 (-0.75, 0.34)
eGFR			0.00 (-0.01, 0.00)	0.00 (-0.01, 0.00)
Ischaemic Cardiac			0.02 (-0.05, 0.08)	0.06 (-0.01, 0.13)
Stroke or TIA			-0.13 (-0.23, -0.02)	-0.08 (-0.19, 0.03)
PVD			-0.45 (-0.59, -0.32)	-0.33 (-0.46, -0.19)
<b>Interventions</b>				
Care level, reference group: <7				

Care level: 7				-0.15 (-0.22, -0.07)
Care level: 8				-0.14 (-0.23, -0.05)
Shared care				-1.33 (-1.43, -1.23)
M'brough PCT				0.01 (-0.34, 0.36)
<b>Cons</b>	-1.72 (-2.03, -1.44)	-1.53 (-1.86, -1.17)	4.7 (2.29, 5.37)	5.43 (5.02, 5.88)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.40 (0.25, 0.64)	0.39 (0.24, 0.62)	0.4 (0.24, 0.64)	0.32 (0.2, 0.51)
<b>Patient level</b>	0.11 (0.03, 0.32)	0.12 (0.04, 0.35)	0.42 (0, 6.4)	0.01 (0, 0.03)
<b>Bayesian DIC</b>	35856.53	35826.93	27984.07	27148.92

Table 66: Stepwise logistic regression multilevel model examining metformin or sulphonylureas only with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		0.20 (-0.12, 0.55)	0.24 (-0.09, 0.57)	0.27 (-0.04, 0.59)
High		-0.53 (-0.89, -0.14)	-0.48 (-0.85, -0.16)	-0.49 (-0.87, -0.14)
Visit year, reference group: 1999				
2000		0.09 (-0.17, 0.37)	0.11 (-0.16, 0.37)	0.11 (-0.14, 0.37)
2001		0.14 (-0.12, 0.41)	0.15 (-0.13, 0.40)	0.10 (-0.15, 0.34)
2002		0.25 (0.02, 0.49)	0.25 (0.00, 0.49)	0.17 (-0.05, 0.39)
2003		0.29 (0.06, 0.52)	0.28 (0.04, 0.50)	0.17 (-0.04, 0.39)
2004		0.44 (0.23, 0.67)	0.46 (0.22, 0.67)	0.30 (0.09, 0.51)
2005		0.48 (0.26, 0.70)	0.49 (0.25, 0.71)	0.31 (0.10, 0.52)
2006		0.59 (0.38, 0.81)	0.54 (0.31, 0.76)	0.34 (0.14, 0.55)
2007		0.66 (0.44, 0.89)	0.68 (0.45, 0.90)	0.53 (0.32, 0.75)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		-0.18 (-0.64, 0.26)	-0.23 (-0.68, 0.23)	-0.28 (-0.73, 0.16)
Mid. SES*2001		-0.38 (-0.82, 0.05)	-0.44 (-0.88, 0.01)	-0.52 (-0.95, -0.08)
Mid. SES*2002		-0.25 (-0.67, 0.14)	-0.29 (-0.69, 0.12)	-0.34 (-0.73, 0.05)
Mid. SES*2003		-0.20 (-0.59, 0.18)	-0.27 (-0.66, 0.12)	-0.31 (-0.68, 0.06)
Mid. SES*2004		-0.18 (-0.56, 0.18)	-0.24 (-0.61, 0.14)	-0.25 (-0.60, 0.10)
Mid. SES*2005		-0.29 (-0.68, 0.07)	-0.33 (-0.70, 0.04)	-0.34 (-0.70, 0.01)
Mid. SES*2006		-0.38 (-0.75, -0.02)	-0.39 (-0.75, -0.02)	-0.40 (-0.76, -0.05)
Mid. SES*2007		-0.39 (-0.77, -0.02)	-0.44 (-0.81, -0.06)	-0.44 (-0.80, -0.09)
High SES*2000		0.01 (-0.49, 0.50)	-0.07 (-0.53, 0.42)	-0.09 (-0.59, 0.40)
High SES*2001		0.30 (-0.17, 0.75)	0.25 (-0.17, 0.71)	0.22 (-0.23, 0.68)
High SES*2002		0.27 (-0.15, 0.70)	0.22 (-0.17, 0.65)	0.23 (-0.19, 0.66)
High SES*2003		0.36 (-0.07, 0.77)	0.32 (-0.06, 0.73)	0.33 (-0.06, 0.76)
High SES*2004		0.34 (-0.08, 0.74)	0.29 (-0.07, 0.69)	0.31 (-0.08, 0.72)
High SES*2005		0.46 (0.05, 0.86)	0.43 (0.07, 0.83)	0.45 (0.07, 0.87)
High SES*2006		0.41 (0.01, 0.80)	0.41 (0.06, 0.80)	0.43 (0.05, 0.84)
High SES*2007		0.43 (0.03, 0.82)	0.39 (0.04, 0.78)	0.41 (0.03, 0.82)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.02 (-0.05, 0.09)	-0.03 (-0.09, 0.04)
Age: 75+ years			0.06 (-0.03, 0.16)	-0.01 (-0.10, 0.08)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.44 (-0.50, -0.38)	-0.42 (-0.48, -0.36)
Duration 10+ years			-1.29 (-1.37, -1.20)	-1.20 (-1.28, -1.11)
Ethnicity, reference: White				
South Asian			0.07 (-0.07, 0.21)	0.07 (-0.08, 0.21)
Other Ethnicity			-0.44 (-0.79, -0.10)	-0.37 (-0.73, -0.04)
Male			-0.19 (-0.24, -0.13)	-0.18 (-0.24, -0.12)
Smoking status, reference group: non-smoker				
Smoker			0.07 (-0.01, 0.16)	0.06 (-0.02, 0.14)
Ex-smoker			0.03 (-0.03, 0.10)	0.03 (-0.03, 0.09)
Obesity category, reference: Under and normal weight				
Overweight			0.47 (0.37, 0.56)	0.46 (0.36, 0.56)
Obese			0.62 (0.53, 0.72)	0.63 (0.54, 0.73)
HbA1c			-0.09 (-0.11, -0.07)	-0.07 (-0.09, -0.06)

Hypertensive			-0.05 (-0.11, 0.00)	-0.02 (-0.08, 0.04)
Cholesterol			0.00 (-0.03, 0.02)	-0.01 (-0.04, 0.02)
Creatinine > 300 eGFR			-3.13 (-6.33, -1.20)	-3.11 (-6.17, -1.20)
Ischaemic Cardiac Stroke or TIA			0.01 (0.01, 0.01)	0.01 (0.01, 0.01)
PVD			-0.06 (-0.13, 0.00)	-0.04 (-0.11, 0.02)
			-0.09 (-0.19, 0.01)	-0.07 (-0.17, 0.03)
			-0.12 (-0.24, -0.01)	-0.06 (-0.18, 0.05)
<b>Interventions</b>				
Care level, reference group: <7				
Care level: 7				0.08 (0.00, 0.15)
Care level: 8				0.15 (0.06, 0.23)
Shared care				-0.54 (-0.61, -0.46)
Middlesbrough PCT				0.14 (-0.05, 0.32)
<b>Cons</b>	-1.59 (-1.87, -1.36)	-1.92 (-2.22, -1.67)	-1.77 (-2.10, -1.40)	-1.73 (-2.08, -1.40)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.07 (0.04, 0.12)	0.07 (0.04, 0.11)	0.07 (0.04, 0.12)	0.09 (0.05, 0.14)
<b>Patient level</b>	0.09 (0.02, 0.28)	0.06 (0.02, 0.19)	0.01 (0.00, 0.02)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	35249.83	35041.67	33153.45	32952.79

Table 67: Stepwise logistic regression multilevel model examining blood glucose treatments, with no insulin, with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		0.21 (-0.06, 0.48)	0.20 (-0.08, 0.49)	0.24 (-0.06, 0.53)
High		0.12 (-0.14, 0.37)	0.02 (-0.27, 0.28)	0.00 (-0.28, 0.27)
Visit year, reference group: 1999				
2000		-0.27 (-0.50, -0.04)	-0.28 (-0.52, -0.03)	-0.25 (-0.51, 0.00)
2001		-0.30 (-0.52, -0.07)	-0.36 (-0.59, -0.13)	-0.39 (-0.64, -0.14)
2002		-0.32 (-0.52, -0.11)	-0.37 (-0.58, -0.15)	-0.45 (-0.68, -0.23)
2003		-0.58 (-0.78, -0.38)	-0.61 (-0.82, -0.39)	-0.72 (-0.94, -0.49)
2004		-0.91 (-1.12, -0.70)	-0.94 (-1.16, -0.72)	-1.10 (-1.32, -0.88)
2005		-0.93 (-1.13, -0.72)	-0.96 (-1.18, -0.75)	-1.17 (-1.40, -0.94)
2006		-1.02 (-1.22, -0.82)	-1.06 (-1.28, -0.84)	-1.25 (-1.48, -1.02)
2007		-1.23 (-1.45, -0.99)	-1.28 (-1.51, -1.04)	-1.48 (-1.73, -1.24)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		0.15 (-0.23, 0.53)	0.11 (-0.29, 0.50)	0.03 (-0.37, 0.44)
Mid. SES*2001		0.02 (-0.36, 0.39)	-0.01 (-0.40, 0.36)	-0.11 (-0.51, 0.29)
Mid. SES*2002		-0.18 (-0.52, 0.17)	-0.21 (-0.58, 0.16)	-0.27 (-0.64, 0.12)
Mid. SES*2003		-0.11 (-0.46, 0.24)	-0.13 (-0.50, 0.22)	-0.19 (-0.55, 0.19)
Mid. SES*2004		0.03 (-0.32, 0.37)	-0.02 (-0.38, 0.34)	-0.06 (-0.42, 0.31)
Mid. SES*2005		-0.26 (-0.61, 0.09)	-0.31 (-0.68, 0.04)	-0.33 (-0.71, 0.05)
Mid. SES*2006		-0.17 (-0.52, 0.18)	-0.24 (-0.60, 0.12)	-0.25 (-0.62, 0.12)
Mid. SES*2007		-0.21 (-0.60, 0.17)	-0.25 (-0.65, 0.14)	-0.28 (-0.68, 0.13)
High SES*2000		0.06 (-0.30, 0.42)	0.10 (-0.28, 0.49)	0.08 (-0.30, 0.47)
High SES*2001		0.15 (-0.20, 0.49)	0.20 (-0.16, 0.58)	0.19 (-0.18, 0.57)
High SES*2002		0.10 (-0.22, 0.43)	0.13 (-0.21, 0.48)	0.17 (-0.17, 0.53)
High SES*2003		0.13 (-0.18, 0.45)	0.18 (-0.15, 0.52)	0.22 (-0.12, 0.56)
High SES*2004		0.11 (-0.20, 0.44)	0.12 (-0.22, 0.46)	0.16 (-0.18, 0.50)
High SES*2005		-0.01 (-0.33, 0.32)	0.00 (-0.34, 0.34)	0.05 (-0.29, 0.40)
High SES*2006		-0.03 (-0.34, 0.30)	-0.02 (-0.35, 0.33)	0.02 (-0.31, 0.37)
High SES*2007		0.02 (-0.33, 0.36)	0.02 (-0.34, 0.39)	0.04 (-0.32, 0.42)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.27 (0.18, 0.36)	0.21 (0.12, 0.30)
Age: 75+ years			0.63 (0.52, 0.73)	0.52 (0.41, 0.63)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 yrs			0.11 (0.03, 0.19)	0.14 (0.07, 0.22)
Duration 10+ yrs			-0.39 (-0.48, -0.30)	-0.27 (-0.36, -0.18)
Ethnicity, reference group: white				
South Asian			0.12 (-0.06, 0.28)	0.12 (-0.06, 0.29)
Other Ethnicity			-0.17 (-0.63, 0.24)	-0.11 (-0.56, 0.31)

Male			0.34 (0.27, 0.41)	0.34 (0.26, 0.41)
Smoking status, reference group: non-smoker				
Smoker			0.09 (-0.01, 0.19)	0.07 (-0.03, 0.17)
Ex-smoker			0.00 (-0.07, 0.08)	0.02 (-0.06, 0.09)
Obesity category, reference group: under and normal weight				
Overweight			-0.26 (-0.35, -0.17)	-0.25 (-0.34, -0.17)
Obese			-0.72 (-0.82, -0.63)	-0.70 (-0.79, -0.61)
HbA1c			-0.01 (-0.04, 0.01)	0.01 (-0.01, 0.04)
Hypertensive			-0.01 (-0.08, 0.06)	0.02 (-0.05, 0.09)
Cholesterol			0.01 (-0.02, 0.04)	0.00 (-0.03, 0.03)
Creatinine > 300			0.23 (-0.22, 0.67)	0.25 (-0.21, 0.69)
eGFR			-0.01 (-0.02, -0.01)	-0.01 (-0.02, -0.01)
Ischaemic Cardiac			-0.03 (-0.11, 0.04)	-0.01 (-0.08, 0.06)
Stroke or TIA			0.03 (-0.08, 0.13)	0.06 (-0.04, 0.17)
PVD			-0.18 (-0.31, -0.06)	-0.09 (-0.22, 0.03)
<b>Interventions</b>				
Care level, reference group: <7				
Care level: 7				0.06 (-0.03, 0.14)
Care level: 8				-0.05 (-0.15, 0.05)
Shared care				-0.62 (-0.71, -0.53)
M'brough PCT				-0.01 (-0.19, 0.18)
Cons	-1.97 (-2.08, -1.87)	-1.37 (-1.56, -1.19)	-0.39 (-0.72, -0.03)	-0.22 (-0.65, 0.19)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.08 (0.05, 0.15)	0.07 (0.04, 0.13)	0.07 (0.04, 0.11)	0.07 (0.04, 0.13)
<b>Patient level</b>	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)
<b>Bayesian DIC</b>	27417.14	26846.58	25873.66	25654.38

Table 68: Stepwise logistic regression multilevel model examining insulin only with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		0.00 (-0.31, 0.31)	-0.08 (-0.42, 0.28)	-0.26 (-0.61, 0.09)
High		-0.04 (-0.32, 0.25)	-0.13 (-0.44, 0.17)	-0.11 (-0.45, 0.24)
Visit year, reference group: 1999				
2000		0.12 (-0.11, 0.35)	0.08 (-0.18, 0.34)	0.06 (-0.22, 0.33)
2001		-0.03 (-0.26, 0.20)	-0.06 (-0.31, 0.21)	0.11 (-0.17, 0.39)
2002		-0.41 (-0.63, -0.19)	-0.48 (-0.73, -0.23)	-0.23 (-0.49, 0.03)
2003		-0.46 (-0.66, -0.24)	-0.53 (-0.77, -0.28)	-0.21 (-0.46, 0.04)
2004		-0.58 (-0.78, -0.37)	-0.66 (-0.90, -0.42)	-0.14 (-0.39, 0.11)
2005		-0.69 (-0.90, -0.48)	-0.79 (-1.03, -0.54)	-0.12 (-0.38, 0.13)
2006		-0.83 (-1.04, -0.62)	-0.73 (-0.97, -0.48)	-0.05 (-0.31, 0.21)
2007		-0.71 (-0.93, -0.49)	-0.71 (-0.96, -0.46)	-0.03 (-0.29, 0.24)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		-0.30 (-0.73, 0.12)	-0.15 (-0.63, 0.32)	-0.01 (-0.50, 0.46)
Mid. SES*2001		-0.45 (-0.88, -0.03)	-0.37 (-0.84, 0.10)	-0.11 (-0.59, 0.37)
Mid. SES*2002		0.05 (-0.34, 0.44)	0.22 (-0.22, 0.66)	0.46 (0.00, 0.90)
Mid. SES*2003		-0.10 (-0.48, 0.28)	0.17 (-0.25, 0.58)	0.40 (-0.04, 0.84)
Mid. SES*2004		-0.11 (-0.48, 0.27)	0.07 (-0.37, 0.48)	0.19 (-0.25, 0.62)
Mid. SES*2005		0.08 (-0.30, 0.46)	0.28 (-0.14, 0.68)	0.36 (-0.06, 0.80)
Mid. SES*2006		0.14 (-0.24, 0.52)	0.28 (-0.16, 0.70)	0.37 (-0.06, 0.80)
Mid. SES*2007		0.07 (-0.32, 0.46)	0.27 (-0.16, 0.68)	0.40 (-0.04, 0.84)
High SES*2000		0.08 (-0.30, 0.47)	0.34 (-0.07, 0.76)	0.34 (-0.12, 0.80)
High SES*2001		-0.07 (-0.44, 0.32)	0.07 (-0.34, 0.48)	0.07 (-0.38, 0.52)
High SES*2002		0.17 (-0.18, 0.54)	0.44 (0.05, 0.84)	0.38 (-0.04, 0.80)
High SES*2003		0.18 (-0.16, 0.53)	0.45 (0.08, 0.82)	0.38 (-0.03, 0.79)
High SES*2004		0.19 (-0.14, 0.54)	0.40 (0.05, 0.77)	0.31 (-0.10, 0.72)
High SES*2005		0.16 (-0.20, 0.51)	0.38 (0.01, 0.76)	0.23 (-0.19, 0.65)
High SES*2006		0.31 (-0.03, 0.66)	0.51 (0.14, 0.89)	0.46 (0.05, 0.86)
High SES*2007		0.10 (-0.25, 0.46)	0.34 (-0.03, 0.73)	0.32 (-0.11, 0.73)
<b>Covariates</b>				
Age, reference group: <60 years				

Age: 60-74 years				-0.62 (-0.71, -0.53)	-0.46 (-0.55, -0.36)
Age: 75+ years				-0.99 (-1.11, -0.88)	-0.63 (-0.76, -0.50)
Duration of diabetes, reference group: 0-3 years					
Duration: 4-9 yrs				0.69 (0.58, 0.79)	0.57 (0.46, 0.68)
Duration 10+ yrs				1.77 (1.67, 1.87)	1.42 (1.31, 1.53)
Ethnicity, reference group: white					
South Asian				-0.51 (-0.70, -0.33)	-0.58 (-0.77, -0.39)
Other Ethnicity				0.72 (0.37, 1.04)	0.55 (0.19, 0.89)
Male				-0.07 (-0.15, 0.00)	-0.07 (-0.15, 0.01)
Smoking status, reference group: non-smoker					
Smoker				0.01 (-0.09, 0.12)	0.16 (0.05, 0.27)
Ex-smoker				0.00 (-0.08, 0.08)	0.00 (-0.08, 0.08)
Obesity status, reference group: under and normal weight					
Overweight				-0.31 (-0.41, -0.21)	-0.40 (-0.51, -0.30)
Obese				-0.61 (-0.71, -0.51)	-0.87 (-0.97, -0.76)
HbA1c				0.31 (0.28, 0.33)	0.24 (0.22, 0.26)
Hypertensive				0.03 (-0.04, 0.10)	-0.13 (-0.21, -0.05)
Cholesterol				-0.03 (-0.06, 0.01)	0.02 (-0.01, 0.05)
Creatinine > 300				0.34 (-0.09, 0.76)	0.46 (-0.01, 0.93)
eGFR				-0.03 (-0.03, -0.02)	-0.02 (-0.03, -0.02)
Ischaemic Cardiac				0.25 (0.18, 0.33)	0.18 (0.10, 0.27)
Stroke or TIA				0.23 (0.12, 0.33)	0.15 (0.04, 0.26)
PVD				0.65 (0.55, 0.76)	0.38 (0.27, 0.50)
<b>Interventions</b>					
Care level, reference group: <7					
Care level: 7					-0.07 (-0.17, 0.03)
Care level: 8					-0.04 (-0.15, 0.07)
Shared care					2.12 (2.04, 2.21)
M'brough PCT					-0.13 (-0.35, 0.08)
<b>Cons</b>	-1.81 (-2.04, -1.57)	-1.40 (-1.65, -1.15)	-2.44 (-2.83, -2.06)		-3.42 (-3.88, -2.97)
<b>Variance estimate at:</b>					
<b>Practice level</b>	0.15 (0.09, 0.25)	0.15 (0.09, 0.25)	0.12 (0.07, 0.20)		0.11 (0.06, 0.18)
<b>Patient level</b>	0.08 (0.02, 0.23)	0.05 (0.01, 0.16)	0.01 (0.00, 0.02)		0.00 (0.00, 0.01)
<b>Bayesian DIC</b>	26513.48	26310.16	22115.83		19561.96

Table 69: Stepwise logistic regression multilevel model examining no other blood glucose treatments with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		-0.05 (-0.34, 0.25)	-0.07 (-0.37, 0.22)	-0.10 (-0.42, 0.20)
High		0.21 (-0.05, 0.47)	0.25 (-0.02, 0.50)	0.22 (-0.06, 0.50)
Visit year, reference group: 1999				
2000		0.29 (0.07, 0.50)	0.32 (0.09, 0.54)	0.29 (0.06, 0.52)
2001		0.32 (0.11, 0.53)	0.49 (0.26, 0.71)	0.47 (0.24, 0.70)
2002		0.46 (0.27, 0.66)	0.65 (0.44, 0.86)	0.64 (0.44, 0.84)
2003		0.69 (0.51, 0.88)	0.81 (0.61, 1.01)	0.80 (0.61, 1.00)
2004		0.86 (0.68, 1.04)	0.97 (0.77, 1.16)	0.96 (0.76, 1.15)
2005		0.93 (0.76, 1.11)	1.03 (0.83, 1.23)	1.04 (0.84, 1.24)
2006		0.86 (0.68, 1.03)	1.04 (0.84, 1.23)	1.03 (0.84, 1.23)
2007		0.94 (0.75, 1.12)	1.05 (0.85, 1.24)	1.08 (0.88, 1.27)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		-0.01 (-0.40, 0.37)	0.12 (-0.27, 0.51)	0.16 (-0.24, 0.56)
Mid. SES*2001		0.2 (-0.16, 0.56)	0.32 (-0.06, 0.70)	0.36 (-0.02, 0.76)
Mid. SES*2002		0.01 (-0.34, 0.35)	0.09 (-0.27, 0.45)	0.12 (-0.23, 0.50)
Mid. SES*2003		-0.07 (-0.40, 0.25)	0.01 (-0.32, 0.35)	0.04 (-0.29, 0.39)
Mid. SES*2004		-0.11 (-0.44, 0.22)	-0.04 (-0.36, 0.30)	0.00 (-0.33, 0.35)
Mid. SES*2005		-0.09 (-0.42, 0.23)	-0.02 (-0.34, 0.32)	0.01 (-0.33, 0.36)
Mid. SES*2006		0.05 (-0.27, 0.36)	0.11 (-0.22, 0.44)	0.14 (-0.20, 0.48)
Mid. SES*2007		0.07 (-0.26, 0.39)	0.11 (-0.21, 0.45)	0.13 (-0.20, 0.48)
High SES*2000		-0.36 (-0.71, -0.01)	-0.27 (-0.62, 0.08)	-0.24 (-0.61, 0.13)
High SES*2001		-0.32 (-0.65, 0.02)	-0.28 (-0.63, 0.06)	-0.25 (-0.61, 0.11)
High SES*2002		-0.36 (-0.67, -0.05)	-0.33 (-0.65, 0.00)	-0.30 (-0.64, 0.03)
High SES*2003		-0.44 (-0.73, -0.13)	-0.41 (-0.70, -0.10)	-0.38 (-0.71, -0.06)
High SES*2004		-0.38 (-0.67, -0.09)	-0.32 (-0.60, -0.02)	-0.30 (-0.60, 0.02)
High SES*2005		-0.41 (-0.70, -0.12)	-0.35 (-0.63, -0.04)	-0.33 (-0.64, -0.01)
High SES*2006		-0.40 (-0.68, -0.11)	-0.38 (-0.66, -0.09)	-0.35 (-0.66, -0.04)
High SES*2007		-0.45 (-0.74, -0.15)	-0.38 (-0.67, -0.08)	-0.36 (-0.67, -0.04)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			-0.01 (-0.07, 0.05)	0.00 (-0.06, 0.06)
Age: 75+ years			-0.32 (-0.41, -0.24)	-0.31 (-0.39, -0.22)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 yrs			1.15 (1.09, 1.21)	1.14 (1.08, 1.20)
Duration 10+ yrs			1.45 (1.39, 1.52)	1.43 (1.36, 1.50)
Ethnicity, reference group: white				
South Asian			0.08 (-0.05, 0.20)	0.08 (-0.05, 0.21)
Other Ethnicity			-0.17 (-0.44, 0.11)	-0.18 (-0.45, 0.09)
Male			-0.18 (-0.23, -0.12)	-0.17 (-0.23, -0.12)
Smoking status, reference group: non-smoker				
Smoker			-0.06 (-0.14, 0.01)	-0.05 (-0.13, 0.02)
Ex-smoker			0.05 (-0.01, 0.11)	0.05 (-0.01, 0.10)
Obesity category, reference group: under and normal weight				
Overweight			0.33 (0.24, 0.41)	0.32 (0.24, 0.40)
Obese			0.73 (0.65, 0.81)	0.72 (0.65, 0.81)
HbA1c			0.27 (0.26, 0.29)	0.27 (0.25, 0.28)
Hypertensive			0.05 (0.00, 0.10)	0.04 (-0.01, 0.09)
Cholesterol			-0.18 (-0.20, -0.16)	-0.18 (-0.20, -0.15)
Creatinine > 300			-1.14 (-1.78, -0.56)	-1.15 (-1.79, -0.56)
eGFR			0.01 (0.01, 0.01)	0.01 (0.01, 0.01)
Ischaemic Cardiac			-0.11 (-0.17, -0.05)	-0.11 (-0.17, -0.06)
Stroke or TIA			-0.03 (-0.11, 0.05)	-0.04 (-0.12, 0.04)
PVD			-0.08 (-0.17, 0.01)	-0.09 (-0.18, 0.00)
<b>Interventions</b>				
Care level, reference group: <7				
Care level: 7				0.10 (0.03, 0.16)
Care level: 8				0.12 (0.05, 0.20)
Shared care				0.09 (0.03, 0.15)

M'brough PCT				
<b>Cons</b>	-0.49 (-0.65, -0.32)	-1.12 (-1.38, -0.84)	-4.71 (-5.01, -4.38)	-0.08 (-0.28, 0.10)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.08 (0.05, 0.13)	0.07 (0.04, 0.12)	0.08 (0.05, 0.13)	0.09 (0.05, 0.14)
<b>Patient level</b>	0.05 (0.01, 0.14)	0.08 (0.02, 0.24)	0.01 (0, 0.02)	0.01 (0, 0.02)
<b>Bayesian DIC</b>	45701.39	45279.41	40377.85	40361.07

## Blood pressure treatments

Table 70: Stepwise logistic regression multilevel model examining no blood pressure treatments with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
<b>Social-economic status, reference group: Low</b>				
Mid.		0.15 (-0.12, 0.42)	0.12 (-0.21, 0.43)	0.11 (-0.18, 0.42)
High		<b>0.37 (0.13, 0.62)</b>	<b>0.37 (0.09, 0.65)</b>	<b>0.35 (0.06, 0.63)</b>
Visit year, reference group: 1999				
2000		-0.23 (-0.45, -0.01)	-0.22 (-0.47, 0.03)	-0.21 (-0.45, 0.03)
2001		-0.33 (-0.54, -0.12)	-0.29 (-0.53, -0.05)	-0.27 (-0.50, -0.04)
2002		-0.45 (-0.64, -0.25)	-0.47 (-0.70, -0.24)	-0.45 (-0.66, -0.24)
2003		-0.64 (-0.83, -0.45)	-0.75 (-0.97, -0.53)	-0.73 (-0.94, -0.52)
2004		-0.73 (-0.92, -0.54)	-0.83 (-1.06, -0.62)	-0.81 (-1.01, -0.60)
2005		-0.93 (-1.12, -0.74)	-1.01 (-1.23, -0.79)	-0.99 (-1.20, -0.79)
2006		-0.89 (-1.07, -0.70)	-0.95 (-1.18, -0.74)	-0.92 (-1.12, -0.71)
2007		-1.05 (-1.24, -0.85)	-1.10 (-1.32, -0.88)	-1.11 (-1.33, -0.89)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		-0.11 (-0.49, 0.26)	-0.18 (-0.61, 0.26)	-0.17 (-0.59, 0.24)
Mid. SES*2001		-0.22 (-0.58, 0.13)	-0.19 (-0.61, 0.22)	-0.18 (-0.58, 0.21)
Mid. SES*2002		-0.27 (-0.60, 0.06)	-0.20 (-0.57, 0.19)	-0.18 (-0.55, 0.18)
Mid. SES*2003		-0.17 (-0.49, 0.15)	-0.07 (-0.44, 0.32)	-0.06 (-0.42, 0.30)
Mid. SES*2004		-0.12 (-0.43, 0.19)	0.02 (-0.34, 0.40)	0.02 (-0.33, 0.37)
Mid. SES*2005		-0.20 (-0.52, 0.12)	-0.10 (-0.47, 0.28)	-0.09 (-0.44, 0.26)
Mid. SES*2006		-0.25 (-0.57, 0.07)	-0.15 (-0.51, 0.23)	-0.14 (-0.48, 0.21)
Mid. SES*2007		-0.29 (-0.62, 0.04)	-0.19 (-0.55, 0.19)	-0.18 (-0.56, 0.18)
High SES*2000		0.04 (-0.30, 0.37)	-0.02 (-0.40, 0.36)	0.00 (-0.38, 0.38)
High SES*2001		-0.17 (-0.50, 0.14)	-0.21 (-0.57, 0.16)	-0.20 (-0.56, 0.17)
High SES*2002		-0.46 (-0.77, -0.16)	-0.44 (-0.78, -0.09)	-0.42 (-0.76, -0.07)
High SES*2003		-0.43 (-0.73, -0.14)	-0.41 (-0.74, -0.07)	-0.39 (-0.72, -0.06)
High SES*2004		-0.37 (-0.67, -0.09)	-0.30 (-0.63, 0.02)	-0.29 (-0.61, 0.05)
High SES*2005		-0.37 (-0.66, -0.07)	-0.29 (-0.62, 0.04)	-0.27 (-0.59, 0.06)
High SES*2006		-0.38 (-0.67, -0.10)	-0.32 (-0.64, 0.00)	-0.30 (-0.61, 0.03)
High SES*2007		-0.34 (-0.63, -0.04)	-0.27 (-0.60, 0.06)	-0.26 (-0.59, 0.08)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			-0.43 (-0.50, -0.36)	-0.43 (-0.50, -0.36)
Age: 75+ years			-0.42 (-0.52, -0.32)	-0.41 (-0.51, -0.31)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 yrs			-0.24 (-0.31, -0.18)	-0.24 (-0.31, -0.18)
Duration 10+ yrs			-0.21 (-0.29, -0.13)	-0.21 (-0.29, -0.13)
Ethnicity, reference group: white				
South Asian			<b>0.45 (0.32, 0.59)</b>	<b>0.44 (0.31, 0.58)</b>
Other Ethnicity			0.21 (-0.07, 0.51)	0.20 (-0.09, 0.49)
Male			0.01 (-0.05, 0.07)	0.01 (-0.05, 0.07)
Smoking status, reference group: non-smoker				
Smoker			<b>0.25 (0.17, 0.33)</b>	<b>0.25 (0.17, 0.33)</b>
Ex-smoker			-0.07 (-0.14, -0.01)	-0.07 (-0.14, 0.00)
Obesity category, reference group: under and normal weight				
Overweight			-0.37 (-0.46, -0.29)	-0.37 (-0.45, -0.29)
Obese			-0.87 (-0.95, -0.78)	-0.87 (-0.95, -0.78)
sBP			-0.02 (-0.02, -0.02)	-0.02 (-0.02, -0.02)
dBp			0.01 (0.00, 0.01)	0.01 (0.00, 0.01)
HbA1c			<b>0.07 (0.05, 0.08)</b>	<b>0.07 (0.05, 0.08)</b>
Cholesterol			<b>0.14 (0.12, 0.17)</b>	<b>0.14 (0.12, 0.17)</b>
eGFR			<b>0.02 (0.02, 0.02)</b>	<b>0.02 (0.02, 0.02)</b>
Ischaemic Cardiac			-1.66 (-1.75, -1.57)	-1.66 (-1.75, -1.58)
Stroke or TIA			-0.49 (-0.61, -0.37)	-0.49 (-0.61, -0.37)
PVD			-0.32 (-0.46, -0.19)	-0.32 (-0.46, -0.19)
<b>Interventions</b>				
Care level, reference group: <7				

Care level: 7				-0.03 (-0.10, 0.05)
Care level: 8				-0.14 (-0.22, -0.05)
M. PCT				0.08 (-0.12, 0.28)
Shared care				0.05 (-0.03, 0.12)
<b>Cons</b>	-1.12 (-1.24, -1.01)	-0.48 (-0.69, -0.28)	1.06 (0.59, 1.51)	1.05 (0.55, 1.49)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.09 (0.05, 0.16)	0.09 (0.05, 0.16)	0.09 (0.06, 0.16)	0.10 (0.06, 0.16)
<b>Patient level</b>	0.01 (0.00, 0.02)	0.02 (0.01, 0.06)	0.00 (0.00, 0.02)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	37784.65	37165.96	31026.01	31019.58
N = 34,231				

Table 71: Stepwise logistic regression multilevel model examining ACE inhibitors only with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
<b>Social-economic status, reference group: Low</b>				
Mid.		0.37 (-0.04, 0.81)	0.30 (-0.15, 0.75)	0.30 (-0.14, 0.74)
High		<b>0.39 (0.01, 0.77)</b>	0.33 (-0.10, 0.73)	0.33 (-0.07, 0.73)
Visit year, reference group: 1999				
2000		<b>0.35 (0.02, 0.68)</b>	<b>0.41 (0.06, 0.76)</b>	<b>0.40 (0.05, 0.75)</b>
2001		<b>0.37 (0.05, 0.69)</b>	<b>0.47 (0.13, 0.81)</b>	<b>0.46 (0.12, 0.80)</b>
2002		<b>0.36 (0.06, 0.66)</b>	<b>0.48 (0.16, 0.80)</b>	<b>0.48 (0.16, 0.79)</b>
2003		<b>0.37 (0.09, 0.67)</b>	<b>0.44 (0.14, 0.75)</b>	<b>0.44 (0.14, 0.75)</b>
2004		<b>0.31 (0.03, 0.60)</b>	<b>0.38 (0.07, 0.68)</b>	<b>0.37 (0.07, 0.67)</b>
2005		<b>0.40 (0.13, 0.70)</b>	<b>0.51 (0.20, 0.80)</b>	<b>0.50 (0.21, 0.81)</b>
2006		<b>0.46 (0.19, 0.75)</b>	<b>0.58 (0.27, 0.88)</b>	<b>0.56 (0.27, 0.87)</b>
2007		<b>0.47 (0.19, 0.76)</b>	<b>0.58 (0.27, 0.89)</b>	<b>0.60 (0.31, 0.91)</b>
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		0.00 (-0.56, 0.54)	0.03 (-0.54, 0.62)	0.04 (-0.53, 0.62)
Mid. SES*2001		-0.07 (-0.61, 0.44)	-0.03 (-0.58, 0.52)	-0.02 (-0.56, 0.51)
Mid. SES*2002		-0.21 (-0.73, 0.27)	-0.17 (-0.69, 0.36)	-0.17 (-0.68, 0.33)
Mid. SES*2003		-0.39 (-0.89, 0.09)	-0.31 (-0.83, 0.20)	-0.31 (-0.81, 0.19)
Mid. SES*2004		-0.38 (-0.87, 0.09)	-0.29 (-0.79, 0.22)	-0.28 (-0.77, 0.21)
Mid. SES*2005		-0.33 (-0.82, 0.14)	-0.25 (-0.74, 0.26)	-0.25 (-0.74, 0.24)
Mid. SES*2006		-0.31 (-0.80, 0.15)	-0.23 (-0.72, 0.27)	-0.23 (-0.71, 0.25)
Mid. SES*2007		-0.20 (-0.69, 0.27)	-0.13 (-0.62, 0.38)	-0.13 (-0.61, 0.35)
High SES*2000		-0.24 (-0.75, 0.27)	-0.26 (-0.78, 0.27)	-0.26 (-0.79, 0.28)
High SES*2001		-0.36 (-0.86, 0.12)	-0.36 (-0.88, 0.15)	-0.36 (-0.87, 0.15)
High SES*2002		-0.20 (-0.67, 0.25)	-0.2 (-0.67, 0.30)	-0.2 (-0.66, 0.28)
High SES*2003		-0.25 (-0.68, 0.19)	-0.21 (-0.66, 0.27)	-0.21 (-0.67, 0.26)
High SES*2004		-0.34 (-0.76, 0.09)	-0.27 (-0.71, 0.19)	-0.27 (-0.72, 0.19)
High SES*2005		-0.39 (-0.83, 0.03)	-0.34 (-0.78, 0.14)	-0.34 (-0.79, 0.12)
High SES*2006		-0.35 (-0.77, 0.07)	-0.31 (-0.75, 0.15)	-0.32 (-0.76, 0.13)
High SES*2007		-0.28 (-0.71, 0.15)	-0.23 (-0.67, 0.24)	-0.23 (-0.67, 0.23)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			-0.13 (-0.21, -0.04)	-0.13 (-0.21, -0.05)
Age: 75+ years			-0.37 (-0.49, -0.25)	-0.37 (-0.49, -0.25)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 yrs			<b>0.32 (0.24, 0.40)</b>	<b>0.32 (0.24, 0.40)</b>
Duration 10+ yrs			<b>0.43 (0.34, 0.52)</b>	<b>0.42 (0.33, 0.52)</b>
Ethnicity, reference group: white				
South Asian			-0.05 (-0.22, 0.12)	-0.05 (-0.22, 0.12)
Other Ethnicity			-1.01 (-1.55, -0.53)	-1.01 (-1.55, -0.53)
Male			<b>0.25 (0.18, 0.32)</b>	<b>0.25 (0.18, 0.33)</b>
Smoking status, reference group: non-smoker				
Smoker			0.00 (-0.10, 0.10)	0.01 (-0.10, 0.11)
Ex-smoker			0.05 (-0.03, 0.12)	0.04 (-0.03, 0.12)
Obesity category, reference group: under and normal weight				
Overweight			0.01 (-0.09, 0.12)	0.01 (-0.09, 0.11)
Obese			-0.10 (-0.20, 0.01)	-0.10 (-0.20, 0.00)
sBP			0.00 (0.00, 0.00)	0.00 (0.00, 0.00)

dBp			0.01 (0.01, 0.02)	0.01 (0.01, 0.02)
HbA1c			0.02 (-0.01, 0.04)	0.01 (-0.01, 0.04)
Cholesterol			-0.02 (-0.05, 0.01)	-0.02 (-0.05, 0.01)
eGFR			0.01 (0.00, 0.01)	0.01 (0.00, 0.01)
Ischaemic Cardiac Stroke or TIA			-0.93 (-1.02, -0.84)	-0.93 (-1.03, -0.84)
PVD			0.2 (0.09, 0.32)	0.2 (0.09, 0.32)
			0.29 (0.17, 0.41)	0.29 (0.16, 0.41)
<b>Interventions</b>				
Care level, reference group: <7				
Care level: 7				0.04 (-0.05, 0.13)
Care level: 8				0.10 (0.00, 0.20)
M'brough PCT				0.01 (-0.21, 0.25)
Shared care				0.01 (-0.07, 0.09)
<b>Cons</b>	-1.92 (-2.05, -1.78)	-2.35 (-2.64, -2.07)	-3.8 (-4.27, -3.3)	-3.81 (-4.33, -3.28)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.11 (0.06, 0.18)	0.11 (0.07, 0.18)	0.12 (0.07, 0.19)	0.12 (0.07, 0.20)
<b>Patient level</b>	0.01 (0.00, 0.04)	0.01 (0.00, 0.04)	0.01 (0.00, 0.02)	0.01 (0.00, 0.03)
<b>Bayesian DIC</b>	25520.15	25527.71	24579.91	24581.11

Table 72: Stepwise logistic regression multilevel model examining ACE inhibitors and other blood pressure treatments with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
<b>Social-economic status, reference group: Low</b>				
Mid.		0.00 (-0.34, 0.32)	-0.01 (-0.34, 0.34)	0.00 (-0.37, 0.35)
High		-0.32 (-0.68, 0.03)	-0.35 (-0.73, -0.02)	-0.36 (-0.72, 0.00)
Visit year, reference group: 1999				
2000		0.37 (0.10, 0.63)	0.30 (0.04, 0.57)	0.30 (0.03, 0.58)
2001		0.63 (0.38, 0.88)	0.57 (0.32, 0.82)	0.57 (0.30, 0.83)
2002		0.82 (0.58, 1.06)	0.77 (0.54, 1.00)	0.78 (0.52, 1.03)
2003		0.90 (0.67, 1.13)	0.88 (0.66, 1.10)	0.89 (0.65, 1.13)
2004		0.99 (0.76, 1.21)	0.95 (0.73, 1.16)	0.96 (0.71, 1.20)
2005		1.06 (0.83, 1.29)	0.98 (0.76, 1.20)	0.99 (0.75, 1.22)
2006		1.05 (0.83, 1.28)	0.97 (0.75, 1.20)	0.98 (0.73, 1.22)
2007		1.15 (0.92, 1.38)	1.04 (0.82, 1.26)	1.06 (0.82, 1.30)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		-0.05 (-0.48, 0.39)	0.04 (-0.42, 0.49)	0.02 (-0.44, 0.50)
Mid. SES*2001		0.01 (-0.39, 0.41)	0.01 (-0.41, 0.42)	0.00 (-0.42, 0.44)
Mid. SES*2002		0.01 (-0.36, 0.39)	-0.01 (-0.40, 0.37)	-0.03 (-0.42, 0.38)
Mid. SES*2003		0.10 (-0.25, 0.47)	0.11 (-0.28, 0.48)	0.09 (-0.29, 0.49)
Mid. SES*2004		-0.01 (-0.36, 0.36)	-0.05 (-0.42, 0.32)	-0.06 (-0.45, 0.34)
Mid. SES*2005		0.00 (-0.34, 0.37)	0.00 (-0.38, 0.36)	-0.01 (-0.40, 0.38)
Mid. SES*2006		0.02 (-0.31, 0.38)	0.00 (-0.37, 0.36)	-0.01 (-0.40, 0.38)
Mid. SES*2007		0.04 (-0.30, 0.41)	0.03 (-0.35, 0.39)	0.01 (-0.38, 0.41)
High SES*2000		0.11 (-0.34, 0.55)	0.21 (-0.23, 0.68)	0.22 (-0.23, 0.68)
High SES*2001		0.03 (-0.40, 0.45)	0.07 (-0.36, 0.50)	0.07 (-0.35, 0.50)
High SES*2002		0.18 (-0.22, 0.58)	0.17 (-0.22, 0.60)	0.18 (-0.21, 0.58)
High SES*2003		0.31 (-0.08, 0.69)	0.33 (-0.03, 0.74)	0.34 (-0.05, 0.72)
High SES*2004		0.36 (-0.02, 0.74)	0.37 (0.00, 0.77)	0.37 (-0.01, 0.76)
High SES*2005		0.25 (-0.13, 0.63)	0.26 (-0.11, 0.66)	0.26 (-0.12, 0.65)
High SES*2006		0.21 (-0.17, 0.58)	0.21 (-0.15, 0.61)	0.21 (-0.17, 0.59)
High SES*2007		0.19 (-0.19, 0.57)	0.21 (-0.15, 0.62)	0.22 (-0.16, 0.60)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.19 (0.13, 0.26)	0.19 (0.13, 0.26)
Age: 75+ years			0.01 (-0.08, 0.09)	0.01 (-0.07, 0.09)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 yrs			0.23 (0.18, 0.29)	0.23 (0.17, 0.29)
Duration 10+ yrs			0.40 (0.33, 0.46)	0.39 (0.33, 0.46)
Ethnicity, reference: white				
South Asian			-0.54 (-0.69, -0.39)	-0.53 (-0.68, -0.39)
Other Ethnicity			-0.10 (-0.42, 0.20)	-0.10 (-0.41, 0.19)

Male			0.27 (0.22, 0.32)	0.27 (0.22, 0.32)
Smoking status, reference: non-smoker				
Smoker			-0.12 (-0.19, -0.04)	-0.11 (-0.19, -0.04)
Ex-smoker			0.03 (-0.03, 0.08)	0.02 (-0.03, 0.08)
Obesity category, reference: under and normal weight				
Overweight			0.29 (0.21, 0.37)	0.29 (0.21, 0.37)
Obese			0.58 (0.51, 0.66)	0.58 (0.50, 0.66)
sBP			0.01 (0.01, 0.01)	0.01 (0.01, 0.01)
dBp			-0.01 (-0.01, -0.01)	-0.01 (-0.01, -0.01)
HbA1c			-0.03 (-0.04, -0.01)	-0.03 (-0.05, -0.01)
Cholesterol			-0.11 (-0.14, -0.09)	-0.11 (-0.14, -0.09)
eGFR			-0.01 (-0.02, -0.01)	-0.01 (-0.02, -0.01)
Ischaemic Cardiac			0.81 (0.76, 0.86)	0.81 (0.76, 0.86)
Stroke or TIA			0.16 (0.08, 0.24)	0.16 (0.08, 0.23)
PVD			0.03 (-0.06, 0.12)	0.02 (-0.07, 0.11)
<b>Interventions</b>				
Care level, reference group: <7				
Care level: 7				0.01 (-0.05, 0.08)
Care level: 8				0.04 (-0.03, 0.12)
M'brough PCT				-0.08 (-0.22, 0.07)
Shared care				0.03 (-0.03, 0.10)
<b>Cons</b>	-0.68 (-0.77, -0.58)	-1.55 (-1.78, -1.31)	-2.00 (-2.4, -1.58)	-1.96 (-2.39, -1.60)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.04 (0.02, 0.07)	0.04 (0.02, 0.07)	0.04 (0.02, 0.07)	0.04 (0.02, 0.08)
<b>Patient level</b>	0.01 (0.00, 0.02)	0.02 (0, 0.05)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)
<b>Bayesian DIC</b>	43407.29	42922.75	39650.30	39653.78

Table 73: Stepwise logistic regression multilevel model examining of blood pressure with no ACE inhibitors treatments with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
<b>Social-economic status, reference group: Low</b>				
Mid.		-0.27 (-0.57, 0.02)	-0.20 (-0.49, 0.09)	-0.23 (-0.52, 0.08)
High		-0.34 (-0.61, -0.05)	-0.26 (-0.54, 0.00)	-0.31 (-0.62, -0.01)
Visit year, reference group: 1999				
2000		-0.17 (-0.39, 0.05)	-0.20 (-0.42, 0.02)	-0.23 (-0.46, 0.00)
2001		-0.30 (-0.51, -0.08)	-0.38 (-0.60, -0.17)	-0.44 (-0.66, -0.21)
2002		-0.36 (-0.56, -0.16)	-0.44 (-0.64, -0.25)	-0.51 (-0.72, -0.30)
2003		-0.27 (-0.46, -0.07)	-0.33 (-0.51, -0.14)	-0.40 (-0.60, -0.19)
2004		-0.25 (-0.43, -0.05)	-0.30 (-0.49, -0.12)	-0.38 (-0.58, -0.18)
2005		-0.20 (-0.39, -0.02)	-0.27 (-0.45, -0.09)	-0.36 (-0.56, -0.15)
2006		-0.26 (-0.44, -0.07)	-0.33 (-0.52, -0.15)	-0.43 (-0.63, -0.23)
2007		-0.26 (-0.45, -0.07)	-0.33 (-0.52, -0.14)	-0.42 (-0.63, -0.21)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		0.13 (-0.25, 0.53)	0.10 (-0.30, 0.49)	0.11 (-0.29, 0.50)
Mid. SES*2001		0.21 (-0.16, 0.59)	0.15 (-0.22, 0.53)	0.16 (-0.23, 0.55)
Mid. SES*2002		0.31 (-0.04, 0.66)	0.24 (-0.12, 0.59)	0.25 (-0.10, 0.60)
Mid. SES*2003		0.18 (-0.15, 0.53)	0.09 (-0.25, 0.43)	0.11 (-0.24, 0.46)
Mid. SES*2004		0.26 (-0.07, 0.59)	0.15 (-0.18, 0.48)	0.18 (-0.16, 0.51)
Mid. SES*2005		0.27 (-0.05, 0.60)	0.18 (-0.16, 0.50)	0.20 (-0.14, 0.54)
Mid. SES*2006		0.28 (-0.03, 0.62)	0.19 (-0.14, 0.51)	0.22 (-0.12, 0.54)
Mid. SES*2007		0.20 (-0.13, 0.54)	0.10 (-0.23, 0.45)	0.14 (-0.20, 0.47)
High SES*2000		0.00 (-0.37, 0.37)	-0.02 (-0.39, 0.36)	0.02 (-0.37, 0.42)
High SES*2001		0.40 (0.05, 0.75)	0.39 (0.04, 0.75)	0.43 (0.05, 0.81)
High SES*2002		0.48 (0.15, 0.81)	0.43 (0.09, 0.76)	0.47 (0.12, 0.82)
High SES*2003		0.33 (0.02, 0.65)	0.27 (-0.04, 0.59)	0.32 (-0.02, 0.67)
High SES*2004		0.27 (-0.04, 0.59)	0.18 (-0.12, 0.49)	0.23 (-0.11, 0.57)
High SES*2005		0.41 (0.10, 0.71)	0.31 (0.01, 0.62)	0.37 (0.03, 0.71)
High SES*2006		0.45 (0.14, 0.76)	0.38 (0.08, 0.69)	0.43 (0.11, 0.76)
High SES*2007		0.38 (0.07, 0.69)	0.29 (-0.01, 0.60)	0.35 (0.01, 0.68)
<b>Covariates</b>				
Age, reference group: <60 years				

Age: 60-74 years			0.40 (0.33, 0.46)	0.39 (0.32, 0.45)
Age: 75+ years			0.61 (0.53, 0.69)	0.59 (0.50, 0.67)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.19 (-0.25, -0.13)	-0.18 (-0.24, -0.13)
Duration 10+ years			-0.47 (-0.54, -0.40)	-0.44 (-0.51, -0.37)
Ethnicity, reference: White				
South Asian			-0.04 (-0.17, 0.10)	-0.03 (-0.17, 0.10)
Other Ethnicity			0.21 (-0.08, 0.50)	0.24 (-0.06, 0.53)
Male			-0.40 (-0.46, -0.35)	-0.40 (-0.46, -0.35)
Smoking status, reference: non-smoker				
Smoker			-0.10 (-0.17, -0.02)	-0.10 (-0.18, -0.03)
Ex-smoker			0.01 (-0.04, 0.07)	0.01 (-0.04, 0.07)
Obesity category, reference: under and normal weight				
Overweight			0.03 (-0.05, 0.11)	0.03 (-0.05, 0.11)
Obese			0.17 (0.09, 0.24)	0.17 (0.10, 0.25)
sBP			0.00 (0.00, 0.01)	0.00 (0.00, 0.01)
dBp			0.00 (0.00, 0.00)	0.00 (0.00, 0.00)
HbA1c			-0.04 (-0.06, -0.03)	-0.04 (-0.06, -0.02)
Cholesterol			0.00 (-0.02, 0.02)	0.00 (-0.03, 0.02)
eGFR			0.00 (-0.01, 0.00)	0.00 (-0.01, 0.00)
Ischaemic Cardiac			0.48 (0.42, 0.53)	0.48 (0.43, 0.54)
Stroke or TIA			-0.05 (-0.13, 0.03)	-0.04 (-0.12, 0.04)
PVD			-0.08 (-0.17, 0.01)	-0.06 (-0.15, 0.03)
<b>Interventions</b>				
Care level, reference group: <7				
Care level: 7				0.00 (-0.06, 0.07)
Care level: 8				0.02 (-0.05, 0.10)
M. PCT				0.00 (-0.14, 0.15)
Shared care				-0.16 (-0.23, -0.10)
<b>Cons</b>	-0.94 (-1.03, -0.85)	-0.69 (-0.87, -0.51)	-0.70 (-1.06, -0.38)	-0.68 (-1.11, -0.28)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.05 (0.03, 0.09)	0.05 (0.03, 0.09)	0.05 (0.03, 0.09)	0.05 (0.03, 0.09)
<b>Patient level</b>	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)
<b>Bayesian DIC</b>	41236.49	41249.01	39766.32	39748.14

## Antithrombotic and Lipid profile treatments

Table 74: Saturated logistic regression multilevel model examining lipid therapies with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
<b>Social-economic status, reference group: Low</b>				
Mid.		-0.14 (-0.65, 0.38)	-0.10 (-0.66, 0.39)	-0.05 (-0.52, 0.41)
High		0.07 (-0.42, 0.46)	0.12 (-0.31, 0.49)	0.11 (-0.34, 0.52)
Visit year, reference group: 1999				
2000		0.20 (-0.14, 0.52)	0.15 (-0.19, 0.47)	0.16 (-0.17, 0.47)
2001		0.41 (0.09, 0.72)	0.39 (0.07, 0.69)	0.39 (0.07, 0.69)
2002		0.90 (0.59, 1.20)	0.90 (0.59, 1.18)	0.89 (0.59, 1.17)
2003		1.54 (1.24, 1.84)	1.58 (1.27, 1.86)	1.56 (1.27, 1.84)
2004		2.12 (1.82, 2.42)	2.13 (1.82, 2.41)	2.11 (1.81, 2.38)
2005		2.45 (2.14, 2.75)	2.42 (2.11, 2.70)	2.38 (2.08, 2.66)
2006		2.61 (2.30, 2.91)	2.60 (2.29, 2.88)	2.56 (2.27, 2.84)
2007		2.73 (2.42, 3.03)	2.71 (2.39, 3.00)	2.70 (2.39, 2.98)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		0.14 (-0.44, 0.73)	0.20 (-0.38, 0.81)	0.15 (-0.40, 0.70)
Mid. SES*2001		0.19 (-0.36, 0.77)	0.21 (-0.33, 0.81)	0.15 (-0.38, 0.67)
Mid. SES*2002		0.17 (-0.38, 0.70)	0.18 (-0.33, 0.76)	0.13 (-0.37, 0.63)
Mid. SES*2003		0.10 (-0.43, 0.65)	0.08 (-0.43, 0.66)	0.03 (-0.46, 0.52)
Mid. SES*2004		0.20 (-0.34, 0.74)	0.17 (-0.34, 0.74)	0.12 (-0.38, 0.61)

Mid. SES*2005		0.16 (-0.38, 0.70)	0.16 (-0.35, 0.74)	0.12 (-0.38, 0.61)
Mid. SES*2006		0.04 (-0.50, 0.57)	0.04 (-0.47, 0.62)	-0.01 (-0.50, 0.49)
Mid. SES*2007		0.17 (-0.37, 0.72)	0.16 (-0.36, 0.74)	0.11 (-0.40, 0.62)
High SES*2000		-0.13 (-0.60, 0.41)	-0.09 (-0.57, 0.42)	-0.08 (-0.58, 0.43)
High SES*2001		-0.19 (-0.64, 0.33)	-0.17 (-0.61, 0.32)	-0.16 (-0.62, 0.32)
High SES*2002		-0.15 (-0.57, 0.36)	-0.13 (-0.54, 0.34)	-0.12 (-0.56, 0.35)
High SES*2003		-0.28 (-0.69, 0.23)	-0.29 (-0.70, 0.17)	-0.28 (-0.71, 0.20)
High SES*2004		-0.08 (-0.49, 0.43)	-0.08 (-0.49, 0.37)	-0.08 (-0.51, 0.39)
High SES*2005		-0.16 (-0.58, 0.34)	-0.14 (-0.55, 0.30)	-0.13 (-0.57, 0.33)
High SES*2006		-0.12 (-0.53, 0.38)	-0.09 (-0.50, 0.37)	-0.08 (-0.51, 0.39)
High SES*2007		-0.21 (-0.63, 0.29)	-0.19 (-0.60, 0.26)	-0.19 (-0.63, 0.29)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.02 (-0.05, 0.08)	0.01 (-0.06, 0.08)
Age: 75+ years			-0.65 (-0.74, -0.57)	-0.67 (-0.75, -0.58)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 yrs			0.04 (-0.02, 0.10)	0.04 (-0.02, 0.10)
Duration 10+ yrs			-0.12 (-0.19, -0.05)	-0.10 (-0.17, -0.03)
Ethnicity, reference: White				
South Asian			-0.35 (-0.48, -0.22)	-0.35 (-0.49, -0.22)
Other Ethnicity			-0.71 (-0.99, -0.42)	-0.71 (-0.98, -0.42)
Male			-0.33 (-0.39, -0.28)	-0.33 (-0.39, -0.28)
Smoking status, reference: non-smoker				
Smoker			0.09 (0.01, 0.17)	0.09 (0.01, 0.17)
Ex-smoker			0.10 (0.04, 0.15)	0.10 (0.04, 0.15)
Obesity category, reference: under and normal weight				
Overweight			0.43 (0.35, 0.51)	0.43 (0.35, 0.51)
Obese			0.45 (0.37, 0.52)	0.45 (0.37, 0.53)
Hypertensive			-0.02 (-0.07, 0.03)	-0.01 (-0.07, 0.04)
HbA1c			0.05 (0.04, 0.07)	0.06 (0.04, 0.08)
Cholesterol			-0.28 (-0.30, -0.25)	-0.28 (-0.30, -0.25)
eGFR			0.00 (-0.01, 0.00)	-0.01 (-0.01, 0.00)
Ischaemic Cardiac			0.95 (0.89, 1.01)	0.95 (0.89, 1.01)
Stroke or TIA			0.23 (0.14, 0.32)	0.23 (0.15, 0.32)
PVD			0.16 (0.06, 0.26)	0.17 (0.07, 0.27)
<b>Interventions</b>				
Care level, reference group: <7				
Care level: 7				0.14 (0.07, 0.21)
Care level: 8				0.10 (0.03, 0.18)
M. PCT				0.17 (-0.03, 0.37)
Shared care				-0.10 (-0.16, -0.03)
Cons	0.51 (0.40, 0.62)	-1.17 (-1.49, -0.85)	-0.41 (-0.81, 0.01)	-0.41 (-0.98, 1.64)
<b>Variance estimate at:</b>				
Practice level	0.11 (0.07, 0.18)	0.09 (0.06, 0.16)	0.09 (0.06, 0.16)	0.09 (0.05, 0.15)
Patient level	0.00 (0.00, 0.01)	0.05 (0.01, 0.15)	0.03 (0.00, 0.1)	0.41 (0.00, 5.15)
Bayesian DIC	43995.34	39063.65	36576.41	36560.43

Table 75: Stepwise logistic regression multilevel model examining aspirin with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
<b>Social-economic status, reference group: Low</b>				
Mid.		-0.12 (-0.53, 0.31)	-0.07 (-0.51, 0.41)	-0.02 (-0.56, 0.45)
High		-0.28 (-0.65, 0.09)	-0.29 (-0.70, 0.08)	-0.19 (-0.67, 0.23)
Visit year, reference group: 1999				
2000		0.10 (-0.17, 0.39)	-0.02 (-0.32, 0.27)	0.06 (-0.29, 0.38)
2001		0.16 (-0.11, 0.44)	0.04 (-0.25, 0.34)	0.16 (-0.17, 0.48)
2002		0.16 (-0.09, 0.43)	0.00 (-0.28, 0.27)	0.14 (-0.18, 0.44)
2003		0.43 (0.18, 0.70)	0.27 (0.00, 0.53)	0.42 (0.11, 0.72)
2004		0.54 (0.30, 0.81)	0.37 (0.10, 0.64)	0.55 (0.23, 0.84)
2005		0.64 (0.39, 0.91)	0.45 (0.18, 0.71)	0.64 (0.32, 0.94)

2006		0.69 (0.44, 0.95)	0.52 (0.25, 0.79)	0.72 (0.40, 1.02)
2007		0.83 (0.59, 1.10)	0.67 (0.40, 0.94)	0.86 (0.53, 1.16)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		0.03 (-0.47, 0.51)	0.13 (-0.42, 0.65)	0.10 (-0.45, 0.70)
Mid. SES*2001		-0.07 (-0.56, 0.41)	-0.06 (-0.59, 0.45)	-0.08 (-0.62, 0.50)
Mid. SES*2002		0.17 (-0.30, 0.63)	0.18 (-0.34, 0.67)	0.14 (-0.38, 0.69)
Mid. SES*2003		0.07 (-0.40, 0.50)	0.03 (-0.47, 0.50)	-0.01 (-0.51, 0.54)
Mid. SES*2004		0.14 (-0.32, 0.57)	0.09 (-0.42, 0.55)	0.04 (-0.45, 0.59)
Mid. SES*2005		0.15 (-0.30, 0.59)	0.14 (-0.36, 0.60)	0.08 (-0.41, 0.63)
Mid. SES*2006		0.14 (-0.31, 0.58)	0.12 (-0.37, 0.58)	0.06 (-0.44, 0.61)
Mid. SES*2007		0.18 (-0.27, 0.62)	0.18 (-0.32, 0.64)	0.11 (-0.39, 0.66)
High SES*2000		-0.07 (-0.51, 0.36)	0.04 (-0.43, 0.54)	-0.06 (-0.56, 0.48)
High SES*2001		-0.21 (-0.63, 0.21)	-0.19 (-0.64, 0.28)	-0.28 (-0.75, 0.26)
High SES*2002		0.08 (-0.32, 0.48)	0.15 (-0.27, 0.59)	0.04 (-0.42, 0.55)
High SES*2003		0.07 (-0.33, 0.46)	0.13 (-0.28, 0.56)	0.02 (-0.43, 0.53)
High SES*2004		0.23 (-0.16, 0.61)	0.27 (-0.13, 0.71)	0.16 (-0.27, 0.66)
High SES*2005		0.19 (-0.20, 0.57)	0.24 (-0.16, 0.68)	0.12 (-0.31, 0.62)
High SES*2006		0.24 (-0.15, 0.62)	0.32 (-0.08, 0.75)	0.21 (-0.23, 0.71)
High SES*2007		0.12 (-0.27, 0.50)	0.19 (-0.21, 0.62)	0.08 (-0.36, 0.58)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.44 (0.38, 0.51)	0.47 (0.40, 0.53)
Age: 75+ years			0.41 (0.33, 0.50)	0.46 (0.37, 0.54)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.08 (0.02, 0.14)	0.07 (0.01, 0.13)
Duration 10+ years			0.26 (0.19, 0.32)	0.20 (0.14, 0.27)
Ethnicity, reference: White				
South Asian			0.03 (-0.10, 0.16)	0.03 (-0.11, 0.16)
Other Ethnicity			0.04 (-0.26, 0.32)	-0.01 (-0.31, 0.29)
Male			0.27 (0.21, 0.32)	0.27 (0.21, 0.32)
Smoking status, reference: non-smoker				
Smoker			0.21 (0.13, 0.28)	0.22 (0.14, 0.30)
Ex-smoker			0.10 (0.04, 0.15)	0.10 (0.04, 0.15)
Obesity category, reference: under and normal weight				
Overweight			0.22 (0.14, 0.30)	0.22 (0.14, 0.30)
Obese			0.33 (0.25, 0.41)	0.32 (0.24, 0.40)
Hypertensive			0.08 (0.03, 0.13)	0.06 (0.01, 0.11)
HbA1c			0.01 (-0.01, 0.02)	0.00 (-0.02, 0.01)
Cholesterol			-0.12 (-0.14, -0.09)	-0.11 (-0.13, -0.08)
eGFR			0.00 (0.00, 0.00)	0.00 (0.00, 0.00)
Ischaemic Cardiac			1.74 (1.68, 1.79)	1.73 (1.67, 1.79)
Stroke or TIA			0.87 (0.79, 0.96)	0.86 (0.78, 0.95)
PVD			0.37 (0.27, 0.46)	0.33 (0.23, 0.43)
<b>Interventions</b>				
Care level, reference group: <7				
Care level: 7				0.04 (-0.03, 0.10)
Care level: 8				-0.01 (-0.09, 0.07)
M. PCT				0.12 (-0.14, 0.38)
Shared care				0.29 (0.22, 0.36)
Cons	-0.21 (-0.32, -0.10)	-0.62 (-0.93, -0.33)	-1.49 (-1.88, -1.04)	-1.78 (-2.22, -1.24)
<b>Variance estimate at:</b>				
Practice level	0.08 (0.05, 0.14)	0.09 (0.06, 0.15)	0.16 (0.10, 0.26)	0.16 (0.10, 0.26)
Patient level	0.01 (0.00, 0.02)	0.03 (0.01, 0.09)	0.00 (0.00, 0.02)	0.00 (0.00, 0.01)
Bayesian DIC	45543.27	44955.91	37700.8	37629.34

## Shared care

Table 76: Stepwise logistic regression multilevel model examining shared care with interaction effect between SES and visit from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		0.30 (-0.18, 0.78)	0.25 (-0.28, 0.73)	0.25 (-0.27, 0.80)
High		-0.52 (-0.90, -0.13)	-0.66 (-1.05, -0.25)	-0.61 (-1.08, -0.17)
Visit year, reference group: 1999				
2000		-0.54 (-0.85, -0.22)	-0.57 (-0.89, -0.25)	-0.82 (-1.19, -0.46)
2001		-1.18 (-1.49, -0.88)	-1.19 (-1.49, -0.87)	-1.51 (-1.88, -1.17)
2002		-1.61 (-1.90, -1.31)	-1.68 (-1.97, -1.38)	-1.91 (-2.26, -1.59)
2003		-1.96 (-2.25, -1.66)	-2.07 (-2.36, -1.77)	-2.35 (-2.70, -2.03)
2004		-2.27 (-2.55, -1.98)	-2.47 (-2.76, -2.17)	-2.84 (-3.20, -2.52)
2005		-2.59 (-2.88, -2.30)	-2.84 (-3.14, -2.54)	-3.16 (-3.51, -2.84)
2006		-2.91 (-3.20, -2.62)	-3.00 (-3.29, -2.71)	-3.52 (-3.88, -3.19)
2007		-2.81 (-3.10, -2.51)	-3.01 (-3.32, -2.70)	-3.08 (-3.44, -2.75)
SES*Visit year, reference group: Low SES*1999				
Mid. SES*2000		-0.52 (-1.05, 0.03)	-0.44 (-1.00, 0.16)	-0.30 (-0.93, 0.33)
Mid. SES*2001		-0.60 (-1.13, -0.07)	-0.58 (-1.14, 0.00)	-0.51 (-1.13, 0.09)
Mid. SES*2002		-0.43 (-0.94, 0.09)	-0.32 (-0.85, 0.24)	-0.42 (-1.01, 0.15)
Mid. SES*2003		-0.43 (-0.94, 0.09)	-0.30 (-0.82, 0.27)	-0.33 (-0.93, 0.24)
Mid. SES*2004		-0.23 (-0.72, 0.27)	-0.09 (-0.62, 0.46)	-0.04 (-0.63, 0.53)
Mid. SES*2005		-0.05 (-0.56, 0.47)	0.12 (-0.41, 0.68)	0.14 (-0.45, 0.71)
Mid. SES*2006		0.02 (-0.49, 0.53)	0.15 (-0.38, 0.71)	0.15 (-0.43, 0.72)
Mid. SES*2007		-0.01 (-0.52, 0.51)	0.15 (-0.39, 0.71)	0.13 (-0.46, 0.72)
High SES*2000		0.31 (-0.16, 0.76)	0.51 (0.02, 0.98)	0.47 (-0.06, 1.01)
High SES*2001		0.33 (-0.12, 0.76)	0.45 (-0.02, 0.91)	0.44 (-0.06, 0.97)
High SES*2002		0.59 (0.16, 1.01)	0.83 (0.38, 1.26)	0.72 (0.24, 1.23)
High SES*2003		0.61 (0.18, 1.03)	0.84 (0.39, 1.28)	0.76 (0.29, 1.26)
High SES*2004		0.62 (0.20, 1.02)	0.86 (0.41, 1.29)	0.76 (0.28, 1.25)
High SES*2005		0.66 (0.22, 1.07)	0.93 (0.47, 1.36)	0.85 (0.37, 1.34)
High SES*2006		0.63 (0.20, 1.04)	0.85 (0.40, 1.28)	0.72 (0.24, 1.22)
High SES*2007		0.49 (0.05, 0.91)	0.76 (0.30, 1.21)	0.69 (0.20, 1.19)
<b>Covariates</b>				
Age, reference group: <60				
60-74			-0.55 (-0.62, -0.48)	-0.46 (-0.53, -0.38)
75+			-1.06 (-1.16, -0.96)	-0.85 (-0.96, -0.75)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.41 (0.34, 0.49)	0.01 (-0.07, 0.09)
Duration 10+ years			1.27 (1.19, 1.35)	0.54 (0.45, 0.63)
Ethnicity, reference group: white				
South Asian			-0.04 (-0.18, 0.10)	0.13 (-0.02, 0.28)
Other Ethnicity			0.76 (0.44, 1.07)	0.72 (0.39, 1.05)
Male			0.02 (-0.05, 0.08)	0.06 (0.00, 0.13)
Smoking status, reference group: non-smoker				
Smoker			-0.32 (-0.42, -0.24)	-0.32 (-0.42, -0.22)
Ex-smoker			-0.01 (-0.08, 0.06)	-0.04 (-0.12, 0.03)
Obesity status, reference group: Under and normal weight				
Overweight			0.03 (-0.07, 0.12)	0.08 (-0.02, 0.18)
Obese			0.28 (0.19, 0.38)	0.39 (0.28, 0.49)
HbA1c			0.26 (0.24, 0.28)	0.11 (0.09, 0.13)
Hypertensive			0.42 (0.36, 0.48)	0.47 (0.41, 0.54)
Cholesterol			-0.13 (-0.16, -0.1)	-0.08 (-0.11, -0.05)
Creatinine > 300			0.21 (-0.26, 0.66)	0.06 (-0.47, 0.58)
eGFR			-0.01 (-0.01, -0.01)	-0.01 (-0.01, 0.00)
Ischaemic Cardiac			0.25 (0.19, 0.32)	0.21 (0.13, 0.29)
Stroke or TIA			0.24 (0.15, 0.34)	0.17 (0.06, 0.27)
PVD			0.87 (0.77, 0.97)	0.7 (0.59, 0.81)
<b>Interventions</b>				

Care level, reference group: <7				
Care level: 7				0.60 (0.52, 0.69)
Care level: 8				1.25 (1.15, 1.35)
Diabetes treatment, reference group diet alone				
Sulphonylures / metformin only				0.75 (0.62, 0.88)
OHA comb.				0.78 (0.65, 0.92)
Insulin only				3.28 (3.14, 3.43)
Insulin & OHAs				1.59 (1.47, 1.71)
BP treatment, reference group no treatments				
ACE inhibitors only				-0.04 (-0.15, 0.07)
ACE & other(s)				-0.01 (-0.10, 0.08)
Other BP				-0.10 (-0.19, -0.01)
Aspirin				0.22 (0.15, 0.30)
Lipid therapy				-0.20 (-0.28, -0.13)
M. PCT				0.65 (0.17, 1.21)
<b>Cons</b>	-0.73 (-1.06, -0.37)	1.33 (0.92, 1.75)	-0.06 (-0.55, 0.41)	-1.19 (-1.77, -0.50)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.89 (0.54, 1.41)	0.94 (0.58, 1.52)	0.96 (0.59, 1.56)	1.00 (0.62, 1.62)
<b>Patient level</b>	0.14 (0.04, 0.39)	0.06 (0.02, 0.17)	0.02 (0.00, 0.06)	0.01 (0.00, 0.03)
<b>Bayesian DIC</b>	35835.41	33341.44	29475.68	25638.09

## Appendix H. Stepwise models for intermediate outcomes and long-term complications with interaction between interventions and socio-economic status

### Intermediate outcomes

Table 77: Stepwise linear regression multilevel models examining HbA1c by SES from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low SES				
Mid. SES		-0.11 (-0.15, -0.07)	-0.06 (-0.10, 0.02)	-0.05 (-0.08, 0.01)
High SES		-0.19 (-0.23, -0.15)	-0.10 (-0.14, 0.06)	-0.09 (-0.13, 0.06)
Visit year, reference group: 1999				
2000			-0.29 (-0.38, -0.20)	-0.32 (-0.40, -0.23)
2001			-0.47 (-0.56, -0.38)	-0.47 (-0.55, -0.38)
2002			-0.59 (-0.67, -0.50)	-0.55 (-0.62, -0.47)
2003			-0.63 (-0.71, -0.55)	-0.59 (-0.66, -0.52)
2004			-0.67 (-0.74, -0.59)	-0.63 (-0.71, -0.56)
2005			-0.75 (-0.83, -0.68)	-0.71 (-0.79, -0.64)
2006			-1.26 (-1.33, -1.18)	-1.18 (-1.25, -1.10)
2007			-1.14 (-1.22, -1.06)	-1.12 (-1.20, -1.04)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			-0.46 (-0.50, -0.42)	-0.33 (-0.36, -0.29)
Age: 75+ years			-0.69 (-0.73, -0.64)	-0.41 (-0.46, -0.37)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.38 (0.35, 0.42)	-0.09 (-0.13, -0.06)
Duration 10+ years			0.70 (0.66, 0.74)	0.06 (0.02, 0.09)
Ethnicity, reference group: White				
South Asian			0.47 (0.39, 0.55)	0.46 (0.39, 0.54)
Other Ethnicity			0.66 (0.48, 0.83)	0.47 (0.31, 0.64)
Male			-0.12 (-0.15, -0.09)	-0.06 (-0.09, -0.03)
Smoking status, reference group: Non smoker				
Smoker			0.25 (0.21, 0.30)	0.23 (0.19, 0.27)
Ex-smoker			0.07 (0.04, 0.11)	0.05 (0.02, 0.08)
BMI status, reference group: Low & normal weight				
Overweight			0.06 (0.01, 0.10)	0.02 (-0.02, 0.07)
Obese			0.19 (0.14, 0.24)	0.08 (0.04, 0.13)
Creatinine > 300			-0.84 (-1.09, -0.58)	-0.81 (-1.05, -0.56)
Hypertensive			0.14 (0.11, 0.17)	0.10 (0.07, 0.13)
Ischaemic Cardiac			0.05 (0.01, 0.08)	0.00 (-0.04, 0.03)
Stroke or TIA			-0.01 (-0.06, 0.04)	-0.06 (-0.1, -0.01)

PVD			0.09 (0.04, 0.15)	-0.06 (-0.12, -0.01)
<b>Interventions</b>				
Quality of Care level, reference group: Low quality				
Mid. quality				-0.13 (-0.16, -0.09)
High quality				-0.15 (-0.19, -0.11)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				0.81 (0.77, 0.85)
Combination with no insulin				1.25 (1.20, 1.29)
Insulin only				1.67 (1.61, 1.73)
Combination with insulin				1.75 (1.69, 1.82)
Shared care				0.17 (0.13, 0.21)
Middlesbrough PCT				0.10 (0.01, 0.20)
<b>Cons</b>	7.62 (7.49, 7.74)	7.84 (7.73, 7.95)	8.31 (8.20, 8.42)	7.60 (7.47, 7.72)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.05 (0.03, 0.08)	0.05 (0.03, 0.07)	0.03 (0.02, 0.05)	0.02 (0.01, 0.04)
<b>Patient level</b>	0.02 (0.01, 0.06)	0.01 (0.00, 0.04)	0.00 (0.00, 0.01)	0.00 (0.00, 0.02)
<b>Visit year</b>	2.47 (2.43, 2.50)	2.47 (2.43, 2.5)	2.2 (2.17, 2.23)	1.91 (1.88, 1.94)
<b>Bayesian DIC</b>	145158.08	145299.58	140844.98	133975.02
N = 38,413				

Table 78: Stepwise linear regression multilevel models examining cholesterol by SES from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Lowest SES				
Mid. SES		0.01 (-0.02, 0.04)	0.03 (0.00, 0.06)	0.03 (0.00, 0.06)
High SES		-0.03 (-0.06, 0.00)	0.00 (-0.03, 0.02)	-0.01 (-0.03, 0.02)
Visit year, reference group: 1999				
2000			-0.20 (-0.29, -0.11)	-0.20 (-0.29, -0.11)
2001			-0.21 (-0.30, -0.12)	-0.21 (-0.30, -0.12)
2002			-0.25 (-0.34, -0.17)	-0.22 (-0.30, -0.14)
2003			-0.44 (-0.53, -0.36)	-0.37 (-0.46, -0.29)
2004			-0.70 (-0.78, -0.62)	-0.58 (-0.66, -0.50)
2005			-0.86 (-0.94, -0.78)	-0.73 (-0.82, -0.65)
2006			-0.98 (-1.07, -0.90)	-0.83 (-0.92, -0.75)
2007			-1.05 (-1.13, -0.96)	-0.93 (-1.01, -0.85)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			-0.21 (-0.24, -0.18)	-0.20 (-0.23, -0.17)
Age: 75+ years			-0.23 (-0.26, -0.20)	-0.26 (-0.30, -0.23)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.11 (-0.13, -0.08)	-0.09 (-0.11, -0.06)

Duration 10+ years			-0.16 (-0.19, -0.13)	-0.13 (-0.16, -0.10)
Ethnicity, reference group: White				
South Asian			-0.07 (-0.12, -0.01)	-0.08 (-0.14, -0.02)
Other Ethnicity			0.11 (-0.02, 0.23)	0.08 (-0.05, 0.20)
Male			-0.33 (-0.35, -0.31)	-0.34 (-0.36, -0.32)
Smoking status, reference group: Non smoker				
Smoker			0.08 (0.05, 0.11)	0.08 (0.05, 0.11)
Ex-smoker			-0.02 (-0.04, 0.01)	0.00 (-0.03, 0.02)
BMI, reference group: Under or normal weight				
Overweight			0.00 (-0.03, 0.04)	0.03 (0.00, 0.07)
Obese			0.00 (-0.04, 0.03)	0.03 (0.00, 0.07)
Hypertensive			0.14 (0.12, 0.16)	0.14 (0.12, 0.16)
Ischaemic Cardiac			-0.21 (-0.24, -0.19)	-0.13 (-0.15, -0.10)
Stroke or TIA			-0.05 (-0.09, -0.01)	-0.02 (-0.06, 0.01)
PVD			-0.02 (-0.06, 0.03)	0.01 (-0.03, 0.05)
<b>Interventions</b>				
Quality of Care level, reference group: Low quality				
Mid. quality				-0.10 (-0.13, -0.07)
High quality				-0.15 (-0.18, -0.11)
Aspirin				-0.09 (-0.11, -0.06)
Lipid therapy				-0.28 (-0.31, -0.26)
Middlesbrough PCT				-0.03 (-0.09, 0.03)
Shared care				-0.07 (-0.10, -0.04)
<b>Cons</b>	4.65 (4.60, 4.70)	4.66 (4.6, 4.71)	5.73 (5.63, 5.84)	5.92 (5.81, 6.02)
<b>Variance estimates (Standard Error):</b>				
<b>Practice level</b>	0.01 (0.01, 0.02)	0.01 (0.01, 0.02)	0.01 (0.01, 0.02)	0.01 (0.00, 0.01)
<b>Patient level</b>	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)	0.00 (0.00, 0.01)
<b>Visit year</b>	1.35 (1.33, 1.37)	1.35 (1.33, 1.37)	1.17 (1.15, 1.18)	1.15 (1.13, 1.16)
<b>Bayesian DIC</b>	116373.88	116369.85	111026.13	110335.6

N = 37,085

## Long-term complications

Table 79: Stepwise linear regression multilevel models examining ischaemic cardiac disease socio-economic status from 2000 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		-0.09 (-0.22, 0.04)	-0.14 (-0.28, 0.00)	-0.18 (-0.33, -0.04)
High		-0.15 (-0.28, -0.03)	-0.18 (-0.30, -0.05)	-0.16 (-0.29, -0.02)
Visit year, reference group: 2000				
2001			-0.21 (-0.47, 0.06)	-0.29 (-0.59, 0.01)
2002			-0.13 (-0.37, 0.11)	-0.31 (-0.57, -0.04)
2003			-0.09 (-0.32, 0.14)	-0.40 (-0.66, -0.14)
2004			-0.25 (-0.48, -0.01)	-0.67 (-0.94, -0.40)
2005			-0.78 (-1.03, -0.54)	-1.25 (-1.53, -0.97)
2006			-1.25 (-1.50, -0.98)	-1.67 (-1.96, -1.38)
2007			-1.20 (-1.45, -0.93)	-1.81 (-2.11, -1.51)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.65 (0.51, 0.79)	0.34 (0.19, 0.49)
Age: 75+ years			0.83 (0.66, 1.00)	0.60 (0.42, 0.79)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.62 (-0.75, -0.50)	-0.59 (-0.73, -0.45)
Duration 10+ years			-0.56 (-0.70, -0.42)	-0.63 (-0.79, -0.46)
Ethnicity, reference group: White				
South Asian			-0.11 (-0.41, 0.18)	0.09 (-0.25, 0.41)
Other Ethnicity			-0.63 (-1.45, 0.08)	-0.67 (-1.56, 0.09)
Male			0.35 (0.24, 0.46)	0.32 (0.20, 0.44)
Smoking status, reference group: non smoker				
Smoker			0.16 (0.00, 0.32)	0.15 (-0.02, 0.33)
Ex-smoker			0.35 (0.23, 0.46)	0.31 (0.19, 0.44)
Obesity category, reference group: under & normal weight				
Overweight			0.1 (-0.06, 0.26)	-0.01 (-0.19, 0.16)
Obese			0.35 (0.19, 0.51)	0.12 (-0.05, 0.30)
HbA1c			0.06 (0.02, 0.09)	0.07 (0.03, 0.11)
Hypertensive			-0.19 (-0.30, -0.08)	-0.27 (-0.39, -0.15)
Cholesterol			-0.33 (-0.38, -0.28)	-0.23 (-0.29, -0.17)
eGFR			-0.02 (-0.02, -0.01)	-0.01 (-0.01, 0.00)
<b>Interventions</b>				
Quality of care level, reference group: Low quality				
Mid. quality				-0.30 (-0.44, -0.16)
High quality				-0.40 (-0.56, -0.24)

Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				-0.28 (-0.43, -0.14)
Combination, no insulin				-0.40 (-0.59, -0.21)
Insulin only				0.12 (-0.12, 0.36)
Combination with insulin				-0.31 (-0.60, -0.02)
Blood pressure treatment, reference group: No treatments				
ACE inhibitors only				0.27 (0.01, 0.53)
ACEI + other(s)				1.51 (1.31, 1.70)
Combination/other				1.25 (1.05, 1.45)
Aspirin				1.37 (1.26, 1.49)
Lipid therapy				0.61 (0.47, 0.74)
Middlesbrough PCT				-0.20 (-0.39, 0.00)
Shared care				0.27 (0.11, 0.43)
<b>Cons</b>	-3.98 (-5.22, -2.58)	-4.02 (-5.28, -2.72)	-1.87 (-2.86, -0.85)	-3.96 (-4.98, -3.00)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.04 (0.02, 0.09)	0.05 (0.02, 0.09)	0.03 (0.01, 0.07)	0.05 (0.02, 0.11)
<b>Patient level</b>	2.44 (0.59, 8.18)	2.48 (0.61, 8.09)	2.18 (0.59, 6.78)	2.05 (0.55, 6.71)
<b>Bayesian DIC</b>	11620.93	11617.33	10718.16	9194.77
N = 24,004				

Table 80: Stepwise linear regression multilevel models examining stroke or TIA socio-economic status from 2000 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status, reference group: Low</b>				
Mid.		0.01 (-0.18, 0.20)	-0.01 (-0.21, 0.18)	-0.01 (-0.21, 0.18)
High		-0.03 (-0.21, 0.15)	-0.07 (-0.25, 0.12)	-0.02 (-0.20, 0.17)
<b>Covariates</b>				
<b>Visit year, reference group: 2000</b>				
2001			0.10 (-0.29, 0.50)	0.21 (-0.17, 0.61)
2002			-0.02 (-0.38, 0.34)	0.04 (-0.31, 0.41)
2003			0.10 (-0.25, 0.45)	0.14 (-0.21, 0.51)
2004			-0.02 (-0.35, 0.34)	0.04 (-0.32, 0.39)
2005			-0.26 (-0.61, 0.11)	-0.19 (-0.56, 0.18)
2006			-0.86 (-1.24, -0.47)	-0.80 (-1.20, -0.41)
2007			-0.74 (-1.12, -0.34)	-0.71 (-1.11, -0.31)
Age, reference group: <60 years				
Age: 60-74 years			0.87 (0.64, 1.10)	0.75 (0.51, 0.99)
Age: 75+ years			1.22 (0.96, 1.49)	1.10 (0.83, 1.39)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.31 (-0.49, -0.13)	-0.30 (-0.50, -0.11)
Duration 10+ years			-0.04 (-0.22, 0.15)	-0.18 (-0.40, 0.02)
Ethnicity, reference group: White				
South Asian			0.04 (-0.39, 0.43)	0.11 (-0.34, 0.53)
Other Ethnicity			-1.18 (-3.09, 0.12)	-1.16 (-3.06, 0.17)
Male			0.01 (-0.15, 0.17)	-0.10 (-0.27, 0.06)
Smoking status, reference group: non smoker				
Smoker			0.31 (0.09, 0.53)	0.27 (0.04, 0.50)
Ex-smoker			0.21 (0.03, 0.38)	0.13 (-0.04, 0.31)
Obesity category, reference group: under & normal weight				
Overweight			-0.06 (-0.27, 0.16)	-0.09 (-0.30, 0.13)
Obese			-0.09 (-0.31, 0.13)	-0.20 (-0.42, 0.02)
HbA1c			0.03 (-0.02, 0.07)	0.02 (-0.03, 0.08)
Hypertensive			0.16 (0.00, 0.31)	0.15 (-0.01, 0.30)
Cholesterol			-0.11 (-0.18, -0.04)	-0.09 (-0.16, -0.01)
eGFR			-0.01 (-0.02, -0.01)	-0.01 (-0.01, 0.00)
<b>Interventions</b>				
Quality of care level, reference group: Low quality				
Mid. quality				-0.16 (-0.35, 0.03)
High quality				-0.03 (-0.24, 0.19)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				-0.18 (-0.38, 0.03)
Combo., no insulin				-0.30 (-0.56, -0.03)
Insulin only				-0.08 (-0.40, 0.23)
Combo., with insulin				-0.29 (-0.67, 0.09)

Blood pressure treatment, reference group: No treatments				0.08)
ACE inhibitors only				0.30 (0.01, 0.59)
ACE + other(s)				0.15 (-0.09, 0.39)
Combination/other				0.18 (-0.06, 0.42)
Aspirin				1.06 (0.89, 1.23)
Lipid therapy				0.09 (-0.08, 0.26)
M. PCT				-0.12 (-0.39, 0.14)
Shared care				0.44 (0.24, 0.65)
<b>Cons</b>	-4.63 (-5.38, -3.86)	-4.63 (-5.42, -3.84)	-4.24 (-5.33, -3.11)	-5.09 (-6.24, -3.99)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.10 (0.04, 0.21)	0.10 (0.04, 0.21)	0.07 (0.02, 0.16)	0.11 (0.04, 0.22)
<b>Patient level</b>	1.23 (0.31, 4.03)	1.23 (0.32, 3.96)	1.31 (0.33, 4.27)	1.32 (0.33, 4.37)
<b>Bayesian DIC</b>	6705.64	6709.29	6434.63	6133.11
N = 29,800				

Table 81: Stepwise linear regression multilevel models examining peripheral vascular disease socio-economic status from 2000 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		-0.16 (-0.37, 0.05)	-0.16 (-0.38, 0.06)	-0.17 (-0.40, 0.05)
High		-0.21 (-0.42, 0.00)	-0.20 (-0.42, 0.01)	-0.21 (-0.43, 0.01)
Visit year, reference group: 2000				
2001			-0.31 (-0.71, 0.10)	-0.22 (-0.63, 0.19)
2002			-0.34 (-0.70, 0.03)	-0.24 (-0.62, 0.14)
2003			-0.12 (-0.46, 0.23)	0.03 (-0.32, 0.40)
2004			-0.18 (-0.52, 0.16)	0.00 (-0.36, 0.36)
2005			-0.51 (-0.87, -0.16)	-0.30 (-0.68, 0.08)
2006			-0.98 (-1.35, -0.59)	-0.79 (-1.19, -0.38)
2007			-1.44 (-1.87, -1.01)	-1.13 (-1.59, -0.66)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.66 (0.43, 0.90)	0.62 (0.38, 0.88)
Age: 75+ years			0.72 (0.44, 1.00)	0.81 (0.51, 1.12)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.25 (0.04, 0.46)	0.13 (-0.09, 0.35)
Duration 10+ years			0.74 (0.53, 0.95)	0.38 (0.15, 0.62)
Ethnicity, reference group: White				
South Asian			-0.89 (-1.61, -0.25)	-0.81 (-1.53, -0.15)
Other Ethnicity			0.01 (-1.03, 0.88)	-0.06 (-1.14, 0.84)
Male			0.40 (0.22, 0.58)	0.40 (0.21, 0.59)
Smoking status, reference group: non smoker				
Smoker			0.90 (0.65, 1.14)	0.93 (0.68, 1.18)
Ex-smoker			0.42 (0.21, 0.63)	0.37 (0.16, 0.58)

Obesity category, reference group: under & normal weight				
Overweight			-0.12 (-0.36, 0.12)	-0.2 (-0.45, 0.05)
Obese			-0.06 (-0.29, 0.18)	-0.25 (-0.51, 0.00)
HbA1c			0.07 (0.01, 0.12)	0.01 (-0.05, 0.07)
Hypertensive			0.17 (0.00, 0.34)	0.13 (-0.05, 0.31)
Cholesterol			-0.13 (-0.21, -0.05)	-0.05 (-0.13, 0.03)
eGFR			-0.01 (-0.02, -0.01)	-0.01 (-0.01, 0.00)
<b>Interventions</b>				
Quality of care level, reference group: Low quality				
Mid. quality				-0.18 (-0.41, 0.06)
High quality				0.18 (-0.06, 0.42)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				-0.06 (-0.31, 0.20)
Combo., no insulin				-0.12 (-0.42, 0.19)
Insulin only				0.33 (0.01, 0.66)
Combo., with insulin				0.34 (-0.03, 0.72)
Blood pressure treatment, reference group: No treatments				
ACE inhibitors only				0.48 (0.15, 0.80)
Combination, with ACEI				0.42 (0.15, 0.70)
Combination, no ACEI				0.37 (0.09, 0.65)
Aspirin				0.58 (0.39, 0.76)
Lipid therapy				0.10 (-0.10, 0.29)
M. PCT				-0.18 (-0.55, 0.19)
Shared care				0.84 (0.63, 1.06)
<b>Cons</b>	-4.77 (-5.45, -4.14)	-4.66 (-5.51, -3.84)	-4.75 (-6.16, -3.57)	-5.92 (-7.33, -4.75)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.29 (0.15, 0.49)	0.29 (0.15, 0.51)	0.26 (0.13, 0.48)	0.27 (0.14, 0.48)
<b>Patient level</b>	0.88 (0.23, 2.63)	0.94 (0.24, 3.01)	1.17 (0.3, 3.84)	1.38 (0.33, 4.62)
<b>Bayesian DIC</b>	5658.78	5657.99	5315.63	5022.90
N = 30,053				

Table 82: Stepwise linear regression multilevel models examining microalbuminuria socio-economic status from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Lowest SES				
Mid. SES		-0.11 (-0.18, -0.03)	-0.11 (-0.19, -0.03)	-0.09 (-0.17, -0.01)
High SES		-0.13 (-0.20, -0.06)	-0.12 (-0.20, -0.05)	-0.09 (-0.17, -0.01)
Visit year, reference group: 1999				
2000			-0.33 (-0.66, -0.00)	-0.42 (-0.75, -0.09)
2001			-0.34 (-0.65, -0.03)	-0.63 (-0.94, -0.31)
2002			-0.44 (-0.74, -0.16)	-0.86 (-1.16, -0.55)
2003			-0.22 (-0.52, 0.07)	-0.75 (-1.05, -0.45)
2004			0.55 (0.26, 0.84)	-0.00 (-0.29, 0.31)
2005			0.79 (0.50, 1.07)	0.19 (-0.10, 0.48)

2006			1.13 (0.84, 1.41)	0.49)
2007			0.76 (0.35, 1.17)	0.56 (0.27, 0.87)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.13 (0.05, 0.20)	-0.01 (-0.06, 0.09)
Age: 75+ years			0.53 (0.44, 0.62)	0.37 (0.28, 0.47)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.01 (-0.08, 0.07)	-0.01 (-0.09, 0.06)
Duration 10+ years			0.08 (0.00, 0.16)	0.18 (0.09, 0.27)
Ethnicity, reference group: White				
South Asian			0.16 (-0.00, 0.32)	0.21 (0.05, 0.38)
Other Ethnicity			0.19 (-0.18, 0.54)	0.30 (-0.09, 0.67)
Male			0.23 (0.17, 0.30)	0.24 (0.18, 0.31)
Smoking status, reference group: Non smoker				
Smoker			0.28 (0.19, 0.37)	0.26 (0.17, 0.36)
Ex-smoker			0.08 (0.01, 0.15)	0.07 (-0.00, 0.14)
BMI, reference group: Under or normal weight				
Overweight			0.04 (-0.06, 0.13)	-0.01 (-0.11, 0.09)
Obese			0.12 (0.03, 0.21)	0.06 (-0.03, 0.15)
Hypertensive			0.14 (0.07, 0.20)	0.18 (0.11, 0.24)
Cholesterol			0.03 (-0.00, 0.05)	0.03 (0.00, 0.06)
HbA1c			0.06 (0.03, 0.07)	0.09 (0.06, 0.11)
<b>Interventions</b>				
Quality of Care level, reference group: Low quality				
Mid. quality				-0.11 (-0.54, 0.35)
High quality				-0.22 (-0.65, 0.23)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				0.14 (0.04, 0.24)
Combination, no insulin				0.02 (-0.09, 0.14)
Insulin only				0.18 (0.04, 0.32)
Combination with insulin				0.21 (0.11, 0.31)
Blood pressure treatment, reference group: No treatments				
ACE inhibitors only				0.37 (0.26, 0.47)
Combination with ACEI				0.53 (0.44, 0.62)
Combination, no ACEI				0.32 (0.23, 0.41)
Aspirin				0.07 (0.01, 0.14)
Lipid therapy				-0.05 (-0.12, 0.02)
Middlesbrough PCT				0.58 (0.28, 0.87)
Shared care				-0.93 (-1.02, -0.84)
<b>Cons</b>	-1.06 (-1.33, -0.84)	-0.99 (-1.24, -0.77)	-2.47 (-2.86, -2.05)	-2.45 (-3.04, -1.89)
<b>Variance estimates (Standard Error):</b>				
<b>Practice level</b>	0.23 (0.15, 0.37)	0.23 (0.14, 0.37)	0.24 (0.15, 0.38)	0.21 (0.13, 0.34)
<b>Patient level</b>	0.05 (0.01, 0.21)	0.05 (0.01, 0.19)	0.01 (0.00, 0.04)	0.01 (0.00, 0.03)
<b>Bayesian DIC</b>	27573.79	27,564.87	26,091.68	25467.71
N = 23,304				

Table 83: Stepwise linear regression multilevel models examining any retinopathy socio-economic status from 1999 to 2007, conditional on relevant explanatory variables

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid.		-0.09 (-0.19, 0.01)	-0.08 (-0.19, 0.02)	-0.08 (-0.19, 0.04)
High		-0.08 (-0.18, 0.01)	-0.07 (-0.18, 0.03)	-0.07 (-0.18, 0.03)
Visit year, reference group: 1999				
2000			-0.03 (-0.32, 0.26)	0.03 (-0.26, 0.33)
2001			-0.16 (-0.44, 0.13)	0.00 (-0.29, 0.30)
2002			-0.14 (-0.42, 0.14)	0.02 (-0.26, 0.31)
2003			-0.27 (-0.55, 0.01)	-0.09 (-0.38, 0.20)
2004			-0.17 (-0.45, 0.11)	0.09 (-0.20, 0.39)
2005			0.08 (-0.21, 0.38)	<b>0.36 (0.06, 0.66)</b>
2006			<b>-0.94 (-1.24, -0.65)</b>	<b>-0.67 (-0.97, -0.37)</b>
2007			0.08 (-0.20, 0.37)	<b>0.39 (0.08, 0.69)</b>
<b>Covariates</b>				
Age: 60-74 years				
Age: 75+ years			-0.06 (-0.16, 0.05)	0.01 (-0.11, 0.11)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			<b>0.64 (0.51, 0.76)</b>	<b>0.47 (0.34, 0.59)</b>
Duration 10+ years			<b>2.00 (1.87, 2.12)</b>	<b>1.60 (1.47, 1.72)</b>
Ethnicity, reference group: White				
South Asian			-0.23 (-0.46, 0.00)	-0.15 (-0.38, 0.08)
Other Ethnicity			<b>0.64 (0.21, 1.06)</b>	<b>0.52 (0.07, 0.94)</b>
Male			<b>0.25 (0.16, 0.34)</b>	<b>0.25 (0.16, 0.34)</b>
Smoking status, reference group: non smoker				
Smoker			-0.14 (-0.27, 0.00)	-0.13 (-0.27, 0.00)
Ex-smoker			-0.10 (-0.19, 0.00)	<b>-0.12 (-0.22, -0.02)</b>
Obesity status, reference group: under and normal weight				
Overweight			-0.06 (-0.19, 0.07)	-0.09 (-0.22, 0.05)
Obese			0.02 (-0.11, 0.15)	-0.10 (-0.24, 0.04)
HbA1c			<b>0.16 (0.13, 0.19)</b>	<b>0.05 (0.02, 0.08)</b>
Hypertensive			<b>0.38 (0.29, 0.47)</b>	<b>0.33 (0.24, 0.42)</b>
Cholesterol			<b>-0.05 (-0.09, -0.01)</b>	-0.01 (-0.05, 0.03)
eGFR			<b>-0.01 (-0.02, -0.01)</b>	<b>-0.01 (-0.01, -0.01)</b>
<b>Interventions</b>				
Quality of care level, reference group: Low and Medium quality				
High quality				-0.06 (-0.17, 0.04)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				<b>0.40 (0.24, 0.56)</b>
Combination, no insulin				<b>0.70 (0.53, 0.87)</b>
Insulin only				<b>1.04 (0.85, 1.23)</b>
Combination with insulin				<b>1.16 (0.96, 1.36)</b>
Blood pressure treatment, reference group: No treatments				
ACE inhibitors only				<b>0.32 (0.17, 0.46)</b>
Combination with ACEI				<b>0.22 (0.09, 0.34)</b>
Combination, no ACEI				0.13 (0.00, 0.26)
Aspirin				0.05 (-0.04, 0.14)
Lipid therapy				-0.06 (-0.16, 0.04)
Middlesbrough PCT				-0.04 (-0.21, 0.13)
Shared care				<b>0.53 (0.43, 0.64)</b>
<b>Cons</b>	<b>-1.19 (-1.50, -0.88)</b>	<b>-1.15 (-1.47, -0.81)</b>	<b>-2.55 (-3.05, -2.04)</b>	<b>-3.03 (-3.56, -2.49)</b>
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.07 (0.04, 0.12)	0.07 (0.03, 0.11)	0.06 (0.03, 0.10)	0.05 (0.03, 0.10)
<b>Patient level</b>	0.18 (0.05, 0.55)	0.18 (0.05, 0.58)	0.02 (0.00, 0.07)	0.00 (0.00, 0.02)
<b>Bayesian DIC</b>	17531.30	17532.21	15136.72	14525.43

N= 18,665

Table 84: Stepwise linear regression multilevel models examining microalbuminuria socio-economic status from 1999 to 2007, conditional on relevant explanatory variables (timeliness of diagnosis model)

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Lowest SES				
Mid. SES		-0.13 (-0.26, 0.01)	-0.14 (-0.28, 0.00)	-0.12 (-0.26, 0.02)
High SES		-0.11 (-0.23, 0.02)	-0.10 (-0.23, 0.03)	-0.07 (-0.21, 0.05)
Visit year, reference group: 1999				
2000			-1.33 (-2.43, -0.24)	-1.53 (-2.59, -0.49)
2001			-0.84 (-1.73, 0.11)	-1.53 (-2.37, -0.64)
2002			-0.84 (-1.68, 0.06)	-1.77 (-2.57, -0.92)
2003			-0.48 (-1.29, 0.41)	-1.58 (-2.36, -0.76)
2004			0.49 (-0.31, 1.37)	-0.65 (-1.41, 0.15)
2005			0.75 (-0.05, 1.63)	-0.46 (-1.22, 0.35)
2006			1.13 (0.33, 2.01)	-0.02 (-0.78, 0.79)
2007			0.58 (-0.33, 1.56)	-0.52 (-1.41, 0.41)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			0.01 (-0.12, 0.13)	-0.11 (-0.24, 0.02)
Age: 75+ years			0.26 (0.09, 0.43)	0.17 (-0.01, 0.34)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.00 (-0.12, 0.11)	-0.01 (-0.14, 0.11)
Ethnicity, reference group: White				
South Asian			0.25 (-0.06, 0.56)	0.22 (-0.11, 0.55)
Other Ethnicity			-0.06 (-0.80, 0.66)	0.19 (-0.58, 0.94)
Male			0.13 (0.03, 0.25)	0.18 (0.07, 0.30)
Smoking status, reference group: Non smoker				
Smoker			0.39 (0.24, 0.55)	0.37 (0.21, 0.53)
Ex-smoker			0.23 (0.11, 0.35)	0.22 (0.10, 0.34)
BMI, reference group: Under or normal weight				
Overweight			0.20 (0.03, 0.38)	0.13 (-0.04, 0.31)
Obese			0.30 (0.13, 0.47)	0.20 (0.02, 0.37)
eGFR			-0.01 (-0.01, 0.00)	-0.01 (-0.01, 0.00)
Hypertensive			0.11 (-0.01, 0.22)	0.17 (0.06, 0.29)
Cholesterol			0.02 (-0.03, 0.07)	0.03 (-0.02, 0.08)
<b>Interventions</b>				
HbA1c at diagnosis				0.04 (0.01, 0.07)
Quality of Care level, reference group: Low quality				
Mid. quality				0.32 (-0.53, 1.26)
High quality				0.09 (-0.76, 1.02)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				0.20 (0.06, 0.34)
Combination, no insulin				0.05 (-0.13, 0.24)
Insulin only				0.09 (-0.24, 0.42)
Combination with insulin				0.32 (0.15, 0.48)

Blood pressure treatment, reference group: No treatments

ACE inhibitors only				0.26 (0.08, 0.45)
Combination with ACEI				0.34 (0.19, 0.50)
Combination, no ACEI				0.14 (-0.01, 0.29)
Aspirin				-0.04 (-0.15, 0.08)
Lipid therapy				0.12 (0.00, 0.25)
Middlesbrough PCT				0.76 (0.37, 1.15)
Shared care				-1.26 (-1.45, -1.08)
<b>Cons</b>	-0.93 (-1.20, -0.66)	-0.88 (-1.16, -0.60)	-1.63 (-2.69, -0.72)	-1.57 (-2.74, -0.40)

**Variance estimates at:**

<b>Practice level</b>	0.46 (0.28, 0.75)	0.45 (0.27, 0.73)	0.46 (0.28, 0.76)	0.38 (0.23, 0.63)
<b>Patient level</b>	0.04 (0.00, 0.18)	0.04 (0.00, 0.17)	0.01 (0.00, 0.04)	0.01 (0.00, 0.04)
<b>Bayesian DIC</b>	9767.56	9767.53	9178.9	8917.21

N = 8,260

Table 85: Stepwise linear regression multilevel models examining any retinopathy socio-economic status from 1999 to 2007, conditional on relevant explanatory variables (timeliness of diagnosis model)

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low				
Mid		-0.10 (-0.32, 0.13)	-0.09 (-0.33, 0.14)	-0.11 (-0.35, 0.14)
High		0.08 (-0.13, 0.30)	0.04 (-0.19, 0.28)	0.08 (-0.16, 0.31)
Visit year, reference group: 1999				
2000			-0.64 (-1.89, 0.71)	-0.68 (-1.90, 0.68)
2001			-0.28 (-1.34, 0.93)	-0.27 (-1.28, 0.93)
2002			-0.20 (-1.12, 0.95)	-0.23 (-1.19, 0.94)
2003			-0.26 (-1.22, 0.91)	-0.25 (-1.18, 0.94)
2004			-0.10 (-1.04, 1.07)	-0.01 (-0.95, 1.17)
2005			0.30 (-0.66, 1.44)	0.38 (-0.56, 1.57)
2006			-1.18 (-2.14, -0.00)	-1.04 (-1.99, 0.12)
2007			0.45 (-0.48)	0.59 (-0.33, 1.77)
<b>Covariates</b>				
Age: 60-74 years				
Age: 75+ years			-0.01 (-0.24, 0.23)	0.07 (-0.18, 0.32)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.31 (-0.52, 0.73)	0.15 (-0.07, 0.38)
Ethnicity, reference group: White				
South Asian			0.13 (-0.52, 0.73)	0.10 (-0.56, 0.70)
Other Ethnicity			0.81 (-0.06, 1.59)	0.73 (-0.18, 1.54)
Male			0.17 (-0.03, 0.37)	0.15 (-0.05, 0.35)
Smoking status, reference group: non smoker				
Smoker			-0.07 (-0.38, 0.21)	-0.13 (-0.44, 0.17)
Ex-smoker			0.00 (-0.21, 0.21)	0.00 (-0.21, 0.21)
Obesity status, reference group: under and normal weight				
Overweight			-0.22 (-0.52, 0.07)	-0.25 (-0.54, 0.04)
Obese			-0.30 (-0.58, -0.02)	-0.40 (-0.68, -0.12)
Hypertensive			0.55 (0.36, 0.75)	0.48 (0.28, 0.68)
Cholesterol			-0.08 (-0.17, 0.01)	-0.05 (-0.15, 0.04)
eGFR			-0.01 (-0.02, -0.01)	-0.01 (-0.02, -0.00)
<b>Interventions</b>				
HbA1c at diagnosis				
Quality of care level, reference group: Low and Mid. quality				0.10 (0.05, 0.15)
High quality				-0.05 (-0.31, 0.21)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				0.34 (0.08, 0.61)
Combination, no insulin				0.57 (0.24, 0.89)
Insulin only				0.33 (-0.17, 0.79)
Combination with insulin				0.68 (0.19, 1.15)
Blood pressure treatment, reference group: No treatments				
ACE inhibitors only				0.48 (0.14, 0.81)
Combination with ACEI				0.46 (0.17, 0.75)
Combination, no ACEI				0.22 (-0.07, 0.51)
Aspirin				-0.00 (-0.20, 0.21)
Lipid therapy				-0.10 (-0.33, 0.12)
Middlesbrough PCT				-0.17 (-0.50, 0.16)
Shared care				0.28 (0.02, 0.55)
<b>Cons</b>	-2.32 (-2.65, -1.91)	-2.32 (-2.64, -1.89)	-1.35 (-2.75, -0.22)	-2.91 (-4.46, -1.60)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.12 (0.04, 0.25)	0.12 (0.04, 0.25)	0.13 (0.05, 0.27)	0.15 (0.05, 0.30)
<b>Patient level</b>	0.14 (0.02, 0.58)	0.15 (0.02, 0.63)	0.02 (0.00, 0.10)	0.01 (0.00, 0.07)
<b>Bayesian DIC</b>	3757.30	3,758.73		3512.47

N= 7,012

## Appendix I. Stepwise models for intermediate outcomes with interaction between visit year and socio-economic status prior to general practice level data being added to the model

Table 86: Stepwise linear regression multilevel models examining HbA1c by SES from 1999 to 2007, with interaction effect between SES and visit year conditional on relevant explanatory variables, prior to general practice level data being added to the model

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low SES				
Mid. SES		-0.14 (-0.24, -0.03)	-0.07 (-0.17, 0.03)	-0.07 (-0.16, 0.02)
High SES		-0.16 (-0.25, -0.07)	-0.06 (-0.15, 0.03)	-0.08 (-0.17, 0.01)
Visit year, reference group: 2004				
2005		-0.05 (-0.14, 0.02)	-0.05 (-0.13, 0.03)	-0.06 (-0.14, 0.01)
2006		-0.59 (-0.67, -0.51)	-0.57 (-0.65, -0.50)	-0.55 (-0.62, -0.49)
2007		-0.46 (-0.54, -0.38)	-0.46 (-0.54, -0.38)	-0.52 (-0.60, -0.45)
SES x Visit year, reference group: Low SES x 2004				
Mid SES x 2005		-0.01 (-0.16, 0.13)	-0.01 (-0.15, 0.13)	0.01 (-0.12, 0.14)
Mid SES x 2006		0.06 (-0.08, 0.20)	0.05 (-0.09, 0.18)	0.03 (-0.09, 0.16)
Mid SES x 2007		0.09 (-0.06, 0.23)	0.08 (-0.06, 0.21)	0.08 (-0.04, 0.21)
High SES x 2005		-0.08 (-0.21, 0.04)	-0.08 (-0.20, 0.04)	-0.06 (-0.18, 0.07)
High SES x 2006		-0.04 (-0.16, 0.09)	-0.05 (-0.17, 0.07)	-0.01 (-0.12, 0.10)
High SES x 2007		-0.08 (-0.21, 0.04)	-0.07 (-0.20, 0.05)	-0.02 (-0.14, 0.10)
<b>Covariates</b>				
Age, reference group: <60 years				
Age: 60-74 years			-0.48 (-0.52, -0.43)	-0.36 (-0.40, -0.31)
Age: 75+ years			-0.67 (-0.72, -0.61)	-0.4 (-0.46, -0.35)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			0.31 (0.27, 0.36)	0.03 (-0.01, 0.07)
Duration 10+ years			0.67 (0.62, 0.72)	0.04 (-0.01, 0.09)
Ethnicity, reference group: White				
South Asian			0.46 (0.36, 0.55)	0.46 (0.37, 0.55)
Other Ethnicity			0.47 (0.26, 0.68)	0.33 (0.14, 0.52)
Male			-0.05 (-0.09, -0.02)	-0.02 (-0.06, 0.02)
Smoking status, reference group: Non smoker				
Smoker			0.24 (0.18, 0.29)	0.22 (0.17, 0.28)
Ex-smoker			0.01 (-0.03, 0.06)	0.03 (-0.01, 0.07)
BMI status, reference group: Low & normal weight				
Overweight			0.02 (-0.04, 0.08)	-0.01 (-0.06, 0.05)
Obese			0.17 (0.11, 0.22)	0.07 (0.01, 0.12)

Creatinine > 300			-0.85 (-1.16, -0.54)	-0.78 (-1.08, -0.49)
Hypertensive			0.18 (0.14, 0.22)	0.12 (0.08, 0.16)
Ischaemic Cardiac			0.05 (0.01, 0.09)	0.01 (-0.03, 0.04)
Stroke or TIA			-0.03 (-0.09, 0.03)	-0.08 (-0.13, -0.02)
PVD			0.10 (0.03, 0.17)	-0.05 (-0.12, 0.01)
<b>Interventions, Patient level</b>				
Quality of Care level, reference group: Low quality				
Mid. quality				-0.16 (-0.21, -0.12)
High quality				-0.20 (-0.26, -0.15)
Diabetes treatment, reference group diet alone				
Metformin/sulphonylureas only				0.74 (0.69, 0.78)
Combination with no insulin				1.14 (1.08, 1.20)
Insulin only				1.64 (1.56, 1.72)
Combination with insulin				1.74 (1.66, 1.82)
Shared care				0.07 (0.02, 0.13)
Middlesbrough PCT				0.09 (-0.03, 0.20)
<b>Cons</b>	7.46 (7.37, 7.55)	7.84 (7.73, 7.95)	7.67 (7.55, 7.79)	7.13 (6.98, 7.29)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.05 (0.03, 0.08)	0.04 (0.02, 0.06)	0.03 (0.01, 0.04)	0.03 (0.02, 0.05)
<b>Patient level</b>	0.00 (0.00, 0.02)	0.01 (0.00, 0.02)	0.01 (0.00, 0.03)	0.01 (0.00, 0.04)
<b>Visit year</b>	2.14 (2.10, 2.18)	2.08 (2.04, 2.12)	1.92 (1.89, 1.96)	1.67 (1.64, 1.70)
<b>Bayesian DIC</b>	79447.87	78770.17	77070.99	73133.00

Table 87: Stepwise linear regression multilevel models examining cholesterol by SES from 1999 to 2007, with interaction effect between SES and visit year conditional on relevant explanatory variables, prior to general practice level data being added to the model

	Null	Model 1	Model 2	Model 3
<b>Social-economic status</b>				
Social-economic status, reference group: Low SES				
Mid. SES		-0.02 (-0.10, 0.06)	0.00 (-0.08, 0.07)	0.00 (-0.08, 0.07)
High SES		-0.06 (-0.14, 0.01)	-0.03 (-0.10, 0.04)	-0.03 (-0.10, 0.04)
Visit year, reference group: 2004				
2005		-0.18 (-0.24, -0.12)	-0.17 (-0.23, -0.11)	-0.16 (-0.22, -0.11)
2006		-0.34 (-0.40, -0.28)	-0.32 (-0.38, -0.27)	-0.29 (-0.34, -0.23)
2007		-0.38 (-0.44, -0.32)	-0.35 (-0.42, -0.29)	-0.38 (-0.44, -0.32)
SES x Visit year, reference group: Low SES x 2004				
Mid SES x 2005		-0.01 (-0.12, 0.10)	-0.01 (-0.12, 0.09)	-0.01 (-0.11, 0.10)
Mid SES x 2006		0.09 (-0.02, 0.19)	0.09 (-0.01, 0.19)	0.09 (0.00, 0.19)
Mid SES x 2007		0.03 (-0.08, 0.14)	0.04 (-0.06, 0.15)	0.05 (-0.05, 0.16)
High SES x 2005		0.03 (-0.08, 0.13)	0.02 (-0.08, 0.12)	0.02 (-0.08, 0.11)
High SES x 2006		0.07 (-0.03, 0.17)	0.07 (-0.02, 0.16)	0.08 (-0.01, 0.17)
High SES x 2007		0.03 (-0.07, 0.12)	0.02 (-0.08, 0.12)	0.02 (-0.08, 0.11)
<b>Covariates</b>				

Age, reference group: <60 years				
Age: 60-74 years			-0.23 (-0.27, -0.20)	-0.21 (-0.24, -0.17)
Age: 75+ years			-0.24 (-0.28, -0.19)	-0.26 (-0.30, -0.22)
Duration of diabetes, reference group: 0-3 years				
Duration: 4-9 years			-0.17 (-0.20, -0.14)	-0.14 (-0.17, -0.10)
Duration 10+ years			-0.25 (-0.29, -0.21)	-0.22 (-0.25, -0.18)
Ethnicity, reference group: White				
South Asian			-0.02 (-0.10, 0.05)	-0.05 (-0.12, 0.03)
Other Ethnicity			0.19 (0.04, 0.35)	0.14 (-0.01, 0.29)
Male			-0.30 (-0.33, -0.27)	-0.31 (-0.34, -0.28)
Smoking status, reference group: Non smoker				
Smoker			0.11 (0.07, 0.16)	0.11 (0.06, 0.15)
Ex-smoker			0.00 (-0.03, 0.03)	0.01 (-0.03, 0.04)
BMI status, reference group: Low & normal weight				
Overweight			-0.02 (-0.07, 0.03)	0.02 (-0.02, 0.07)
Obese			-0.04 (-0.08, 0.01)	0.01 (-0.03, 0.06)
Hypertensive			0.15 (0.12, 0.18)	0.15 (0.12, 0.18)
Ischaemic Cardiac			-0.20 (-0.23, -0.16)	-0.12 (-0.15, -0.09)
Stroke or TIA			-0.05 (-0.09, 0.00)	-0.03 (-0.07, 0.02)
PVD			-0.02 (-0.07, 0.03)	0.00 (-0.05, 0.05)
<b>Interventions, Patient level</b>				
Quality of Care level, reference group: Low quality				
Mid. quality				-0.14 (-0.18, -0.10)
High quality				-0.23 (-0.27, -0.18)
Aspirin				-0.08 (-0.11, -0.05)
Lipid therapies				-0.37 (-0.40, -0.34)
Shared care				-0.06 (-0.10, -0.03)
Middlesbrough PCT				-0.03 (-0.12, 0.05)
<b>Cons</b>	4.38 (4.24, 4.51)	4.62 (4.42, 4.77)	5.1 (4.98, 5.22)	5.49 (5.37, 5.61)
<b>Variance estimate at:</b>				
<b>Practice level</b>	0.02 (0.01, 0.03)	0.02 (0.01, 0.03)	0.02 (0.01, 0.03)	0.02 (0.01, 0.03)
<b>Patient level</b>	0.02 (0, 0.1)	0.03 (0.24, 0.16)	0.01 (0, 0.05)	0.01 (0.00, 0.03)
<b>Visit year</b>	1.24 (1.22, 1.27)	1.22 (0.20, 1.25)	1.15 (1.13, 1.17)	1.11 (1.09, 1.14)
<b>Bayesian DIC</b>	67652.43	67343.07	65945.30	65294.04

## Appendix J: Representativeness of South Tees Hospitals NHS Trust Diabetes Register of type 2 diabetes patients in the South Tees area

Table 88 compares the prevalence of type 2 diabetes patients per practice as identified through the South Tees Hospitals NHS Trust Diabetes Register with Quality and Outcome Framework (QOF) diabetes prevalence per year. Both prevalence indicators use the practice list sizes per practice from the QOF as the denominator to allow comparison between indicators. Comparing these indicators with other figures should be cautious as the denominator counts patients of all ages whereas the Diabetes Register and QOF prevalence numerator [47] have patients 17 and above only.

The third column calculates the proportion of diabetes prevalence of QOF prevalence. England and worldwide estimates indicate that type 2 diabetes make up between 90-95% of all diabetes patients [41]. Those proportions which fall into this range are highlighted in bold indicating, arguably, the data are representative of type 2 diabetes for these practices for that year. There are a number of practices which capture more than 95% of the expected number of type 2 diabetes patients. This maybe because there a number of diabetes patients in the South Tees area known to secondary care but in primary care [50]. There are also a number of practices which notably fewer type 2 diabetes patients than what is expected. This maybe because be due to a drop off in data being collected from primary care. This could also explain the sharp drop off in the number of type 2 diabetes from some practices which can be seen in Figure one.

Table 88: Prevalence of type 2 diabetes in South Tees Hospitals NHS Trust Diabetes Register, Prevalence of type 1 and type 2 diabetes in Quality and Outcomes Framework and Proportion of South Tees Hospitals NHS Trust Diabetes Register of Quality and Outcomes Framework prevalence

	2004			2005			2006			2007		
Practice	S. Tees Register Prevalence	QOF Prevalence	Proportion of S. Tees of QOF prevalence	S. Tees Register Prevalence	QOF Prevalence	Proportion of S. Tees of QOF prevalence	S. Tees Register Prevalence	QOF Prevalence	Proportion of S. Tees of QOF prevalence	S. Tees Register Prevalence	QOF Prevalence	Proportion of S. Tees of QOF prevalence
1	3.62	4.13	<b>87.80</b>	4.08	4.36	<b>93.40</b>	4.08	4.38	<b>93.23</b>	4.39	4.48	97.95
2							3.48	3.63	95.96	3.78	3.87	97.78
3	3.00	3.47	<b>86.63</b>	3.43	3.82	<b>89.72</b>	3.77	3.69	102.05	1.36	4.08	33.33
4	2.29	2.67	<b>85.94</b>	2.38	2.78	<b>85.45</b>	2.64	2.80	<b>94.05</b>	1.26	2.95	42.71
5	3.52	3.72	<b>94.76</b>	3.82	4.01	95.08	3.89	4.20	<b>92.56</b>	3.82	4.13	<b>92.34</b>
6	2.68	3.09	<b>86.75</b>				3.64	4.02	<b>90.50</b>	3.86	4.17	<b>92.64</b>
7	3.33	3.63	<b>91.74</b>	3.63	3.76	96.75	3.56	3.84	<b>92.74</b>	3.82	3.79	100.83
8	3.12	3.22	96.80	3.12	3.43	<b>90.85</b>	3.13	3.31	<b>94.63</b>	3.29	3.53	<b>93.23</b>
9	3.41	3.74	<b>91.12</b>	3.92	4.24	<b>92.47</b>	4.32	4.61	<b>93.70</b>	4.50	4.89	<b>92.13</b>
10	3.09	3.21	96.44	3.25	3.50	<b>93.07</b>	3.45	3.43	100.57	3.35	3.43	97.73
11	3.58	3.61	99.31	3.48	3.58	97.19	3.75	3.52	106.45	3.71	3.72	99.66
12	3.74	3.39	110.37	3.88	3.56	109.21	4.21	3.64	115.59	4.38	3.83	114.19
13	2.71	2.93	<b>92.24</b>	3.02	3.33	<b>90.76</b>	3.36	3.56	<b>94.19</b>	3.32	3.74	<b>88.92</b>
14	0.82	2.75	29.69				0.97	3.12	31.22	0.79	3.25	24.24
15	2.83	2.97	95.44	2.98	3.22	<b>92.47</b>	3.18	3.09	103.14	3.30	3.44	95.91
16	3.13	3.46	<b>90.43</b>	3.31	3.56	<b>92.89</b>	3.42	3.68	<b>92.97</b>	3.77	4.01	<b>93.97</b>
17	2.52	2.72	<b>92.63</b>	2.76	2.95	<b>93.49</b>	2.95	2.93	100.90	3.09	3.41	<b>90.74</b>
18	2.55	2.95	<b>86.44</b>				2.79	3.15	<b>88.69</b>	3.09	3.46	<b>89.34</b>
19				3.27	3.32	98.47	3.43	3.48	98.59	3.66	3.76	97.39
20	2.77	2.96	<b>93.77</b>	2.93	3.11	<b>93.97</b>	3.15	3.14	100.57	3.18	3.55	<b>89.39</b>
21	3.12	3.47	<b>89.97</b>	3.39	3.63	<b>93.25</b>	3.68	3.84	96.02	3.88	4.04	96.19
	2004			2005			2006			2007		

Practice	S. Tees Register Prevalence	QOF Prevalence	Proportion of S. Tees of QOF prevalence	S. Tees Register Prevalence	QOF Prevalence	Proportion of S. Tees of QOF prevalence	S. Tees Register Prevalence	QOF Prevalence	Proportion of S. Tees of QOF prevalence	S. Tees Register Prevalence	QOF Prevalence	Proportion of S. Tees of QOF prevalence
22				3.93	4.46	<b>88.10</b>	4.35	4.57	95.32	4.53	4.86	<b>93.26</b>
23	2.87	3.32	<b>86.57</b>	3.20	3.51	<b>91.20</b>	3.23	3.62	<b>89.33</b>	3.84	4.16	<b>92.31</b>
24	2.59	2.88	<b>89.91</b>	3.05	3.36	<b>90.64</b>	3.29	3.59	<b>91.67</b>	1.65	3.63	45.55
25				3.78	4.07	<b>92.83</b>	3.64	4.17	<b>87.30</b>	4.07	4.37	<b>92.97</b>
26	3.61	3.94	<b>91.54</b>	3.62	4.06	<b>89.31</b>	4.03	4.31	<b>93.48</b>	4.20	4.63	<b>90.82</b>
27	4.22	4.53	<b>93.19</b>	4.41	4.66	<b>94.65</b>	4.34	4.67	<b>93.03</b>	4.93	5.15	95.59
28	3.67	3.74	98.01	3.88	3.90	99.52	4.23	4.36	97.00	2.15	4.49	47.90
29	2.81	3.25	<b>86.29</b>	3.05	3.44	<b>88.80</b>	3.32	3.58	<b>92.94</b>	3.45	3.80	<b>90.85</b>
30	3.42	3.63	<b>94.24</b>	3.56	3.88	<b>91.69</b>	4.01	4.30	<b>93.41</b>	4.15	4.34	95.61
31	4.21	4.39	95.95	4.61	4.69	98.26	4.89	4.97	98.35	5.07	4.82	105.14
32	3.03	3.28	<b>92.28</b>	3.11	3.42	<b>90.78</b>	3.30	3.53	<b>93.58</b>	3.44	3.71	<b>92.58</b>
33	1.86	2.23	83.54	2.18	2.47	<b>88.14</b>	2.35	2.60	<b>90.58</b>	2.58	2.84	<b>90.57</b>
34	2.41	2.79	<b>86.23</b>	2.40	2.71	<b>88.69</b>	2.59	2.80	<b>92.61</b>	2.62	2.86	<b>91.40</b>
35	2.66	3.03	<b>87.76</b>	2.92	3.34	<b>87.50</b>	2.96	3.79	77.97	3.84	4.00	96.03
36							4.13	4.08	101.23	1.44	4.18	34.57
37	3.21	3.11	103.13				3.16	3.49	<b>90.54</b>	3.43	3.53	97.37
38				3.98	4.23	<b>94.19</b>	2.78	2.85	97.58	3.00	3.24	<b>92.47</b>
39	3.35	3.66	<b>91.52</b>	3.73	3.90	95.47	3.75	3.98	<b>94.12</b>	3.53	3.92	<b>89.96</b>
40				1.86	1.95	95.56	1.78	2.09	<b>85.42</b>	2.12	2.39	<b>89.09</b>
41							0.09	0.52	16.67	0.09	0.51	16.67
42				1.43	1.00	142.86	1.10	1.24	<b>88.89</b>	0.81	1.30	62.50
43	3.03	3.27	<b>92.93</b>				3.67	3.92	<b>93.44</b>	3.76	3.89	96.67

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