An economic analysis of the contribution of health care to health inequality

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Declaration of Authorship

I, Ed Kendall. confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

Signed: _____

Date: _____

Abstract

Reducing socioeconomic-related health inequalities has been a pressing concern for many years, and the focus of much academic research. Until now, however, there have been few efforts to quantify the contribution that health care makes to health inequality. Our contention is that if health care increases health, the distribution of health care affects health inequality, therefore changes in the distribution of health care can be used to reduced health inequalities. We begin by reviewing the literature on the effects of health care on health and health inequality. Next, we show how a cross-sectional model of the effect of health care on health can be used to examine the contribution of health care to health inequality. Through this we find that for the area-level effect of spending on mortality in England, cancer spending leads to a significant reduction (approximately 50%) in cancer mortality inequalities but Coronary Heart Disease (CHD) spending does not. We move on to consider the limitations of modelling health inequality over time when using area-level data and suggest a methodological innovation, using a country-level analysis to demonstrate its application. Returning to the setting of English cancer and CHD mortality we show that the results of an analogous panel data model support our earlier findings. Finally, we use individual-level data to examine the effect that health spending has on health care utilisation, the effect utilisation has on health, and how these effects contribute to health inequality. We find that here is no effect on health from General Practitioner visits. Inpatient days do increase health and, overall, reduce health inequalities by 40%. These findings show the inherent difficulty of measuring the effect of health care on health, but do suggest that health care is currently making substantial contributions to the reduction of health inequalities.

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Chapter 1

Introduction

This thesis will explore the way in which health care in England makes a difference to the distribution of health between rich and poor. Since Arrow's seminal paper in 1963 [4], it has been established in the health economics literature that society's preference over health care has a distributional dimension, that health care is not a purely individualistic endeavour, and that the rich should to some extent subsidise the poor or the healthy subsidise the ill. In England, the National Health Service (NHS), funded by general taxation, embodies this notion. Health care is organised on a regional basis, free to all, and funds treatments up to a pre-determined cost-effectiveness threshold. In principle, then, one might expect there to be no overall effect on health inequality of such a system, as such universal health care ought to improve the health of everyone equally. In practice, a range of factors mean this is not so.

1.1 Definitions

To begin with we define some terms of reference.

Health care is the service provided by qualified medical personnel to improve an individual's health or to minimise a reduction in health. It includes consultations, operations,

drugs, help with changing behavioural habits and therapy, provided in GP surgeries, clinics, hospitals and pharmacies. A number of related concepts will be used in this thesis. Health care utilisation is the consumption of health care by an individual. Health care spending is the money required to provide health care, for a given locality, disease area or care setting.

In the United Kingdom (UK), public sector health expenditure accounted for 87% of overall health expenditure in 2007/08 [34]. In our analysis we focus on National Health Service (NHS) provided health care. This is justified for the following reasons: the ways in which health care contributes to health inequality are different in the private sector from the public sector; data is not publicly available for the private sector; and the NHS accounts for the overwhelming majority of health care. It should also be noted that this thesis will focus on the types of health care that have more easily and widely measured effects, and are therefore amenable to economic analysis. For instance, the effect of cancer treatments in most cases of cancers are recorded in offical mortality statistics, but no such outcome is recorded for any of the benefits mental health services provide mental health patients. The datasets used cover individual, area and country levels.

Health inequality is the structural variation in health across a population. In this thesis the focus will be on socioeconomic-related health inequality. This is the systematic difference in health by subgroups of differing socioeconomic status.

Socioeconomic status is taken to be a measure of a person's combined social and economic position relative to others. Factors that determine socioeconomic status are: income, education, occupation. As these are also highly correlated they can be used to measure socioeconomic status. For the purposes of this thesis we consider socioeconomic status to be separate from health status, though we recognise that some conceptualisations of socioeconomic status include health as a determining factor.

Health care inequality is the structural variation in the quantity or quality of health care

across a population. When unequal health care is regarded as unfair, it is known as health care inequity. Health care inequity is divided into two distinct concepts. Horizontal inequity is defined as being when equal needs do not lead to equal treatment. Vertical inequity is when unequal needs do not lead to appropriately unequal treatment. Research has focused far more on horizontal inequity, which is simpler to measure and requires no value judgements regarding the appropriate level of care for given needs. Provided health care is effective in aiding health, health care inequality describes how health care contributes to health inequality.

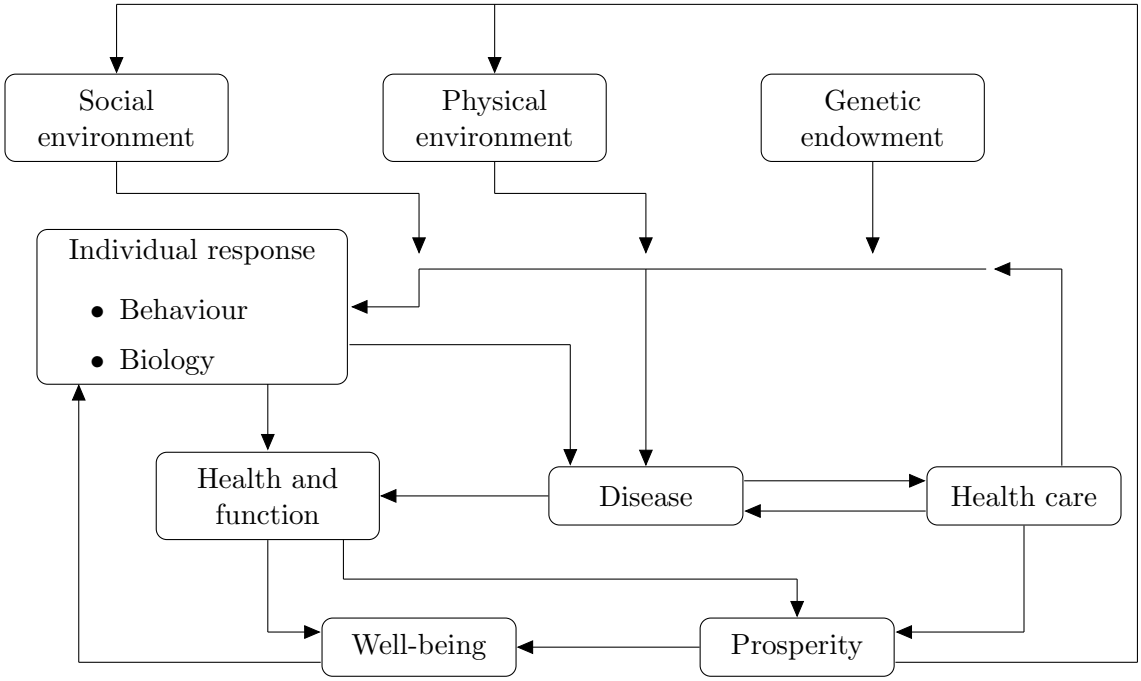

Figure 1.1: Economic model of health care and health

Figure 1.1 shows the causal model of health determination we consider in this thesis. This is taken from Evans and Stoddarts 1980 [25] paper "Producing health, consuming health care (and updated in "Consuming research, producing policy" [26]), in which the authors argue that there has been too narrow a focus on health care as the main determinant of health, and suggest that the inclusion of a wider array of health determinants would lead to more effective health policy. The notion of 'health' is separated into three related but distinct concepts: disease, health and function, and well-being. The first of these, disease,

affects and is affected by health care. This two-way relationship is due to health care being sought when someone suffers a disease, and, once sought, this health care reducing this disease. The disease itself is caused by individual, social, physical environment and genetic factors, which are themselves interlinked as one affects another. Disease affects health and function, while health and function contribute to well-being. Well-being, however, is also affected by prosperity. This is crucial, in the authors view, as a reductionist view that restricts the determinants of health to health care fails to give due consideration to the other ways in which prosperity can be used to increase health (whether in terms of disease reduction, health and function or well-being).

To explore the effect of health care on health inequality, this thesis will focus on the section of the causal model in which disease and health care display a two-way relationship. Thus, in statistical terms, a major challenge in our analysis is to overcome the problem of endogeneity, or reverse causality, between health and health care. For example, suppose we run an Ordinary Least Squares (OLS) regression for health (y) on health care (x) in an attempt to find out the effect of health care on health.

$$y = \alpha + \beta x + \epsilon \tag{1.1}$$

If health also affects health care, because health care resources are concentrated in areas with ill health or individuals only seek health care when they suffer ill health, the reverse relationship is also true.

$$x = \delta + \gamma y + \upsilon \tag{1.2}$$

Substituting Equation 1.1 into Equation 1.2 yields Equation 1.3.

$$x = \delta + \gamma\left[\alpha + \beta x + \epsilon\right] + \upsilon$$
$$x = \frac{\delta + \gamma\alpha}{1 - \gamma\beta} + \frac{\gamma\epsilon}{1 - \gamma\beta} + \frac{\upsilon}{1 - \gamma\beta} \tag{1.3}$$

There is therefore correlation between x and ϵ, violating the exogeneity assumption in Equation 1.1, as $E(x, \epsilon) \neq 0$

As most of the chapters in this thesis base analysis at the area-level, it is not possible to accurately specify every part of Evan and Stoddart's model due to data availability. The important aspect of the model that is maintained throughout the thesis is the notion that there are more (and more important) determinants to health than health care.

1.2 Background

In the UK there is broad consensus that efforts should be made to reduce or eliminate socioeconomic health inequality. The Black (1980) [22], Acheson(1998) [20] and Marmot(2012) [45] Reviews all concluded that health inequality in the UK exists and is a problem, despite the NHS aims of equitable access and reducing avoidable health inequalities. The NHS can be considered a product of the consensus:

> "The NHS was born [in 1948] out of a long-held ideal that good health care should be available to all, regardless of wealth." [68]

The NHS itself is the principal provider of health care in the UK. It is a universal health care system, funded from general taxation and free at the point of use for nearly all services. Health care in the UK is a devolved issue; Scotland, Northern Ireland, Wales and England have their own separate NHSs, though they have been organised along similar lines and operate in fairly similar environments with fairly similar populations. During the period

of study the NHS was organised on regional lines, with local Primary Care Trusts (PCTs) commissioning health services in an area and larger Strategic Health Authorities (SHAs) overseeing collections of PCTs.

How health care contributes, both positively and negatively, to health inequality is a crucial political issue within the United Kingdom. With broad popular support for the goal of health equality, massive spending in the health care sector [68], and large structural changes to health care in the offing [56], analysis of this relationship is important in order both to make informed policy decisions and evaluate current efforts.

It should be noted that though the subject of research is important, it is unreasonable to expect the solution to health inequality to lie within a health care policy, or set of health care policy interventions. Health care is not the sole determinant of health, as shown in Figure 1.1. Therefore it cannot be expected to eliminate health inequality. Many other determinants of health are covered extensively in the World Health Organisation's report on the social determinants of health [46].

Since 1998 there has been a strong Government focus on reducing health inequalities following the Acheson Report [43]. The Government response the following year set the target to reduce health inequalities by 10% for 2010, with inequality measured as the difference in life expectancy and child mortality between the richest and poorest areas and social groups respectively [21]. Though many schemes and policies were successfully introduced, the targets were missed. In 2008 the Secretary of State for Health asked Professor Sir Michael Marmot to chair a review to determine the best evidence-based ways of reducing health inequality.

Meanwhile, the House of Commons Health Select Committee produced a report on health inequalities in 2009 [36]. In it they noted that though much effort had been expended on reducing health inequalities, health inequalities had still increased during the previous decade. They recommended a far greater general focus on evaluation and evidence in

policies to reduce health inequalities. Regarding specific NHS contributions they found that lack of access to health care was not a major cause of health inequalities; though some recommendations were made relating to specific NHS policies, the majority concerned other causes of health inequalities, such as nutrition, education, the built environment and tobacco.

The Marmot Review [45] analyses from, and bases its recommendations in, a social determinants of health framework. Within this context, health care is included as one of the determinants of health that is socially graded; the ability to extract health benefits varies across social groups. Therefore, though the majority of proposals in the Review pertain to determinants of health other than health care (such as taxation, fair employment and the built environment) there are specific changes to health care that the Review recommends, and which are of interest in this document. Specifically the report suggests policy objectives in three areas:

- give every child the best start in life;
- create fair employment and good work for all;
- strengthen the role and impact of ill health prevention.

The first of these regards the advantages of targeting early childhood. As events early in life affect lifelong health it is important to work against inequality from before birth. The Review goes on to say:

> "There are strong associations between the health of mothers and the health of babies and equally strong associations between the health of mothers and their socioeconomic circumstances. This means that early intervention before birth is as critical as giving ongoing support during their child's early years."

As such, the Review recommends resources be targeted at pre- and post-natal interventions.

The second deals with the contribution that health care can make to good psychosocial health in the work context. The report notes that work can both harm and protect health, and as such is an important determinant of health. It therefore proposes more emphasis be placed on stress management guidance and 'Fitness to Work' notes, in order to prevent the harm work can cause to health. The Fitness to Work idea allows doctors more flexibility when deciding whether an individual is fit to work by advising on adjustments that would allow a return to work, where previously the choice was binary - to sign a sick note or not.

The third objective includes the most substantive changes to health care in the UK. Firstly, the report notes the responsibility of promoting health is not solely the NHS. However the definition of health promotion and ill health prevention vary between organisations. To aid the coherence of health promotion, the report proposes that the Government agree on a single definition. The Review argues that the advantaged in society are more likely to respond, and respond successfully, to any health promotion interventions, whether screening or behaviour-changing. Therefore, interventions should target the social gradient, which the authors of the report term 'proportionate universalism'. To this end, the use of preventions that are shown by evidence to work across and against the social gradient is encouraged, for instance drug treatment centres for drug abusers. As well as focusing on the social gradient of ill health prevention, an increase in the funding of ill health prevention of 10% a year up to 0.5% of GDP by 2030 is recommended, along with a refocusing on longer term projects.

More general policy suggestions are also made, such as integrating more closely with local councils and charities, and addressing inequalities in health care that might lead to health inequality. The number of specific health care suggestions seems small due to the focus on alleviating the psychosocial and life-course factors of health inequality.

The Review provoked much discussion and some criticism, with the journal "Social Science and Medicine" devoting a whole issue to the topic. Broadly speaking, many health economists questioned the validity of the research underpinning the suggestions, while epidemiologists and public health researchers criticised the report for not going far enough in its political recommendations. Canning and Bowser [12] questioned the necessity and cost of focusing on social equality when economic growth and technological advances increase health, and dispute the mechanisms that create health inequality. Nevertheless they agree that early childhood health intervention is desirable on societal health grounds. Birch [10] criticises the lack of evidence behind the health inequality-reducing interventions, as well as the assumption that giving people more control over their lives will lead to more health-improving behaviours. Chandra and Vogl [15] question the causation from socioeconomic status to health, and criticise the lack of evidence for, and attention to causality within, the policy prescriptions. Pickett and Dorling [58] claim the Review does not go far enough in advocating for more radical social change, suggesting, amongst other things, a maximum income. Whitehead and Popay [69] praise the Review, but note political issues in its implementation. In this they are joined by Nathanson and Hopper [50], who also question some of the evidence for the causal mechanisms. The most common complaint from economists was whether socio-economic position determined health, or whether health determined later socio-economic position. As evidence, Chandra and Vogl [15] cite Case and Paxson[13] that self reported health is better predicted by future occupational grade than current occupational grade.

Since then the Health and Social Care Act 2012 has set in motion a reformation of the NHS [56]. PCTs have been replaced by Clinical Commissioning Groups (CCGs), led by General Practitioners (GPs), and given the ability to commission services from any qualified provider. The legislation was opposed by many health professionals. It is unclear to what extent the operation of the NHS will actually be changed, and therefore how the contribution of health care to health inequality will be affected. Part 1 Section 4 of the legislation amends the National Health Service Act 2006 to insert the clause

> "Duty as to reducing inequalities
>
> In exercising functions in relation to the health service, the Secretary of State must have regard to the need to reduce inequalities between the people of England with respect to the benefits that they can obtain from the health service."

which indicates that health inequality is still a concern, however as the Secretary of State for Health is devolving the operation of health care it is unclear what exercising of functions this will apply to. In terms of funding, there is concern that a movement towards a more age-based funding formula for CCGs will exacerbate health inequality [8].

1.3 Aims and objectives

This thesis will evaluate the contribution of state-provided health care to socioeconomic health inequality in England - in terms of the theoretical framework set out in Section 1.1 we focus on the two-way relationship between health care and health. To this end, there are three aims:

Aim 1. To determine the extent of socioeconomic inequality in health care and health

Aim 2. To quantify the effect of health care on health

Aim 3. To quantify the contribution of health care to health inequality

To realise these aims the following objectives are adopted:

Objective 1. To use concentration index methods to explore area-level inequality in health spending

Objective 2. To estimate the effect of health spending on area-level health

Objective 3. To estimate the contribution of health spending to area-level health inequality

Objective 4. To explore how area-level health inequality can be studied in a panel data context

Objective 5. To use concentration index methods to estimate individual-level inequality in health care utilisation

Objective 6. To estimate the effect of area-level health spending on individual-level health care utilisation

Objective 7. To estimate the effect of individual-level health care utilisation on individual-level health

Objective 8. To estimate the contribution of individual-level health care utilisation to individual-level health inequality

1.4 Overview

In Chapter 2 we review the research literature. The purpose of this is to determine how much is already known on the subject, and find out which techniques are considered most appropriate to use to achieve our aims and objectives. Two reviews are conducted: a systematic review on papers that explore the effect of health care on health inequality, and a systematic review of the papers that explore the effect of health care on health. The reason for conducting two literature reviews is that health care's effect on health inequality is predominantly through its effect on health. As such, subsequent analysis must have as an intermediate stage an estimation of the effect of health care on health inequality. Very few papers are identified in the first review, which provides a preliminary indication of the

contribution this thesis makes to the literature on health inequality. The second review identifies a large number of papers that quantify the effect of health care on health.

Chapter 3 uses some of the analyses [47] [48] identified in the second literature review as a basis to explore the effect of health expenditure on health inequality, using Concentration Index (CI) decomposition techniques. The Instrumental Variable (IV) PCT-level analysis of Martin et al. [47] is updated and re-specified to allow CI decomposition. This provides a snapshot of health care and its effect on health inequality in England in 2008, which addresses Objectives 1 to 3.

To extend the analysis in Chapter 3 from a static model to a panel dataset in order to address Objective 4, in Chapter 4 we explore the problems faced when trying to measure inequality in a panel data context. The issue of the contribution of time-invariant heterogeneity to inequality is highlighted, discussed, and a possible solution put forward. As an example, the contribution of health care to health at the international level is estimated with data from the World Bank. This analysis introduces the Fixed Effects Re-Decomposition technique for modelling health inequaity over time.

Armed with a method to deal with the inequality due to time-invariant heterogeneity, Chapter 5 returns to the PCT-level analysis of Chapter 3, extending the dataset to cover six years. Combining IVs with the new technique allows us to estimate the effect of health care on health inequality over time, partitioning inequality into long term and temporary segments. This achieves Objective 4.

Moving from area-level data to individual-level data, Chapter 6 analyses data from the British Household Panel Survey, linked to PCT-level variables. In this chapter, self assessed health is used in place of mortality to measure health. A two-stage model is built for both GP visits and hospital inpatient days: in the first stage the effect of PCT spending on health care utilisation is estimated; in the second the effect of health care use on health is estimated. Overall this model specifies how spending at a national level can affect health

of an individual. Using these estimations, the effect of health care spending and health care use on health inequality is found, and Objectives 5 to 8 are addressed.

Finally, Chapter 7 discusses the findings of this thesis. The results are summarised and the limitations of the analysis are noted. Conclusions about the contribution of health care to health inequality are drawn, and the contributions of this thesis to the health economics literature are highlighted.

Chapter 2

Literature Review

2.1 Introduction

This chapter comprises two systematic literature reviews. These are conducted in order to find out what is known of the effect health care on health inequality at the population level. The first of these searches the literature for papers on the effect of health care on health inequality, contributing to our understanding of how best to tackle Aims 1 and 3. Very little is found, and what is available is not particularly pertinent. To expand the scope of literature considered, the second literature review searches for information on the effect of health care on health, in order to aid our understanding of how best to meet Aim 2. As this is a necessary intermediate step of a direct link between health care and health inequality, it is a useful starting point from which to advance to address Aim 3 in subsequent analysis. In this thesis we focus on the direct impact effective health care has on improving health, and do not explore how systems of health care that are organised more equally could have a positive effect on population health, as part of a broader "equality leads to health" influence proposed by Wilkinson and Pickett in their book "The Spirit Level: Why Equality is Better for Everyone"[71].

2.2 The effect of health care on health inequality

2.2.1 Literature search parameters

In this literature review we identified empirical papers that tried to quantify the relationship between health care and health inequality. Papers were extracted from the Embase, Medline and Econlit databases if they included a term from each of six different themes or concepts (see Table 2.1) deemed necessary for a paper to be relevant. There were no restrictions on date of publication, but only papers written in English were included.

Despite a large number of papers being extracted only a very small number were relevant, in part due to the large number of cost effectiveness analyses which mention potential inequity consequences in passing. Over 6000 papers were returned from the search and loaded into Reference Manager. These were than screened twice to reduce the number of papers and increase the relevance of results; firstly by scanning the titles, secondly by reading the abstracts. This reduced the number of papers first to 98, then to three.

Papers were excluded for three main reasons. Some looked at equality concerns in developing countries with developing health care systems. In such situations, interventions are often focused on infectious diseases, or infant mortality, and the ability to scale up in the face of large disparaties of infrastructural development. These features are very different from the situtation in England, where the main concerns for the future are the aging population, increased numbers living with multiple co-morbidities and an increasing burden of mental health problems.

Other papers evaluated specific treatment regimes and their related cost-effectiveness, and in which either the illness or the method of treatment had some connection (often loose) with socioeconomic factors/subgroups. These were excluded on the basis that they were not relevant to the population-level relationship between health care an health inequality, they merely evaluated for a given quantity of health care resource what could be gained

for a specific sub-population. If every conceivable treatment for every conceivable sub-population had been evaluated it would be possible to build a 'ground up' model of the effect of health care on health inequality - however even then time-constraints would have precluded such an endeavour.

Finally, some of the literature focused on changes to user fees in mixed-funding systems of health care. As the NHS is a taxpayer-funded health care provider, it is not necessary to explore what the relationship between price and health care means for health inequality.

Theme	Statement
Health Care	expenditure OR fun* OR spen* OR care OR use OR utili?ation
Health	healt* OR morta* OR morbi* OR life expectancy OR death
Equality	equ* OR inequ*
Data	data
Contribution	contrib* OR reduc* OR rais* OR improv* OR increa* OR decrea* OR effec*
Empirical	empiri* OR econom* OR statisti*

Table 2.1: Search terms for the effect of health care on health inequality

2.2.2 Literature search results

The three key papers identified by the literature review are "Income, health and health care utilization in the UK" by Mangalore [44]; "How much does health care contribute to health inequality in New Zealand" by Tobias and Yeh [63]; and "Inequalities in health and health service delivery: A multilevel study of primary care and hypertension control" by Veugelers, Yip and Burge [67].

Theory: Mangalore 2006

Mangalore develops a three equation model for which health, the decision to access health care, and income are the dependent variables, and each is based on a range of demographic variables and lags of the dependent variables forming a dynamic system of equations.

2.2 The effect of health care on health inequality

Health is affected by the individual's previous income, decision to use health care and demographic variables. Income is determined by health and demographic factors. The decision to access health care is based on the expected utilities of accessing health care against not accessing it given previous gains to health care, some demographic factors and a cost proportional to work time. The only supply variables are the number of General Practitioners (GPs) per thousand in the local authority district and dummies for the Health Authority region.

The paper does not study the relationship between health care and health inequality explicitly. It models health inequality as being determined by individual characteristics and the decision to access health care. In this way, it actually looks at the effect of health care on health and permits socioeconomic inequality in the use of health care and endowment of health. It is included in this section because it provides a framework within which health inequality and health care are brought together, however the effect of health care on health inequality is a derivation of the effect socioeconomic factors have on seeking health care.

Results: Mangalore 2006

The model is estimated using British Household Panel Survey (BHPS) data for the years 1991, 1992 and 1993. For estimation, health (which is a latent variable in the theoretical model) is substituted out. Income is predicted with an OLS regression and fits the data well with an R^2 of 0.672. The decision to access health care is fitted to a probit model and predicts correctly 79.25% of the time. The signs and significance of the coefficients are as one would expect, with the only surprise being the lack of significance of most of the regional dummies. The GP variable in the health utilisation equation is also insignificant.

An issue with the paper is that due to the small number of health care supply variables, the health care inequity is effectively driven by the decision of the individual, which is made

based on their information set and their aversion to the cost of access (the latter is based on demographic factors). Consequently the effect of health care on health inequality is largely down to characteristics of the individual which affect the decision to access health care, and their prior experience (which exacerbates this effect) because there is no mechanism by which people of different social groups can extract more or less benefit from health care, nor is there variability in the quantity or quality of health care available to people of different social classes.

This paper is useful insofar as it considers the behavioural factors that could cause health care to influence health inequality, but says little about actually how health care itself could differentially affect the health of people from different social groups.

Theory: Tobias and Yeh 2007

The Tobias and Yeh paper, on the other hand, does not make an assumption as to how health care might cause or mitigate health inequality. They look at the health inequality in mortality in New Zealand by estimating and comparing a ratio of mortality amenable to health care to total mortality, for different ethnic and socioeconomic groups. Ethnic estimates are standardised for age, sex and socioeconomic group; socioeconomic estimates are standardised for age and sex. They used data on causes of death between 2000 and 2002 in New Zealand, and directly standardised based on World Health Organisation data. Confidence intervals were derived from 1000 bootstrap iterations.

Amenable mortality was defined according to the Australian and New Zealand Atlas of Amenable Mortality, which is updated regularly to take account of health technology advances. Conceptually, amenable mortality is defined as a subset of avoidable mortality. Avoidable mortality includes those deaths from causes which could have been avoided through incidence reduction as well as those which could have been treated through fatality reduction. Amenable mortality comprises the latter of these two, though the authors note

that health care can also influence the former.

The technique is analogous, therefore, to the estimates in the Marmot Review [45] of the number of lives which could be saved if everyone had the health of the rich. In this case, it is modified to be the number of lives saved if everyone benefited from health care to the same degree as the rich benefitted. This gives a high-level overview of what a more equal health care system could deliver. However, it seems likely that if the rich are gaining more from health care they are using health care resources more intensely. In this case this theoretical 'more equal' health system would cost more, as all sub-groups would need to increase their utilisation to the level of the rich. Furthermore, it is not necessarily the case that all sub-groups would gain as much health from an identical level of utilisation. Given the complexity of health determination explored in Evans and Stoddart [25], it seems likely that different ways of working, different mixes of health care inputs and different non-health care determinants of health would be needed to ensure all groups gained equally from health care.

Results: Tobias and Yeh 2007

The results reveal a significant positive contribution of health care to health inequality, both ethnic and socioeconomic (meaning health care increases health inequality). This indicated contribution, however, does not specify any mechanism by which health care influences health inequality. The amenable mortality data takes no account of the health care actually received by the patient, it merely indicates that the cause of death can be treated and that the death rates are different between the groups. In other words there is no consideration that the difference between ethnic and socioeconomic groups could be due to pandemic discrimination within the health service, or massive aversion towards the health service from the people in the demographic group, or even higher incidence of disease and co-morbidities in disadvantaged group which render health care less effective. Because these potential factors are not disentangled, the estimation of the contribution of

health care to health inequality is likely to be an upper limit.

Though this technique is useful in highlighting health care's contribution to health inequality, it is insufficient to explain or describe much about it.

Theory: Veugelers, Yip and Burge 2004

The final paper identified by the review is Veugelers et al's 2004 paper on primary care and hypertension [67]. Using a two level model, they look at whether the availability and access of local primary care influences the diagnosis and management of hypertension in adult residents of Nova Scotia, and therefore contributes to area-level health inequalities due to varying access to care. As care of hypertension falls within the scope of preventative medicine it is hypothesised that in localities where primary care is overstretched and understaffed acute medicine will take precedence, and area-level socioeconomic inequality in hypertension might be the result of inequity of primary care resources.

Data is provided by the 1995 Nova Scotia Heart Health Survey and analysis spans 3094 individuals across the 64 functional geographies, with the first level at the individual level and the second at the geography. The survey includes a home interview and also measurements from a clinical session. Four categorical dependent variables are used: has hypertension previously been diagnosed; is there hypertension that has not been previously diagnosed; is anti-hypertension medicine being used; is there hypertension that is not being treated. Individual level covariates used are: age, gender, smoking, BMI, the presence of chronic conditions, household income, education and self assessed health. Three area level variables are also used: local socioeconomic status, measured by average household income; local health status, measured by local average life expectancy at 1995; and local access and availability of health services, measured by the number of GP visits standardised by age and gender, which is the principal variable of interest.

Results: Veugelers, Yip and Burge 2004

The results are presented as odds ratios, with the full set of covariates used in their standardisation. None of the geographical variables are found to be significant at the 5% level, for any of the dependent variables, so it appears that there is no evidence that local socioeconomics status, local health, or local access to health services has any effect on the detection or treatment of hypertension.

If there is no effect of health service access on health, there can be no effect of health service access on health inequality. However, it is not clear that the measurement used for access is sophisticated enough to determine this. Even standardised for age and gender profiles, the number of GP visits does not necessarily provide any information on how difficult it is to see a GP, or for how long appointments will be and of what quality.

In summary, it appears from the literature review that although health care and health inequality are popular topics there are relatively few papers examining the contribution of health care to health inequality. The two identified that do are useful for considering the relationship, but insufficiently describe how and why this contribution might occur. The 'how' and the 'why' are an important part of this issue, and it is hoped this thesis will go some way towards addressing this gap.

Due to the small number of papers addressing the effect of health care on health inequality, a further literature review was conducted on the effect of health care on health.

2.3 The effect of health care on health

This section reviews literature on the effect health care has on health. This relationship is a clear intermediate stage in the relationship between health care and health inequality. Initially the Embase, Medline, Social Science Citation Index and Econlit databases were

searched by the same criteria as in the previous section, with the AND argument for the inequality theme replaced by a NOT argument. Unfortunately this resulted in an unworkable number of papers (upwards of 14000). Based on a previous informal literature review it was known that the endogenous aspect of the relationship between health and health care was crucial in understanding the effect, so the equality theme was replaced with one to pick out papers which addressed endogeneity as shown in Table 2.2. Additionally, results from the previous informal literature review were included; in this review useful references were extracted from the main paper on health care effectiveness in England, Martin et al. [47], and the references of these referenced papers.

Theme	Statement
Health Care	expenditure OR fun* OR spen* OR care OR use OR utili?ation
Health	healt* OR morta* OR morbi* OR life expectancy OR death
Data	data
Contribution	contrib* OR reduc* OR rais* OR improv* OR increa* OR decrea* OR effec*
Empirical	empiri* OR econom* OR statisti*
Endogenous	endogenous OR endogeneity OR instrument OR instruments OR instrumental

Table 2.2: Search terms for the effect of health care on health

We restricted papers to the English language, but did not restrict date of publication. Papers were included if they estimated the effect on health of health care variables, where 'health care variables' include system-wide aspects of the health care system (such as doctors, health care spending and health care utilisation) but exclude specific micro-level interventions (such as a specific drug intervention). This was done for two reasons: firstly, as the focus is on population-level health inequality it is population-level health care that we are concerned with; secondly, given the vast quantity of cost effectiveness papers and findings it would not be possible within four years to construct a 'bottom-up' model of health determination based on individual treatment practices.

Our search resulted in 25 papers. These fall naturally into four groups: Group 1 - in-

ternational comparisons where health care is treated as exogenous (Table 2.3); Group 2 - intranational comparisons where health care is treated as exogenous (Table 2.4); Group 3 - international comparisons where health care is treated as endogenous (Table 2.5); and Group 4 - intranational comparisons where exogeneity is not assumed (Table 2.6). All findings are statistically significant at the 5% level, unless otherwise stated.

Authors and date	Data, units and time period	Finding
Group 1: International and Exogenous		
Asiskovitch[5]	OECD countries	Some evidence health expenditure increases life expectancy at 65 years
2010	47, 1999-2004	
Baldacci et al. [7]	Developing countries	Health expenditure reduces child but not infant mortality
2002	94, 1996-1998	
Cochrane et al. [16]	Developed countries	Doctors raise mortality, NHS lowers mortality
1978	18, in 1970	
Joumad et al. [40]	OECD countries	Health spending and resources increase life expectancy
2008	23, 1981-2003	
Nixon and Ulmann [51]	EU countries	Health spending slightly increases health, significantly reduces infant mortality
2006	15, 1980-1995	
Fayissa [27]	Sub-Saharan African countries	Public Health Expenditure decreases child mortality
2001	34, no date	
Fayissa and Gutema[28]	Sub-Saharan African countries	Public Health Expenditure lowers life expectancy
2008	31, 1990-2000	
Fayissa and Traian [29]	Eastern European countries	No significant effect of health expenditure on infant mortality
2008	13, 1997-2005	
Gupta et a.l [32]	Developing countries	Health expenditure reduces child and infant mortality
2002	50, 1993-1994	
Or [55]	OECD countries	More doctors increase health
2001	21, 1970-1995	
Young [73]	Developed countries	More doctors increase mortality
2001	29, no date	

Table 2.3: Group 1: International and Exogenous

Authors and date	Data, units and time period	Finding
Group 2: Intranational and Exogenous		
Cremieux et al. [19]	Canadian provinces	Increased spending and number of doctors increase health
1999	15, 1978-1992	
Cremieux et al. [18]	Canadian provinces	Increased pharmaceutical spending increases health
2005	10, 1981-2000	
Holian [37]	Mexican communities	Health care utilisation reduces infant mortality
1989	125, 1976-1977	
Paul [57]	Bangladeshi neonates	Distance to doctor increases risk of neonatal death
1991	1787, 1984	
Ssewanyana and Younger [62]	Ugandan neonates	No effect of health care on one year survival, except for vaccination
2008	"3000+", in 1990	
Young [73]	Japanese prefectures	No effect of physicians on mortality
2001	47, in 1995	
Young [73]	US counties	Hospital beds reduce mortality, physicians and health spending raise mortality
2001	"3000+", in 1990	

Table 2.4: Group 2: Intranational and Exogenous

Authors and date	Data, units and time period	Finding
Group 3: International and Endogenous		
Anyanwu and Erhijakpor[3]	African countries	Health expenditure reduces child and infant mortality
2009	47, 1999-2004	
Filmer and Pritchett [30]	Countries	Public health spending reduces child mortality (at 10% significance level)
1999	98-119, 1992	

Table 2.5: Group 3: International and Endogenous

Authors and date	Data, units and time period	Finding
Group 4: Intranational and Endogenous		
Almond et al. [2]	US births	Spending on neo-natal hospital care reduces mortality
2008	66m, 1983-2002	
Martin et al. [47] [48]	English PCTs	Spending improves outcomes for cancer, circulatory problems, neurological problems,
2008	295, 2004/05	respiratory problems, gastro-intestinal problems, trauma and injuries, and diabetes.
Gravelle et al. [31]	English LA and HSE survey	GPs increase health
2008	Various [1], 1998-2000	
Aakviland Holmas [1]	Norwegian municipalitesl	No effect of GP numbers. Effect of type of GPs
2006	435, 1986-2001	
Ross et al. [60]	US states	Effect of mental health spending on suicide statistically insignificant
2010	51, 1997-2005	

Table 2.6: Group 4: Intranational and Endogenous

2.3 The effect of health care on health

In 1978 Cochrane et al [16] conducted a country-level analysis on the determinants of health. Surprisingly, they found that the number of doctors in a country was positively related to mortality. This result led to a number of papers further examining this relationship. The desirability of good health has led to advanced countries spending large proportions of their income on health care, in the belief it will provide good health. Such a result, if confirmed, would have severe consequences for the future of health care. Researchers in this field found a variety of results, and posited a number of different reasons for them. Young [73] believed the correlation to be spurious, an artefact of the independent movement of health professionals and immigrants to cities as a country develops, with the immigrants suffering stress and ill health once in the city. As such, the inclusion of a variable representing immigration should dissolve the doctor-mortality correlation. He implemented this for three datasets with mixed results. In Japan the positive doctor-mortality correlation dissolved, in the US the correlation lost strength but remained positive and statistically significant, but across OECD countries the inclusion of a refugee variable increased the association (the number of immigrants was unavailable). Other papers in Group 1 find similarly ambiguous results; overall there seems to be little consensus as to whether health care effects health positively, negatively or insignificantly in these papers.

Cremieux et al [19] argue that comparing countries leads to data which exhibits too much heterogeneity, as between countries there are too many different and potentially unobservable factors affecting health. Instead, they recommend using intranational data to measure the effect of health care, in order to reduce heterogeneity. They analyse Canadian provinces and find that both health spending and the number of doctors increase health. They control for any time-invariant heterogeneity in the level of infant mortality and life expectancy by employing fixed effects in a 15 year panel dataset. This implies that a better estimation of the effect of health care on health is gained when using more homogenous regional-level data. Correspondingly, the results in Group 2 are less ambiguous. Excepting Young's US counties analysis, the evidence points to health care having a

positive, if sometimes insignificant, effect on health.

The issue of endogeneity of health care is tackled in the papers of Groups 3 and 4. These papers assume and compensate for the reverse causality between health care and health by using Instrumental Variables. Aakvil et al. [1] construct Arellano Bond instruments from within the panel dataset, and find that the number of GPs has no effect on health. However the type of GP does, specifically, independent contract GPs increase health. The other papers use natural instrumental variables. Of particular note is the Martin et al. 2008 paper that measures the effect on cancer and circulatory disease mortality of health care spending across English PCTs in 2004/05 [47]. In their study, they develop a theoretical model to explain the health care provider's budget decision (Equation 2.1) and the effect of health expenditure on health outcomes (Equation 2.2) measured by Standardised Mortality Ratios (SMR) and Years of Life Lost (YLL). In the expenditure equation, it is assumed that spending on a disease increases with the need for health care and the money available to spend, but is reduced by higher levels of mortality related to other diseases. Health outcomes are determined by the need for health care and the amount spent on health care in the PCT.

Each equation is then estimated by two stage least squares using instruments derived from the 2001 Census. For the cancer model, these are the percentage of unpaid carers and the percentage of lone pension households. For the circulatory model, the instrument set is augmented with the the Index of Multiple Deprivation 2000 (IMD2000). The model estimated is

$$ProgrammeExpenditure_i = \alpha + \beta_1 Need_i + \beta_2 TotalExpenditure_i + \beta_3 NonProgrammeSMR_i + \nu_i \quad (2.1)$$

$$ProgrammeSMR_i = a + b_1 Need_i + b_2 ProgrammeExpenditure_i + \epsilon_i \quad (2.2)$$

where the Need variable is measured by the needs component of the PCT funding allocation formula, which changes the funding for PCTs based on area-level characteristics such as age and deprivation. The results show a substantial negative effect of health care spending on mortality. The Need variable shows a positive coefficient, which indicates a correlation between mortality and deprivation. Additionally, this paper attempts to predict the cost of saving a year of life from either disease, using the health outcome variable Years of Life Lost (YLL). This variable is constructed by summing the difference between age of death and 75 for every under-75 death due to the condition.

This analysis is extended to include more disease areas in 2012 [48], largely supporting the previous results, although the models for other programmes of care generally perform less well.

In both Groups 3 and 4, the literature indicates that health care improves health, with the sole exception of Ross et al's mental health care study [60] in which there is no statistically significant effect. This suggests that endogeneity is a concern in the relationship between health care and health. However, in the two papers of Group 3, the use of instruments does not appear to significantly effect the findings compared with non-IV regressions, whereas in Group 4 (except Ross et al.) IVs make quite a substantial difference.

The two main issues in estimating the effect of health care on health are reverse causality and heterogeneity. Heterogeneity is particularly problematic when making international comparisons. When the units of study are sufficiently different from one another, the relationship under examination varies between the units of observation. In a health context, the relationship which determines health in Scandanavian countries might vary from the determination in Mediterranean countries, due to diet, lifestyle, genetics, health system and social structure. Though time invariant level effects can be accounted for in panel data fixed effects models, this only addresses the heterogeneity in the basic value of the dependent variable, not the heterogeneity one might expect in the relationship between variables. For instance, the effect of health spending on mortality might vary stucturally

between units based on: the effectiveness of the health system; the compliance of populations to medical direction; or the priorities towards which health spending is directed. Unfortunately there is no real solution to this problem. One can try to increase the detail of the model by including more variables (often undesirable when faced with a small and finite dataset) or turn the attention of analysis to a more homogenous unit.

Reverse causality is the more pertinent problem for within-country analyses, especially in the context of national health service-based systems. Simply, when policymakers are allocating health care to effect the most equitable and efficient allocation, and health care exhibits diminishing returns to health, more health care will be targeted in less healthy places. But as health care is unlikely to equalise health throughout the country, the concentration of health care in unhealthy places may be mistaken for the effect of health care on health. This would lead to the false conclusion that health care in fact reduces health. This can be remedied by the use of instrumental variables: variables which only influence health through their influence on health care.

Although neither issue is restricted to intranational or international analyses, it is intuitive to suppose that heterogeneity is a bigger problem the more distinct and larger the units, such as countries; endogeneity is a bigger problem the more similar the units, such as regions of countries. This notion is support by the greater impact instruments have on the papers in Group 4 as opposed to Group 3.

Heterogeneity will exist for any given statistical health care units in England, however, it should be possible to model the majority of it. Endogeneity due to reverse causality, especially in a centrally managed health system such as the NHS, is likely to be the principal obstacle in any analysis of English health care.

2.4 Summary

In this chapter we have explored the literature on health care's effect on health and health inequality. There are not many useful papers on the effect of health care on health inequality. This presents a challenge for our analysis, but also indicates the size of contribution which this thesis can make. The larger number of results found in the survey of papers studying the effect of health care on health is useful, as the issues of heterogeneity and endogeneity encountered will be important in subsequent chapters. Overall, the main discovery in this chapter is the importance of instrumental variables when estimating the effect of health care on health, especially in a single-country analysis, which is highly pertinent for Chapters 3, 5 and 6.

Chapter 3

The effect of health care on area-level health inequality

3.1 Introduction

To understand the relationship between health care and health inequality, we first need to know how NHS resources are deployed. As resources cost money, the simplest way to study this is to analyse NHS expenditure. In 2002 the Department of Health embarked on a national programme budgeting project [53]. This categorises expenditure at the PCT level into specific medical conditions. There are 23 categories and 49 subcategories based on International Classification of Disease codes (ICD-10). In this chapter we shall analyse Primary Care Trust (PCT) level programme budget data in order to determine the extent of socioeconomic-related inequality in health care. Later in the chapter we will examine the effect of health care on health and, combining with our analysis of inequality, estimate the contribution of health care to health inequality (addressing Aims 1 to 3 and Objectives 1 to 3).

Analysis is conducted at the PCT level in the financial year 2007/08. Since then, PCTs have been replaced by Clinical Commissioning Groups, under the Health and Social Care Act 2012. In 2007/08 there were 152 PCTs in England, with an average population of

334,000. PCTs had a variety of roles. They assessed the health needs of the populations they served and commissioned primary, community and secondary care from providers to meet those needs. They held their own budgets and set their own priorities, within the overriding priorities set by the Strategic Health Authority in which they are located and the Department of Health. Collectively PCTs were responsible for spending around 80% of the total NHS budget, which was split between them based on a capitation formula that predicted health care needs. This capitation formula, however, only determined the funding each PCT should get - in reality there was a "Distance from Target" factor that was designed to smoothly move PCTs to their rightful allocation over time.

Firstly, we shall conduct a simple analysis of the relationship between spending and deprivation. Using concentration indices on programme budgets we can describe how the allocation of money was associated with socieconomic inequality. A widely used method of measuring inequality, the concentration index (CI) approach [52] is based on the Gini coefficient.

Secondly, we shall estimate the effect of health spending on health, using as a base Martin et al's (2008) approach discussed in the previous chapter [47]. We restrict our analysis to cancer and coronary heart disease (CHD) mortality and estimate the relationship between spending and health based on 2007/08 data. A critical issue facing macro-level analysis of health care spending is the degree to which health funding and health outcomes are endogenous. Areas with higher levels of morbidity and mortality attract higher levels of funding. This does not imply that funding causes the illness, which might appear to be the case from simple correlations of these two variables. With suitable instrumental variables endogeneity can be purged from the expenditure variable, so the identification and use of such instruments is important for analysis. In this analysis we are specifically focusing on the endogenous relationship between disease and health care that was pointed out in Chapter 1, while controlling, as best we can, for the other determinants of disease.

Finally we estimate the condition-specific mortality CI and decompose it into contributions

based on our regression model. CI decomposition partitions the CI inequality in a variable into the CI of its determinants multiplied by the elasticity of the determinant with respect to the dependent variable.

3.2 The distribution of spending

A necessary step in estimating the contribution of health care to health inequality is examining the inequalities in health care.

Variable	Mean	S. D.	Min	Max
Infectious diseases	23	18	5.8	124
Cancers and Tumours	89	16	46	152
Disorders of Blood	19	6.5	5.7	52
Endocrine, Nutritional and Metabolic problems	39	6.4	21	66
Mental Health Disorders	183	35	115	307
Problems of Learning Disability	54	17	4.5	131
Neurological	62	12	34	112
Problems of Vision	31	6.6	14	54
Problems of Hearing	8	3.4	1.6	20
Problems of Circulation	124	21	58	222
Sub-programme: CHD	0.0408	0.0129	0.0139	0.117
Problems of the Respiratory system	68	11	37	93
Dental problms	60	11	21	89
Problems of the Gastrointestinal system	75	14	40	117
Problems of the skin	30	6.3	19	58
Problems of the Musculoskeletal system	75	18	33	123
Problems due to Trauma and Injury	57	16	5.1	107
Problems of the Genitourinary system	68	13	24	107
Maternity and Reproductive Health	59	15	20	98
Conditions of neonates	16	7.2	0.71	42
Adverse effects and poisoning	16	4.1	6.7	32
Healthy Individuals	31	14	5.4	81
Social Care Needs	39	5.4	0.19	43
Other	230	5.9	116	50
Overall spending	1456	8.1	1290	1977

Table 3.1: Summary Statistics for Programme budgets in 2007/08 (n=152) in £pc

Summary statistics from the programme budgeting data are found in Table 3.1. The units

are pounds per capita. The largest programmes of spending are Mental Health, Circulatory problems and Other. The Other category includes spending on GP services. It should be noted that, relative to the size of the mean, there is more variation in the programmes than in overall spending. This implies that the variations in individual programmes' expenditure are due to differences in PCTs' spending patterns rather than differences in their overall budgets. This would be the result if PCTs were responding to local, heterogenous health care needs.

The concentration index is based on the concentration curve (CC). A cumulative variable y is plotted against a ranking variable r, which is typically income, deprivation or some other socioeconomic variable. If the spending per capita is the same in every PCT regardless of deprivation the line plotting this relationship runs at 45 degrees, known as the line of equality (note, this line only runs at exactly 45 degrees if the x and y axes are standardised to the same length). If spending is concentrated in more (less) deprived areas, the CC will lie above (below) the line of equality.

In Figure 3.1 we plot the cumulative spending per capita in each PCT for 2007/08 against the deprivation rank of the PCT, decreasing in deprivation as measured by Index of Multiple Deprivation 2007 (IMD2007). This measure of deprivation contains seven domains of deprivation (income; employment; health and disability; education, skills and training; barriers to housing and services; living environment; crime), based on 38 indicators. We restrict the figure to the three largest programme budgets and overall spending. The first thing to note is how close the concentration curves are. The most obvious deviation from the line of equality is Mental Health spending; Mental Health has a pro-poor distribution, apparently due to increased spending in the second most-deprived fifth of PCTs. The same group of PCTs also seem to spend slightly less on Circulatory problems, the CC for which is slightly pro-rich. Total and Other spending appear to follow the line of equality, though Other spending does display a pro-rich distribution in the middle of the figure.

The CI is defined as twice the area between the CC and the line of equality, with pro-poor

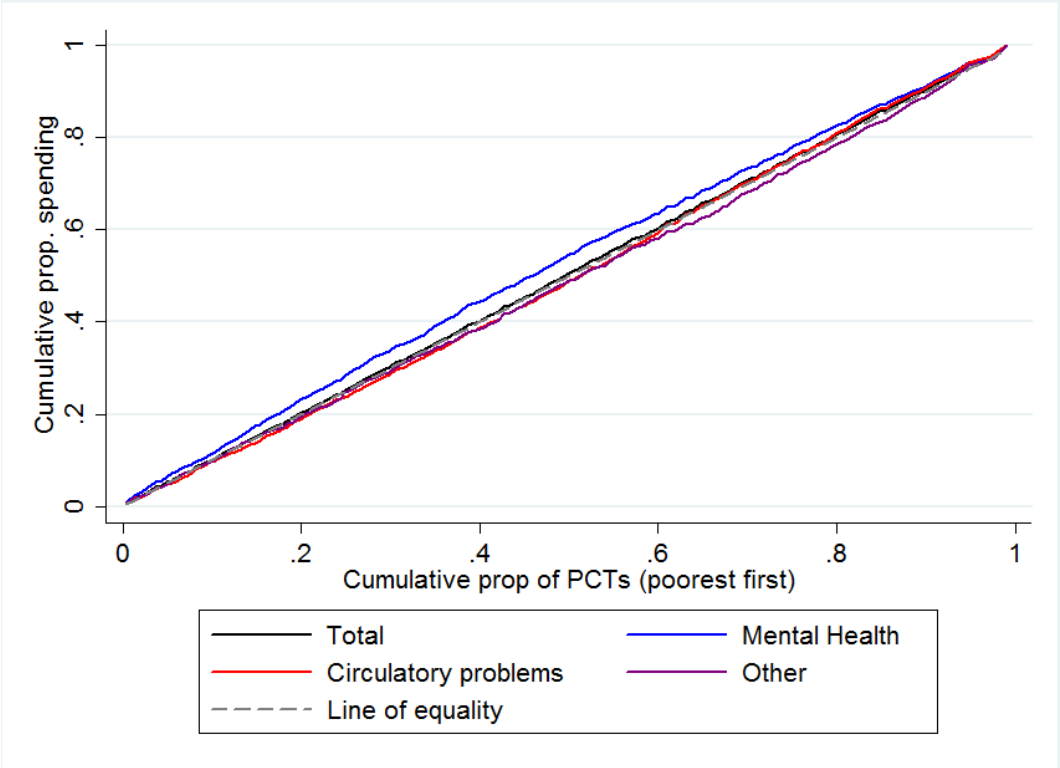

Figure 3.1: Programme budget concentration curves

values defined negatively and pro-rich values positively. This number gives an indication of how much inequality exists. If the CI is 1, this indicates that all the spending is located in the least deprived PCT, if it is -1 all the expenditure is spent in the most deprived PCT, a value of 0 indicates equality. The formula by which this is calculated is:

$$CI_y = \frac{2cov(r, y)}{\mu_y} \tag{3.1}$$

where y is the health variable of interest, μ_y is the mean of the variable and r is the ranking by socio-economic status. As the PCTs are of varying size, we weight these statistics by population.

Concentration indices for all programme budgets are reported in Table 3.2. Our previous CC findings are confirmed: Mental Health spending is pro-poor; Circulatory and Other

Variable	C. I	S.E	95% Conf. Int	
Infectious diseases	-0.135***	0.03	-0.193	-0.077
Cancers and Tumours	0.026***	0.009	0.008	0.043
Disorders of Blood	-0.036	0.026	-0.087	0.016
Endocrine, Nutritional and Metabolic	0.07	0.008	-0.008	0.022
Mental Health Disorders	-0.052***	0.008	-0.067	-0.037
Problems of Learning Disability	0.031	0.19	-0.007	0.069
Neurological	-0.001	0.009	-0.018	0.016
Problems of Vision	0.02**	0.009	0.002	0.038
Problems of Hearing	-0.042*	0.022	-0.085	0.001
Problems of Circulation	0.014*	0.008	-0.002	0.029
Sub-programme: CHD	0.00461	0.0144	-0.0278	0.033
Problems of the Respiratory system	-0.021***	0.008	-0.036	-0.006
Dental problms	-0.029***	0.008	-0.045	-0.013
Problems of the Gastrointestinal system	0.00004	0.008	-0.017	0.017
Problems of the skin	-0.008	0.01	-0.28	0.012
Problems of the Musculoskeletal system	0.051***	0.01	0.032	0.07
Problems due to Trauma and Injury	0.02	0.013	-0.005	0.046
Problems of the Genitourinary system	0.00003	0.009	-0.017	0.017
Maternity and Reproductive Health	-0.032**	0.015	-0.061	-0.002
Conditions of neonates	-0.043*	0.025	-0.093	0.006
Adverse effects and poisoning	0.008	0.013	-0.018	0.033
Healthy Individuals	0.00003	0.038	-0.074	0.074
Social Care Needs	-0.041	0.033	-0.105	0.023
Other	0.026***	0.012	0.003	0.049
Overall spending	-0.002	0.002	-0.005	0.001

Table 3.2: Concentration Indices for Programme Budgets; all standard errors are bootstrapped based on 2000 replications. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=152)

spending is pro-rich; Total spending is insignificantly different from equality. It should be noted that, as PCTs decide what services to commission, it is entirely unsuprising that the differences in spending within specific clinical areas are correlated with deprivation - one would expect health care needs and therefore PCT priorities to vary in such a way. Perhaps more suprising is that Total spending is equally distributed. The PCT resource allocation formula awards more money to areas with deprivation on the basis of that deprivation, in

order to reduce health inequalities. That Total spending is insignifcantly pro-poor suggests that the combination of other parts of the resource allocation formula and the Distance from Target factor are offsetting the premium that poorer PCTs receive. A cursory look at some of the significantly pro-rich and pro-poor programme budgets suggests that this could be due to the age-related portion of the resource allocation formula, as less deprived areas seem to spend more on health problems linked to older people: funding for vision and muscoskeletal problems is pro-rich while funding for maternity, neonates and infectious diseases is pro-poor. The standard errors in this figure are based on 2000 bootstrap replications of the CI.

3.3 The effect of spending

As previously explored in Chapter 2, analyses of the effect of health care are plagued by issues of endogeneity, most importantly heterogeneity and reverse causality. The former is of greatest concern when the dataset is international; in our PCT dataset, though we expect PCTs to to be different from one another, the more concerning source of endogeneity is reverse causality. As the need for health care increases with ill-health areas with more health care are likely to be those that experience worse health statistics. This is more severe the more centrally planned the health care system. The typical econometric method to address this is Instrumental Variable (IV) estimation, which we employ.

To estimate the effect of spending on health we follow the model of Martin et al. [47], and estimate the effect of 2007/08 PCT spending in the cancer and CHD health care on the 2008 PCT-level mortality statistics.

Martin et al. [47] measure the effects of cancer and circulatory disease spending across PCTs in 2004/05 accounting for endogeneity. In their study, they develop a two equation static theoretical model to explain the health care provider's budget decision and the effect of health expenditure on health outcomes (2). Each equation is then separately estimated

by two stage least squares (2SLS) using instruments derived from the 2001 Census and the Index of Multiple Deprivation 2000 (IMD2000), an area deprivation measure based on six domains. For the cancer model, these are the percentage of unpaid carers and the percentage of lone pension households. For the circulatory model, the instrument set is augmented by the Index of Multiple Deprivation 2000. The 2SLS mortality model is

$$X_i = \alpha_0 + \alpha_1 N_i + \alpha_2 Z_i + \epsilon_i \tag{3.2}$$

$$H_i = \beta_0 + \beta_1 N_i + \beta_2 \hat{X}_i + \mu_i \tag{3.3}$$

where X is the programme spending, H is the disease programmes Standardised Mortality Ratio (SMR), N is the needs component of the NHS resource allocation formula for PCTs, Z are the instruments for spending, α and β are parameters to be estimated, i indexes areas (PCTs) and ϵ and μ are error terms.

The results from these models show a substantial negative effect of health care spending on mortality in both programmes. For cancer, the elasticity of standardised mortality with respect to spending is -0.491; for circulatory disease, the elasticity is -1.387. Furthermore, the need variable shows a positive coefficient, which indicates a correlation between mortality and deprivation. Additionally, the paper attempts to predict the incremental cost per life year gained and per quality-adjusted life year gained from either disease, using the health outcome variable Years of Life Lost (YLL). This variable is constructed by summing the difference between age of death and 75 for every under 75 death due to the condition in each area. The estimate for a marginal life year saved from cancer is 13,100, rising to 19,070 when adjusted for health-related quality of life. For circulatory disease the corresponding estimates are 8,000 and 11,960.

Subsequent research [48] supports the original results using newer data and extends the analysis to more disease areas.

Instead of modelling the circulatory disease programme we focus on the CHD sub-programme. This allows us to link prevalence data from the Quality and Outcomes Framework, which allows us to more accurately measure the amount of money spent on each CHD sufferer.

3.3.1 Data and Estimation Strategy

The relationship under consideration is between mortality, demographic factors, need and expenditure, modelled using 2SLS and shown in Equations 3.4 and 3.5. This is similar to the model used by Martin et al. shown in equations 3.2 and 3.3 except that the dependent variable in the health equation is unstandardised deaths, we control for demographic factors, and need is proxied by deprivation.

$$X_i = \delta_0 + \delta_1 A_i + \delta_2 D_i + \delta_3 Z_i + \tau_i \tag{3.4}$$

$$y_i = \gamma_0 + \gamma_1 a_i + \gamma_2 D_i + \gamma_3 \hat{X}_i + \omega_i \tag{3.5}$$

where y is the number of deaths in the programme of care, A is the number of deaths expected when applying national mortality rates for age and sex groups to the areas specific age and sex profile, D is the deprivation of the area as measured by the overall index of the IMD2007, X is the money spent in the area on the care programme per patient with the condition, and τ and ω are error terms. The Z variables are instruments for spending and δ and γ are coefficients to be estimated.

Condition-specific mortality is measured by the number of all-age deaths in 2008 available

on the Health and Social Care Information Centre website [35]. To control for differences in age and gender structures the expected number of deaths (based on indirect standardisation) is used as a covariate, calculated as the number of deaths divided by the age- and gender-based SMR, also available from the HSCIC website. Algebraically, the formula for SMR is

$$SMR_i = \frac{Observed_i}{Expected_i} = \frac{\sum_m D_i}{\sum_m P_i R} \tag{3.6}$$

where m denotes the sub-population groups being standardised for (typically age-gender cohorts), D_i is the number of deaths in those groups in area i, P_i is the number of people in those groups in area i and R is the national death rate for those groups. Based on this it is clear to see that

$$Expected_i = \frac{Observed_i}{SMR_i} \tag{3.7}$$

provides a measure of the expected number of deaths in area i if each group being standardised for were to experience the national death rate.

We model actual deaths controlling for expected deaths as well as modelling SMRs because the inequality measures we subsequently employ are not meaningful with ratio variables. Additionally this allows the effect of age and gender structures more freedom in the regressions than would be the case with SMRs. Other health measures were explored, in particular 'excess deaths' (observed deaths minus expected deaths). However due to the requirements of Concentration Index analysis these were not pursued.

In the Martin et al. paper [47], the Needs component of the NHS resource allocation formula is used to control for differences in needs between areas. This is an index composed

of morbidity, mortality, demographic and deprivation area-level statistics. As health is the dependent variable in our analysis, we exclude the use of health variables as covariates and opt for deprivation as a proxy for need (additionally, the expected number of deaths incorporates the demographic features of area-level need). Deprivation is measured by the Index of Multiple Deprivation 2007 average score [54], which contains seven domains of deprivation (income; employment; health and disability; education, skills and training; barriers to housing and services; living environment; crime), based on 38 indicators. As it is an index it cannot be considered a cardinal measure: if PCT A has double the score of PCT B, A is more deprived but not necessarily twice as deprived. Therefore when elasticities of the deprivation variable are considered, they do not have the same objective meaning as the elasticity of spending with respect to mortality. The effect of deprivation is being controlled for rather than estimated, though the index also provides a ranking variable which we subsequently use for concentration index analysis.

Condition-specific expenditure is based on the programme budgeting data released by the Department of Health, namely the programme expenditure per person for the financial year 2007/08 [53]. The PCT population figure used is the Unified Weighting. However, this funding measure by itself only gives the condition-specific expenditure of a PCT as a proportion of the total number of people in the PCT. Ideally, the expenditure variable would restrict the denominator of this measure to those people with the condition, as the numerator (programme spending) is not being spent on people without the condition. Consider again two PCTs A and B, with the same population, the same total spending on cancer and the same standardised mortality, but A has 10% cancer prevalence and B has 2.5%. The spending variable should be four times higher for B than A as only a quarter of the people are being treated, at the same price, for the same condition, with the same outcome. Though there is no expenditure per affected person data, there is prevalence data for 2007 on the NHS Information Centre website, from the Quality and Outcomes Framework (QOF). Assuming prevalence did not vary substantially between the periods January to March 2007 and January to March 2008, dividing expenditure per person by

prevalence provides a reasonable measure of expenditure per affected person. Some level of time invariance is being assumed to allow for mortality data (which is reported in calendar years) to be regressed on expenditure data (which is reported in financial years). It is due to availability of prevalence data that the CHD sub-programme, rather than the more general circulatory disease programme, was modeled.

The analysis shows that expenditure is endogenous in the health equation. Thus, instruments Z are used to employ instrumental variable (IV) techniques. These instruments ought to affect expenditure, and only correlate with the standardised mortality insofar as they affect expenditure. A range of instruments were tried and tested against a variety of instrument test statistics. Notably, despite its intellectual desirability, Distance From Target was rejected as an instrument due to lack of explanatory power in the first stage.

For the cancer programme the proportion of unpaid carers and the spending on all other care programmes in the PCT were used. The first instrument is from the 2001 Census [54], the second from the programme budgeting data. Intuitively, a higher proportion of unpaid carers in an area means less money needs to be spent on care for those with cancer - it acts as a substitute for health care. If an area has a relatively more generous budget, it is understandable that more would be spent on cancer treatment. Crucially, one would not expect a greater proportion of unpaid carers, or an increase in the amount spent on non-cancer treatments, to increase or decrease the number of cancer deaths.

Similarly, for the CHD programmes of care we use the proportion of unpaid carers and the proportion who claim disability allowance as instruments. The first of these is based on the same intuition as in the cancer model. The second is from the Neighbourhood Statistics website [54], and is likely to increase the cost of cancer CHD treatment as facilities and protocols need to be tailored to suit the specific disability, while not significantly affecting mortality rates after controlling for deprivation.

The data used to estimate the model are summarised in Table 3.3. Although cancer and

3.3 The effect of spending

CHD programme spending are only 9% of total spending, mortality from these two causes accounts for 42% of all deaths in 2008. Please note the mortality standardisation uses mortality rates by age-gender cohort for 2010.

Variable	Mean	Std. Dev.	Minimum	Maximum	Period	Source
Cancer SMR	102.4	12.6	64	135	2008	NHS IC
Cancer deaths	847.4	531.3	241	3131	2008	NHS IC
Cancer expected deaths	847.4	574.1	225	3518	2008	NHS IC
CHD SMR	131	27	49	227	2008	NHS IC
CHD deaths	471	1963	839	11269	2008	NHS IC
CHD expected deaths	403	282	97	1782	2008	NHS IC
IMD 2007	23.7	9.1	8.09	48.26	2001-2005	Neighbourhood Statistics
Cancer spending per patient (000s)	8.69	1.7	5	13.8	2007/08	DH & QOF
CHD spending per patient (000s)	1.2	0.3	0.4	2.4	2007/08	DH & QOF
Cancer prevalence	0.0105	0.00215	0.005	0.053	2007	QOF
CHD prevalence	0.0352	0.00929	0.014	0.017	2007	QOF
Non-cancer spending per person (000s)	1.14	0.1	9.3	1.6	2007/08	DH
Proportion of carers who are unpaid	0.099	0.01	0.07	0.12	2001	2001 Census
Proportion claiming DLA	0.066	0.02	0.03	0.1	2008	Neighbourhood Statistics
Proportion non-white	0.107	0.11	0.007	0.61	2001	2001 Census
Population	333526	167522	100874	1101015	2007/08	DH

Table 3.3: Summary statistics for cancer and CHD regression variables (n=152)

3.3.2 Results

	Variable	SMR		Deaths	
1st Stage		OLS	2SLS	OLS	2SLS
Prog. spending	Expected deaths				-0.00025
					(0.0003)
	IMD2007		0.0672***		0.056***
			(0.0164)		(0.02)
	Non-cancer spending		0.432		0.31
			(1.96)		(1.98)
	Unpaid carers		-44.1***		-42***
			(9.05)		(9.35)
	Constant		11***		11***
			(2.21)		(2.27)
First-stage F(2,148)			12.04***		
First-stage F(2,147)					10.5***
2nd Stage					
Mortality	Expected deaths			0.943***	0.913***
				(0.0228)	(0.0398)
	IMD2007	0.91***	1.34***	6.7***	11***
		(0.0981)	(0.166)	(1.33)	(1.86)
	Cancer spending	-0.327	-7.05***	0.63	-84.8***
		(0.484)	(2.36)	(7.29)	(25.6)
	Constant	83.7***	130***	-110	562**
		(3.56)	(17.67)	(68.3)	(233)
F(2,149)		52.7***	37.4***		
F(3,148)				683***	353***
Hansen J $\chi^2_{(1)}$			1.77		0.388
Kleibergen-Paap $\chi^2_{(2)}$			16.5***		16***
Endogeneity test $\chi^2_{(1)}$		8.3***		17.3***	

Table 3.4: Results from Cancer model with population weights; heteroscedasticitic-robust errors displayed in parentheses. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=152), population weights used

Table 3.4 shows the results for the cancer programme of care. The Ordinary Least Squares (OLS) results assume exogeneity of the regressors. The 2SLS IV model uses as first stage instruments the spending on other programmes and the percentage of unpaid carers in

the PCT to derive predicted values for cancer spending free from endogeneity. The first specification estimates the basic model, regressing need and spending on cancer SMR. The second regresses need, spending and the expected number of deaths on the observed number of deaths, allowing the expected mortality based on gender and age to vary.

It is clear that for each specification, 2SLS provides very different estimates for the coefficient on cancer spending compared with OLS, going from insignificantly different from zero to significantly negative. This confirms previous results in the literature. Further, in both mortality specifications there is evidence that endogeneity is present and instrumental variables are required. In both specifications the Hansen J statistic does not reject the null hypothesis that the instruments are valid, the Kleibergen-Paap rejects the null hypothesis that the equation is under-identified and the first stage F statistic exceeds 10, which indicates the instruments are not weak.

The coefficients on the instruments conform to expectation, with increased non-programme spending indicating slightly more money available for cancer spending, and increased unpaid carers reducing the amount spent on cancer care. The non-programme spending fails to attain significance, though the large F statistic supports the joint significance of the instruments.

Deprivation increases mortality in every specification. The coefficient on expected mortality is close to one, as would be expected in a standardising variable combining sex and age. This means that an extra expected death generally leads to an extra observed death. Importantly, the significantly negative coefficients on spending show that health care reduces cancer mortality. The 2SLS results suggest that for a PCT to cut funding of cancer treatment from £8690 to £8590 per patient, an extra 8.5 people would die every year, increasing the number of cancer deaths from 847 to 856.

Table 3.5 shows the results for the CHD programme of care. The IV model uses as first stage instruments the proportion on Disability Living Allowance and the percentage of

3.3 The effect of spending

	Variable	SMR		Deaths	
1st Stage		OLS	2SLS	OLS	2SLS
Prog. spending	Expected deaths				-0.00025***
					(0.0000591)
	IMD2007		-0.00653		-0.00648
			(0.00489)		(0.00484)
	Unpaid carers		-7.12***		-8.04***
			(2.23)		(2.03)
	DLA claimants		-4.13		0.277
			(3.17)		(3.33)
	Constant		2.31***		2.25***
			(0.305)		(0.305)
First-stage F(2,148)			9.49***		
First-stage F(2,147)					9.16***
2nd Stage					
Mortality	Expected deaths			0.895***	0.802***
				(0.0347)	(0.047)
	IMD2007	1.19***	1.14***	4.38***	1.65
		(0.0993)	(0.141)	(0.684)	(1.49)
	CHD spending	-6.69**	-46.3***	-61.7***	-394***
		(3.26)	(12.9)	(20.1)	(105)
	Constant	83.2***	131***	24.8	534***
		(4.47)	(15.5)	(39.8)	(169)
F(2,149)		76.6***	38.9***		
F(3,148)				245***	310***
Hansen J $\chi^2_{(1)}$			0.016		0.028
Kleibergen-Paap $\chi^2_{(2)}$			13.2***		13***
Endogeneity test $\chi^2_{(1)}$		13.4***		17.3***	

Table 3.5: Results from CHD model with population weights; heteroscedasticitic-robust errors displayed in parentheses. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=152), population weights used

unpaid carers in the PCT to derive predicted values for CHD spending free from endogeneity.

The results from OLS and 2SLS are generally closer for the CHD programme than for the cancer programme, but as before there is evidence of endogeneity in both specifica-

tions, which implies instrumental variables are required. Again, in both specifications the Hansen J statistic does not reject the null hypothesis that the instruments are valid, the Kleibergen-Paap test rejects the null hypothesis that the equation is under-identified and the first stage F statistic nearly exceeds 10, indicating the instruments are jointly significant.

Of the instruments, the percentage of unpaid carers conforms to expectations, significantly reducing CHD spending. Unfortunately the DLA variable is not statistically different from zero.

Deprivation increases mortality in every specification, though in the instrumented regression on CHD deaths it is not statistically different from zero. The coefficient on expected mortality is close to one. Importantly, the significantly negative coefficients on spending show that health care reduces CHD mortality. The effect is greater than for cancer, with a cut of £100 per patient from £1200 to £1100 leading to 31.4 extra deaths per year.

3.4 The effect of spending on inequality

The results so far are consistent with previous studies. We now extend the analysis to investigate the impact of health spending on socioeconomic-related health inequality.

As O'Donnell et al. [52] explain, the CI can be decomposed into the inequality contribution of various factors, based on a model that determines the value of y, as the previous section has done. The contribution of a variable v to the CI of y against deprivation rank (CI_{yr}) is the product of vs elasticity e with respect to y (e_{vy}) and the CI of v against deprivation rank (CI_{vr}). A well-performing regression model estimating y ought to leave almost zero inequality left to be explained by the residual effect of the error term (GC_{ω}). The decomposition is based on the regression model in equation 1.1, and decomposes the inequality in deaths into contributions of demographics, health spending and deprivation:

$$CI_{yr} = e_{Ay}CI_{Ar} + e_{Ny}CI_{Nr} + e_{\hat{X}y}CI_{\hat{X}r} + GC_{\omega} \tag{3.8}$$

where CI_{yr} is the concentration index of the health variable, e_{Ay}, e_{Ny} and $e_{\hat{X}y}$ are the elasticities of expected deaths, deprivation and spending, CI_{Ar}, CI_{Nr} and $CI_{\hat{X}r}$ are the concentration indices for expected deaths, deprivation and spending, and GC_{ω} is the concentration of the residual error term.

It is important to note that the health variable used in the analysis must be meaningfully summable. Using ratio variables like SMRs is inappropriate because they cannot be summed to produce a national mortality figure. To illustrate, areas with values 0.75 and 1.25 when combined into a single area do not produce a combined SMR equal to 2. For this reason, in the previous section the numerator and denominator of the SMR are split into the actual number of deaths (the dependent variable) and the number of deaths expected based on age and gender (the demographic covariate controlled for in the health equation). We only wish to control for demographic factors so we simply add this variable to the right hand side of our regression Equation 1.1. In the decomposition analysis we wish to explain the contribution of expenditure to age- and sex-standardised health inequality, and to calculate this we subtract the contribution of the demographic factors, to calculate the following:

$$CI_{standardised} = (CI_{yr} - e_{Ay}CI_{Ar}) \tag{3.9}$$

$$= e_{Ny}CI_{Nr} + e_{\hat{X}y}CI_{\hat{X}r} + GC_{\omega} \tag{3.10}$$

Table 3.6 shows the CI decomposition results for the cancer deaths model from Table 3.4. The concentration index for (unstandardised) cancer deaths is significant and positive (0.182): deaths are concentrated in the less deprived areas. The other concentration

Variable	Elasticity	CI	Contribution(Elasticity × CI)
Cancer deaths		0.182***	
		(0.036)	
Expected deaths	0.926***	0.227***	0.211***
	(0.0346)	(0.0421)	(0.0316)
IMD2007	0.226***	-0.237***	-0.0537***
	(0.0386)	(0.0227)	(0.0103)
Programme spending	-0.664***	-0.0411***	0.0273**
(per affected person)	(0.212)	(0.0106)	(0.011)
Generalised CI of error			-0.00225
			(0.00167)
Standardised CI		-0.0287***	
		(0.00901)	

Table 3.6: Results from Cancer decomposition; all standard errors are bootstrapped based on 2000 replications. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=152)

Variable	Elasticity	CI	Contribution(Elasticity × CI)
CHD deaths		0.167***	
		(0.0341)	
Expected deaths	0.82***	0.232***	0.19***
	(0.0481)	(0.0389)	(0.0295)
IMD2007	0.0617	-0.237***	-0.0146
	(0.0625)	(0.0229)	(0.0144)
Sub-programme spending	-0.763***	-0.00948	-0.00724
(per affected person)	(0.217)	(0.0108)	(0.00852)
Generalised CI of error			-0.0034
			(0.00839)
Standardised CI		-0.0233*	
		(0.0121)	

Table 3.7: Results from CHD decomposition; all standard errors are bootstrapped based on 2000 replications. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=152)

indices accord with expectations: expected deaths is pro-rich (this is plausible as expected mortality is driven by the age distribution) and deprivation is pro-poor. Cancer spending per patient is pro-poor, which when considered against the earlier finding that

cancer spending per person is pro-rich indicates that the nominal prevalence of cancer is higher in less deprived areas. The 2SLS regression provides the elasticities required for the decomposition analysis: mortality rises almost at parity with expected deaths, rises with deprivation, and falls with cancer spending. Appropriate standardisation of cancer mortality, by subtracting the contribution of expected mortality, leads to a pro-poor concentration of cancer deaths (-0.0287). In other words, when age and sex are taken into account cancer deaths fall disproportionately on the poor. The concentration curves which form this relationship can be seen in Figure 3.2.

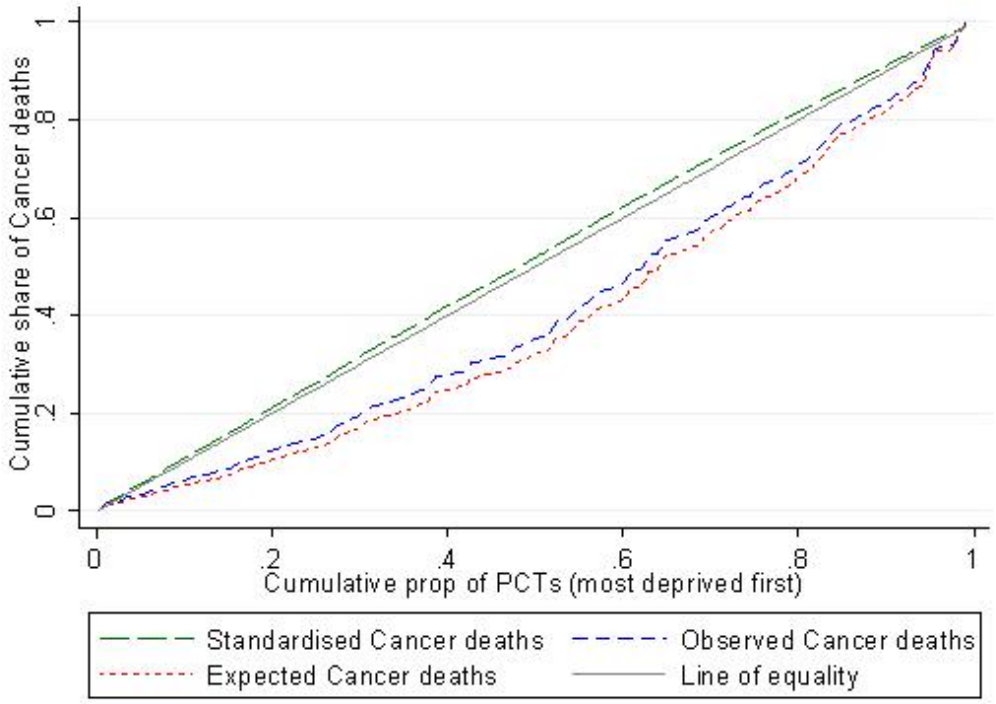

Figure 3.2: Cancer mortality concentration curves

The generalised concentration index of errors is insignificantly different from zero, indicating most of the inequality is explained by the model. The contribution of spending to inequality is positive and significant: the spending makes cancer deaths less pro-poor. This is because spending reduces mortality and spending on cancer patients is concentrated in more deprived areas.

3.4 The effect of spending on inequality

Table 3.7 shows the CI decomposition results for the CHD model from Table 3.5. Again, the concentration index for CHD mortality is significant and positive: expected deaths is pro-rich, deprivation is pro-poor. Spending, however, is neither pro-poor nor pro-rich. The elasticities, derived from the 2SLS regression, show deaths rising almost at parity with expected deaths, falling with CHD spending, but fairly unaffected by deprivation. Appropriate standardisation of CHD mortality, by subtracting the contribution of expected deaths, leads to pro-poor mortality. The corresponding concentration curves are displayed in Figure 3.3.

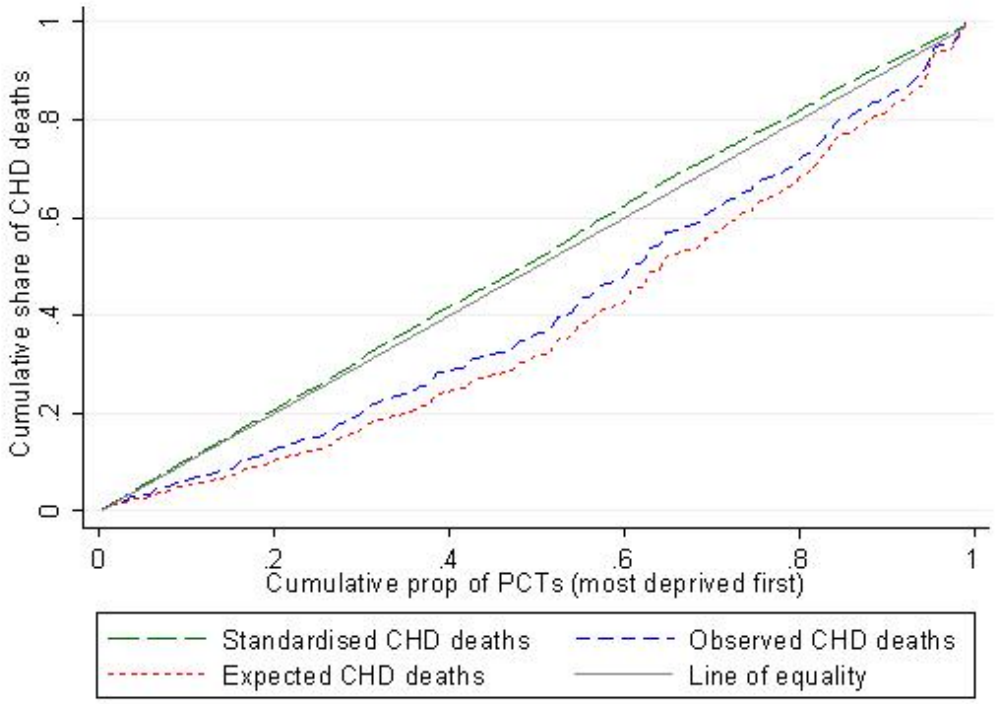

Figure 3.3: CHD mortality concentration curves

The generalised concentration index of errors is significantly different from zero as measured by its bootstrapped standard error. The actual value for the residual inequality is small at 0.003, less than 2% of the dependent variable. Though statistically significant the value is so small it does not raise questions as to the ability of the model to explain the inequality in CHD mortality. There is no real contribution of spending to inequality

either way. This is because, though effective, CHD spending is evenly distributed between rich and poor.

3.5 Concluding remarks

This analysis shows that there are significant differences in spending patterns between PCTs in rich and poor areas. There is a real benefit to health from health care spending across the cancer and CHD disease programmes of care. We describe how the distribution of health care funding across PCTs in areas of affluence and poverty affects the inequality in health outcomes in England within two key areas of NHS spending.

The model for cancer largely supports the results of Martin et al's work. The elasticity of expenditure on mortality of -0.66 is close to the original paper's value of -0.491. It is likely that the small difference between these estimates is due to either the transformation of expenditure from spending per person to spending per affected person, or a change over time in the elasticity of spending.

The CHD model shows that the elasticity of CHD mortality with respect to CHD expenditure is approximately equal to the corresponding effect in the cancer model, and their 95% confidence intervals overlap substantially. This is an interesting result. It suggests that if PCT budgets were cut by ρ%, and each programme and sub-programme were in turn cut by ρ%, the effect on cancer mortality and the effect on CHD mortality would be equivalent (an increase of around 0.645 ρ% for both programmes' mortality).

Though the results are plausible and pass appropriate statistical tests, some caveats should be noted. Firstly, these results are only partial. The Cancer and CHD programmes account for only 6.2% and 2.8% of PCT spending respectively. This analysis looks at the effectiveness of a fairly small proportion of the NHS budget. Furthermore, due to the relatively recent availability of the data it is not possible to take into account health

interventions with a longer term effect on health, such as public health interventions. Similarly, the health measure is partial: without good area-level health-related quality of life data the effect spending has on quality of life will be missed. This is why cancer and CHD, with easily measurable and relatively immediate health outcomes, were suitable for this analysis.

Furthermore, it is important to note the limitations of the estimates derived. The elasticities are point estimates and the effect of a change outside the variability of the data is invalid e.g. a 100% increase in funding will not reduce cancer mortality by 66%. Similarly the estimates from the regressions on which they are based are local average treatment effects, and hence reflect the impact of variations in spending brought about by the instruments. The standard deviation of the predicted cancer spending variable is £781 and for CHD is £139.

It is interesting to note how the CI of the programme budgets change when moving from a per capita unit to a per affect patient unit. For cancer and CHD, the per capita spending CI was 0.026 and 0.00461 respectively and the per affected person spending CI was -0.0411 and -0.00948. Though there is more money for treatment in richer areas, due to lower prevalence more money is spent per person with the disease in poorer areas.

There are also some concerns about the performance of the CHD model with regards to the lack of statistical significance of the deprivation variable in the final specification. What is particularly odd is that either not using instruments for spending, or replacing the observed and expected mortality combination with a simple SMR, yields highly significant positive results, as we would expect to find. The point estimate itself is of the sign and approximate size we would expect, yet the standard error is far larger.

Interestingly, within the cancer analysis the contribution of spending to inequality is approximately equal, but of the opposite sign, to that of standardised mortality. This suggests that if the dispersion of health spending per patient across PCTs were to be

instantaneously equalised and the efficacy of spending were unaffected, the standardised concentration index of cancer mortality would be twice as high. In other words the distribution of cancer spending approximately halves the inequality of cancer mortality.

Within the CHD model, however, spending is largely even between rich and poor areas. This indicates that the increased overall funding of poorer PCTs does not translate to correspondingly higher spending on CHD. This is possibly due to the relatively older age of CHD deaths: 69% are over the age of 75, as opposed to 52% of cancer deaths, this means that ceteris paribus areas with a higher life expectancy (which are probably richer areas) would find CHD a relatively more pressing concern.

Our analysis suggests that reductions in cancer and CHD spending will increase cancer and CHD deaths. However, the consequences of changes in health care funding on socioeconomic-related inequality in mortality are more difficult to predict than those on the level of mortality. Clearly, assuming the effect of health spending on health remains the same, the consequence of a reduction in health spending will be determined by the value of the concentration index of programme spending. But this value reflects the aggregation of the decisions PCTs will have to make in such an event, against various clinical, political and public views and furthermore including large programmes of care not under consideration in this paper. Whether reductions in health care spending will affect the inequality in mortality is further complicated by the possibility that those reductions are unlikely themselves to be proportional across disease programmes.

Overall, the results in this analysis reinforce and expand on previous research. The use of concentration indices to analyse the effects of spending on health allows a better insight to be gained as to the way national resources are being used to improve population health. Though this technique is currently restricted to easily measurable budget programmes that map well to health outcomes, improvements in data collection may allow the whole range of conditions to be modeled.

Chapter 4

On area-level panel data inequality

4.1 Introduction

In this chapter we shall explore how inequality and its determinants may be modelled over time with area-level data, using Concentration Index (CI) decomposition methods to separate long term effects from short term ones. The aim is to provide and test a suitable technique to use on a panel data version of the analysis in the previous chapter, which looked at the effects of health spending, deprivation and age and sex profile on mortality at the PCT area level. As the panel dataset will be at the area level, the preferred econometric estimation technique is likely to be Fixed Effects [72] as this removes endogeneity due to unobserved time-invariant heterogeneity. This is a particular concern in area-level analyses as the differences between two areas are typically greater and more complex to measure than the differences between two individuals.

Firstly, the existing literature regarding Concentration Index analysis in panel data contexts will be surveyed. Secondly, methods based in well-established panel data econometric techniques will be explored. Finally, we shall compare these different ways of measuring panel data inequality using an international panel dataset that attempts to model the effect of a range of determinants on the number of deaths in a country.

4.2 Existing methods to estimate inequality over time

Within the health economics literature there have been relatively few papers using longitudinal individual-level data that have considered inequality over time. One option is to treat the data as T separate datasets [70], where T is the number of periods. Standard OLS-based CI decomposition can then be used and both inequality and its determinants can be studied at the T points in time. A drawback of this approach is examined in Jones and Nicolás' [39] 2004 paper which showed that changes in ranking over time would be masked, because the ranking variable is recalculated each period. Instead Jones and Nicolás describe a technique that compares the short-term CIs with the CI of average health and average income across all periods to produce a health-related income mobility index (Equation 4.1), which indicates the bias as a result of short-term health being systematically related to long-term income ranking (or as the authors put it, individuals being upwardly or downwardly mobile).

$$M^T = 1 - \frac{CI^T}{N \sum_{t=1}^{T} \bar{y}^t CI^t} \tag{4.1}$$

where CI^T is the CI based on time-averaged health and ranked by time-averaged income, CI^t is the CI of health in period t, N is the number of individuals, T is the total number of periods, and $\bar{y}^t$ is the average health across all individuals within period t. If individuals who are downwardly mobile in terms of income rank have relatively poorer health, or those who are upwardly mobile have relatively better health, the index will be negative, meaning short-term CIs underestimate the long-term picture of inequality.

Further, the mobility index can be decomposed into contributions of different determinants, based on a regression model. For regression model

$$y_{it} = \alpha + \sum_{k=1}^{K} \beta_k x_{itk} + u_{it} \tag{4.2}$$

they show that the mobility index of y can be decomposed into the mobility indices of x by

$$M_y^T = \sum_{k=1}^{K} \beta_k \frac{\sum_{t=1}^{T} \bar{x}_k^t CI_{x_k}^t}{\sum_{t=1}^{T} \bar{y}^t CI^t} M_{x_k}^T + residual \tag{4.3}$$

In words, the mobility index of y is the weighted sum of the mobility indices of x_k, with weights set as the elasticities of y with respect to x_k evaluated at the inequality-weighted means of $\bar{y}$ and $\bar{x}_k$. This formula also provides the means to standardise the health-related mobility index by including standardising covariates and subtracting their effect from M_y^T.

This is applied to a panel dataset from the British Household Panel Survey, using a simple OLS regression to model how the Generalised Health Questionaire measure of psychological well-being is effected by a range of covariates. The mobility index has subsequently been applied to other individual level analyses [6], [17], [42]. This technique provides a powerful method to analyse inequality in individual longitudinal studies, particularly as the model does not assume a co-instantaneous relationship between income and health.

We note that the dynamic aspect of the relationship is embedded in the health-related income mobility index, with changes in health, income rank and (in the case of the decomposition) regressors being modelled as the ratio between average CI and short term CI. The use of fixed effects (α) in this context might therefore overspecify the dynamic aspect of the model, as changes in variables over time would be modelled on both the inequality and deterministic sides of the formula. The decomposition

$$M_y^T = \frac{\sum_{t=1}^{T} \alpha^t CI_\alpha^t}{\sum_{t=1}^{T} \bar{y}^t CI^t} M_\alpha^T + \sum_{k=1}^{K} \beta_k \frac{\sum_{t=1}^{T} \bar{x}_k^t CI_{x_k}^t}{\sum_{t=1}^{T} \bar{y}^t CI^t} M_{x_k}^T + residual \tag{4.4}$$

would now include a new term, the mobility index of fixed effects, which measures the relationship between changes in income rank and the time-invariant portion of y. Problematically, this would preclude standardising for characteristics that do not vary over time (e.g. gender), and make the standardising for any characteristics more difficult the less time-varying they are (e.g. population).

Two papers have used Fixed Effects within a Concentration Index decomposition. Wildman [70] uses a FE model of mental health to decompose mental health inequality in Great Britain into its contributions using BHPS data. The health regression is

$$H_{it} = \mu_t + \alpha_i + \beta' X_{it} + \epsilon_{it} \tag{4.5}$$

where H is health, μ is a mean intercept, α are the fixed effects (which in this case are deviations from the mean intercept, μ), β are coefficients of the covariants in X and ϵ is the error term. This model is then used in the decomposition

$$CI_H = \sum_{l=1}^{T} \left(\frac{\beta_l \bar{X}_l}{\bar{H}} \right) CI_{X_l} + \left(\frac{\bar{\epsilon}}{\bar{H}} \right) CI_\epsilon \tag{4.6}$$

Thus, although FE estimation is used, the fixed effects themselves do not contribute to the decomposition, other than through their use in the estimation of β.

To our knowledge only Islam et al's 2010 paper [38] has utilised the fixed effects in the subsequent decomposition. In this paper a FE model is used on an individual level unbal-

anced panel dataset with three time periods at intervals of 8 years, covering a sample of 16-84 year-old Swedes. Health state scores are regressed on age, unemployment rate and GDP growth, income, education, marital status, number of offspring, activity status and immigration status. The effects of the covariates and the fixed effects on health were then applied to the three separate periods' CI decompositions by an extended decomposition formula:

$$CI^t = \left(\frac{\alpha_t}{\bar{H}^t}\right) CI_\alpha^t + \sum_k \left(\frac{\beta_k \bar{X}_k^t}{\bar{H}^t}\right) CI_X^t k + \frac{GC_\epsilon^t}{\bar{H}^T} \tag{4.7}$$

which decomposes the CI at time t into the contribution of the fixed effects, α, the contributions of covariates, X, and the contribution from the residuals, ϵ. To check whether re-ranking of income changed the results, the analysis was also run using the rank of mean income.

Importantly, the fixed effects contribution dominated the decomposition of health inequality. In the first period the fixed effects explained 99% of the CI of health, in the second period this fell to 83%, and in the third to 59%. These contributions were far in excess of the next biggest contribution in each case; in the first two periods the next biggest contributor was the error term at -25% and 11%, in the last period this was the combination of the age dummies at 17%. The fixed effects take the place of all time-invariant heterogeneity in the model, so this means that time-invariant factors are responsible for the bulk of the estimated inequality in health. So, though FE provides a consistent method to estimate the elasticities of the variables in the regression, this comes at the cost of a large part of the inequality being effectively unexplained.

4.3 Fixed Effect Re-Decomposition

In this section we propose a method to measure the relationship between health inequality and its determinants in a panel dataset. Our method is similar to Islam et al's decomposition, in that we include the fixed effects as a source on inequality, but it extends the analysis to determine where the fixed effect's inequality comes from.

We begin by considering how relationships can be modelled in panel datasets. There are four common regression techniques used to model panel data.

The simplest is Ordinary Least Squares. This treats any two observations for any individual and any time as independent from one another. No account is taken of any correlation between two observations taken at the same time, or two observations from the same person.

$$\begin{aligned} y_{it} &= \alpha + x'_{it}\beta + \epsilon_{it} \\ \epsilon_{it} &\sim N(0, \sigma^2) \end{aligned} \tag{4.8}$$

β can then be estimated by $\hat{\beta}$

$$\hat{\beta} = (X'X)^{-1}X'Y$$

Between Effects (BE) averages the data across all the time periods, for each individual. The resulting regression deals with the long-term level of the variables. Mathematically, the model is:

$$\begin{aligned} \bar{y}_i &= \alpha + \bar{x}_i'\beta + \bar{\varepsilon}_i \\ \bar{\varepsilon}_i &\sim N(0, \sigma^2) \end{aligned} \tag{4.9}$$

The variable β can then be estimated by OLS

$$\hat{\beta}_{BE} = (\bar{X}'\bar{X})^{-1}\bar{X}'\bar{Y}$$

where $\bar{X}$ and $\bar{Y}$ are the average over time for each individual.

The Random Effects (RE) models the heterogeneity between individuals as an extra error term, with variance that varies between individuals but not over time.

$$\begin{aligned} y_{it} &= \alpha + x_{it}'\beta + u_i + \varepsilon_{it} \\ \varepsilon_{it} &\sim N(0, \sigma_{it}^2) \\ u_i &\sim N(0, \eta_i^2) \end{aligned} \tag{4.10}$$

In this case β can be estimated by generalised least squares (GLS) if the error term is uncorrelated with the covariates.

$$\hat{\beta}_{RE} = (X'\Omega^{-1}X)^{-1}(X'\Omega^{-1}Y)$$

$$\hat{\Omega} = I \otimes \Sigma$$

where Σ is the covariance matrix.

If the error term is correlated with the covariates, RE is biased. Normally Fixed Effects (FE) is preferred in this case. FE fits a model with a different constant term for each individual.

4.3 Fixed Effect Re-Decomposition

$$y_{it} = \alpha_i + x'_{it}\beta + \varepsilon_{it} \tag{4.11}$$

The Fixed Effects technique is typically estimated by subtracting the mean over time for each variable by each individual, known as the within-group estimation. The estimator is

$$\hat{\beta}_{FE} = [(X - \bar{X})'(X - \bar{X})]^{-1}(X - \bar{X})'(Y - \bar{Y})$$

OLS is not widely used, as its assumption of independence within the observations for an individual and within the observations within a time period are rarely satisfied. If OLS is used when this assumption is not valid the estimates for β will be biased.

By taking the time-average of all observations, the BE strips the dataset of all the variation across the time access, compressing the observations by a factor of T. This would be an appropriate starting point if the aim was to estimate long term inequality in health. However, as all the time-varying information is averaged, we would be ignoring all the information provided in the dataset on the effect of changes in variables on changes in health.

The RE is the efficent estimator of a panel data process, when its assumptions are valid. Unfortunately, the assumption that u_i is uncorrelated with x_{it} is very strong, and in the case of area-level data is not plausible. In particular, Wooldridge [72] argues that in panel datasets where the cross-sectional units are large areas it is not possible to treat the observations as a random sample from a bigger population, and that then fixed effects should be used. In the example at the end of this chapter and for the setting of the previous chapter RE is invalid, and to use it would result in biased estimates.

We turn then to FE. This has the attractive feature that the model to determine health can be thought of as partitioning long term, time-invarying, unobserved factors from short-term, time-varying observed factors automatically.

4.3 Fixed Effect Re-Decomposition

To apply the O'Donnell et al [52] method to decompose inequality into the weighted sum of the determinants' inequality, we treat the individual fixed effects as a single variable with coefficient equal to one:

$$CI_y = e_\alpha CI_\alpha + \sum e_x CI_x + GCI_\varepsilon \tag{4.12}$$

with weights set as the partial elasticities based on the FE estimates, calculated by

$$e_x = \frac{\hat{\beta}_{FE}\bar{x}}{\mu_y} = \frac{[(X-\bar{X})'(X-\bar{X})]^{-1}(X-\bar{X})'(Y-\bar{Y})\bar{x}}{\mu_y} \tag{4.13}$$

Note that instead of measuring how much a generic change in x effects a change in y, the FE elasticities measure how much a change over time in x effects a change in y within the same cross-sectional unit. The information the data contains on the effect of differences in the level of x between individuals (which can be some of the larger differences in the observations of x) is lost when the variables are demeaned. In some cases this is appropriate (if we were to ask "how can we reduce the number of deaths within countries?" instead of "what affects the inequality of deaths between countries?"), but in our case we need to find a way to exploit the information contained in the long-term differences between countries.

The adjustment we propose is to extract the fixed effects from the model and re-decompose them into the time-averaged covariates, as well as other time-invariant variables excluded by the fixed effects that we suspect affect the principal variable in question. The reason for this is that whatever elasticity is being lost through the within-group transformation of the variables is contained, along with time invariant heterogeneity, 'in' the fixed effects. The time invariant heterogeneity cannot be fully specified (if it were, an RE-like technique could be used) but if time invariant variables z are available and suspected (or indicated by

the literature) to be determinants of y they can be included. Clearly, the re-decomposition is not perfect as not all the heterogeneity is observed. For this reason the residual of the auxiliary regression, GCI_υ, will be quite large, and can be thought of as the residual of the parts of the fixed effects we are unable to model.

The auxiliary regression is therefore a regression of the extracted fixed effects (α_i from Equation 4.11) on the averages over time of the covariates x and other time-invariant variables z given by

$$\alpha_i = a + \bar{x}_i'\gamma + z_i'\psi + \upsilon_i \tag{4.14}$$

where a is a constant, υ_i is an error term and γ and ψ are parameter vectors to be estimated.

The re-decomposition measures how the fixed effects' CI is influenced by time-averaged x and other time-invariant covariants, such that

$$CI_\alpha = \sum \epsilon_{\bar{x}} CI_{\bar{x}} + \sum \epsilon_z CI_z + GCI_\upsilon \tag{4.15}$$

Combining (4.12) and (4.15) provides an overall decomposition equation.

$$\begin{aligned} CI_y =& e_\alpha \left(\sum \epsilon_{\bar{x}} CI_{\bar{x}} + \sum \epsilon_z CI_z + GCI_\upsilon \right) + \sum e_x CI_x + GCI_\varepsilon \\ =& \frac{\bar{\alpha}}{\mu_y} \sum \left(\hat{\gamma} \frac{\bar{x}}{\bar{\alpha}} CI_{\bar{x}} + \hat{\psi} \frac{\bar{z}}{\bar{\alpha}} CI_z + GCI_\upsilon \right) + \sum \hat{\beta} \frac{\bar{x}}{\mu_y} CI_x + GCI_\varepsilon \\ =& \sum \frac{\bar{x}}{\mu_y} \left[\hat{\beta} CI_x + \hat{\gamma} CI_{\bar{x}} \right] + \sum \frac{\bar{z}}{\mu_y} \hat{\psi} CI_z + \frac{\bar{\alpha}}{\mu_y} GCI_\upsilon + GCI_\varepsilon \end{aligned} \tag{4.16}$$

As Equation 4.16 shows, the overall decomposition can be rearranged into contributions from the x variables, the z variables and the two error terms. The contribution of x to CI_y is a combination of the contribution from the within-group regression ($\hat{\beta}CI_x$) and the contribution of the underlying regression using time-averaged variables ($\hat{\gamma}CI_{\bar{x}}$).

Extracting and analysing the fixed effects is not common in the economics literature, but is not unknown. Sastry et al [61] extract and regress the fixed effects of a household-level study to analyse the effects of area-level covariates on notions of community. Similarly, Hail and Leuz [33] use the country-level fixed effects of a firm-level regression to find out how country-level institutions affect the cost of equity capital. Both of these papers regress extracted fixed effects in order to find out more about the models they are using; we extend this in order to also find out more about the inequality in the models we are using.

4.4 Demonstration of FE-RD

4.4.1 Model

To demonstrate the re-decomposition technique on a Fixed Effects model, we sought a simple model that would not require instrumental variables. The literature on the determination of health at the country level provided such an example, with both Pritchett and Summers [59] and Filmer and Pritchett [30] running international models on mortality and finding no evidence of endogeneity.

In "Wealthier is Healthier"[59], Pritchett and Summers attempt to determine the effect GDP has on health. They run a regression of infant mortality on GDP per capita, years of schooling, time dummies and fixed effects. Importantly, they use a range of instruments to check for the possibility of endogeneity between GDP and health. They find no substantial evidence for reverse causation between the two primary variables of interest, and the strong

correlation between health and GDP seems to be due to wealth causing health.

In "Child mortality and public spending on health : how much does money matter?"[30] Filmer and Pritchett focus on under-5 mortality and build a larger model of national health. They regress under-5 mortality on a range of covariates: GDP per capita, Government health spending as a share of GDP, female education, income inequality, religion, ethno-linguistic fractionalisation, urbanisation, access to safe water and whether the country is in a tropical area. The potential endogeneity between health and health spending is checked using instruments, namely neighbouring countries' health and defense spending, history of independence and a dummy indicating whether the country's main export is oil. Using the instruments does not substantially change the results, which indicates endogeneity is not a problem.

We therefore collected an international dataset from the World Bank website [9], spanning three five-year periods between 1994 and 2009 and 215 countries. Within each five-year period the averages over the five years were taken for each variable. This was done to maximise the sample size, as many less developed countries had infrequent data records. The variables were: population; number of deaths per thousand; GDP in $ per capita; Government health spending (GHS) in $ per capita; Private health spending (PHS) in $ per capita; the proportion of people under the age of 15; the proportion of people over the age of 65; the proportion of people with access to improved sanitation facilities; the proportion of people living in an urban area; and the proportion of people with HIV. The 215 countries yield 607 observations, meaning that on average each country has 2.82 observations. All monetary variables were in terms of 2010 $.

4.4.2 Methods

We run a fixed effect model of deaths per thousand against the full set of covariates, weighting by population size and using heteroscedastic-robust standard errors.

4.4 Demonstration of FE-RD

Potential issues of endogeneity between health and GDP, and health and health spending, are checked with the use of instrumental variables. The instruments used are broadly the same as those in the papers by Pritchett and Summers and Filmer and Pritchett. To test endogeneity between health and health spending we use neighbouring countries' Government health spending and defense spending per capita, with neighbours defined by a shared land border. These are suitable instruments as it is implausible they have a direct effect on a country's health, but will effect the Government's decisions as to how much to spend on health and defense. For the potential endogeneity between health and GDP we use the ratio of the official exchange rate to the purchasing power parity exchange rate, which was noted in Dollar [23] as an indicator of outward orientation and therefore growth, but should not itself affect mortality. The instruments are checked with an IV fixed effects regression, using population weights.

We then apply our Fixed Effect Re-Decomposition technique to the data. We report the initial decomposition, extract the fixed effects and run the re-decomposition. Finally, we report the combined results of both stages.

All the data are reported in nominal values in Table 4.1. In the subsequent analysis all variables are converted into per capita values. It is important to note the extent of variability in the dataset, both in terms of the variation in figures between the smallest and largest countries, and in terms of the number of observations provided. This is a characteristic of worldwide country-level datasets.

Variable	Mean	Std. Dev.	Min	Max	Observations
Deaths	261163	894665	126	9115997	607
GDP ($m)	200000	913000	66.5	13700000	592
Government health spending ($m)	11800	62200	1.1	977000	561
Private health spending ($m)	8120	64800	0.00036	1100000	561
Under 15	9638390	35200000	19612	362000000	569
Adult	20800000	81500000	54015	938000000	569
Over 65	2300231	8243645	3884	103000000	569
Sanitation	18900000	57900000	7474	710000000	542
Urban	14300000	45500000	4996	556000000	621
HIV positive	319640	905447	258	8676295	436
Total neighbours' GHS ($m)	52900	119000	29.7	978000	465
Average neighbours' GHS ($m)	20100	67800	14.9	977000	468
Total neighbours' Defense spending ($m)	2490000	5310000	49.6	57300000	462
Average neighbours' Defense spending ($m)	1040000	3680000	49.6	57300000	465
PPP ratio	0.561	0.252	0.16	1.5	544

Table 4.1: Summary statistics

4.4.3 Results

Table 4.2 shows the results for the FE model when GDP and GHS are instrumented by 2SLS, and when they are not. The signs of the coefficients accord with expectations, however the lack of general significance is surprising. It should be noted that, as explored in the Section 4.3, the fixed effect estimates measure the effect of a change in a variable on the change in deaths; the effects of healthcare, sanitation and wealth may take longer to influence the rate of mortality.

Regarding the IV estimates, the Sargan test is passed which confirms that the instruments are valid, and the first stage F tests are significant and greater than 10, indicating appropriate strength of the instruments. The similarity between the estimates suggests that endogeneity is not a concern, confirming the results of Pritchett and Summers [59] as well as Filmer and Pritchett [30]. Furthermore the statistical test of endogeneity rejects the need for instrumental variables at the 5% level.

Details of the first stage of the IV regression can be found in Table 4.3. The coefficients on the instruments accord with expectations, with the purchasing power parity exchange rate being associated with higher GDP per capita, and defense and neighbours' health spending being associated with higher public sector health spending. Alternatively, there may simply not be much variation over time in the sample. However, due to the rejection of the endogeneity test, from this point we discard the need for IV estimation, and base our analysis solely on the regular FE model.

Table 4.4 shows the results of the regression of the extracted fixed effects on the time-averaged variables. In general, the coefficients are more significant. As this regression uses the fixed effects as the dependent variable, the variation that is used to estimate the coefficients is variation between countries. With regards to the variables we are using, one would expect much greater variation between countries than within countries over

	FE		IVFE	
Variable	Coef	S.E	Coef	S.E
Deaths				
GDPpc ($ m pc)	0.007	(0.045)	0.038	(0.144)
Government health spending ($ m pc)	-0.744	(0.572)	-1.06	(1.78)
Private health spending ($ m pc)	0.596**	(0.24)	0.585	(0.534)
Under 15	0.006	(0.006)	0.007	(0.007)
Over 65	0.034***	(0.008)	0.034	(0.024)
Sanitation	-0.005	(0.003)	-0.006*	(0.003)
Urban	-0.01*	(0.006)	-0.01*	(0.006)
HIV positive	0.045**	(0.018)	0.05***	(0.017)
Tests	Value	P value	Value	P value
First Stage F(3,219) on GDPpc			31.13***	<0.001
First Stage F(3,219) on Spending			14.56***	<0.001
F(8,139)	18.02***	<0.001		
Second Stage F(8,202)			11.39***	<0.001
Hansen J $\chi^2_{(1)}$			0.162	0.687
Endogeneity test $\chi^2_{(2)}$			0.138	0.933
Countries	409		113	
Observations	409		323	

Table 4.2: Regular and IV FE results. *** is significant at the 1% level; ** at the 5% level; * at the 10% level, all standard errors are hetereoscedastic-robust

15 years. Of particular interest are the significant coefficients on GDP per capita and under 15s per capita. When considering the within-country variation in Table 4.2 neither were significant; now using between-country variation both are. Richer countries have substantially lower mortality rates, while countries with overall younger populations have greater mortality rates.

Table 4.5 reports the mortality CI decomposition based on the FE regression. Inequality in mortality changes substantially when standardised for old age and time effects, from -0.055 to -0.15. This is mainly driven by the contribution of the proportion of over 65s, which means that though the death rate is nominally higher in poorer countries, when we factor in the increased mortality risks of old age and the pro-rich distribution of the

Dependent variable	GDPpc		GHS	
Variable	Coef	S.E	Coef	S.E
First Stages				
Private health spending ($ m pc)	9.99***	(1.78)	0.733***	(0.167)
Under 15	-14648*	(8450)	429	(569)
Over 65	41470	(371489)	19.57	(3027)
Sanitation	-6693*	(3925)	-440	(311)
Urban	-6900	(5909)	381	(411)
HIV positive	11614**	(5563)	150	(272)
Neighbours GHS ($ m pc)	5.26***	(0.789)	0.469***	(0.093)
Defense spending ($ m pc)	0.004	(0.026)	0.004*	(0.002)
PPP ratio	7894***	(1464)	199*	(103)
Tests	Value	P value	Value	P value
First Stage F(3,201) on GDP	31.13	<0.001		
First Stage F(3,201) on GHS			14.56***	<0.001
F(8,139)	18.02***	<0.001		
Hansen J $\chi^2_{(1)}$	0.162		(0.687)	
Endogeneity test $\chi^2_{(1)}$	0.94	0.76	0.124	0.72
Countries	113		113	
Observations	323		323	

Table 4.3: First stage results for IV fixed effects *** is significant at the 1% level; ** at the 5% level; * at the 10% level, all standard errors are hetereoscedastic-robust

aged the inequality becomes even worse for poorer countries. The power of the non-standardising variables to explain the inequality is fairly low compared with the fixed effects, which account for 117% of the standardised CI of mortality. This dwarfs the next largest contribution, that of proportion under the age of 16 at 45% . Overall this suggests that long term factors explain a significant amount of the inequality. Importantly, the contribution of GDP to the mortality CI is positive, due to its small yet positive elasticity.

	OLS	
Variable	Coef	S.E
Fixed effects		
GDP ($ m pc)	-0.221**	(0.103)
Government health spending ($ m pc)	-0.715	(1.33)
Private health spending ($ m pc)	0.956**	(0.464)
Under 15	0.0572***	(0.00733)
Over 65	0.0917***	(0.0208)
Sanitation	-0.000537	(0.00151)
Urban	0.00339	(0.00259)
HIV positive	0.0104	(0.00819)
Constant	-0.011***	(0.00333)
Tests	Value	P value
F(8,131)	74.6***	<0.001
R^2	0.75	
Observations	140	

Table 4.4: Underlying regression of fixed effects on time-averaged variables. *** is significant at the 1% level; ** at the 5% level; * at the 10% level, all standard errors are hetereoscedastic-robust

Dependent variable	Mortality CI					
	-0.055					
Demographic variables	U15		O65			
	0.067		0.108			
Breakdown	Elas	CI	Elast	CI		
	-0.518	-0.128	0.441	0.245		
Covariates	GDP		GHS		PHS	
	0.013		-0.016		0.023	
Breakdown	Elast	CI	Elast	CI	Elast	CI
	0.018	0.722	-0.02	0.803	0.029	0.776
Non variables	Fixed Effects		Period 2		Period 3	
	-0.176		0.002		-0.015	
Breakdown	Elast	CI	Elast	CI	Elast	CI
	1.463	-0.12	-0.037	-0.051	-0.074	0.203

Covariates	Sanitation		Urban		HIV	
	-0.051		-0.021		-0.013	
Breakdown	Elast	CI	Elas	CI	Elast	CI
	-0.214	0.239	-0.1	0.207	0.045	-0.286
Non variables	Residual υ					
	0.025					
Breakdown						

Standardised variable	Standardised Mortality CI
	-0.15

Table 4.5: Decomposition results

Table 4.6 reports the auxiliary decomposition of the FE CI, based on a regression of the extracted FE on the 15-year-averaged values of the same covariates. As the FE capture the time-invariant mortality levels in each country, this regression is analogous to a Between Effects regression. Here we find some of the effects we might have thought lacking in the preceding analysis: GDP per capita and public sector health spending reduce mortality, albeit health spending has a very small effect. Coupled with their pro-rich distribution this means that both contribute to the pro-poor mortality CI. Interestingly, the biggest change is for the variable measuring the proportion under 16. When averaged across the whole 15 years it still displays a pro-poor CI, but its effect on mortality is reversed, from an elasticity of -0.518 to 1.36. Bearing in mind the difference between FE and BE estimators, this may be explained in terms of an effect of a change within (FE) versus the effect of a difference between (BE). It is possible that the negative elasticity indicates that the countries in the dataset that experienced growth in the proportion of under-16s faced falling mortality, whereas the positive elasticity indicates that countries with high proportions of under-16s face high levels of mortality for long-term reasons.

Dependent variable	Fixed Effects -0.12											
Demographic variables	U15 -0.179		O65 0.131									
Breakdown	Elas 1.36	CI -0.132	Elast 0.52	CI 0.252								
Covariates	GDP -0.08		GHS -0.017		PHS 0.015		Sanitation -0.006		Urban 0.027		HIV -0.003	
Breakdown	Elast -0.113	CI 0.709	Elast -0.0214	CI 0.794	Elast 0.019	CI 0.78	Elast -0.024	CI 0.237	Elas 0.129	CI 0.206	Elast 0.008	CI -0.317
Non variables	Residual ε -0.008											

Table 4.6: Re-decomposition results

Table 4.7 reports the overall mortality decomposition when the contribution of the FE are redistributed according to the auxiliary decomposition. The large contribution of the standardising variable to the auxilary decomposition changes the standardised mortality CI from -0.15 to -0.341. Additionally, the inclusion of the auxiliary decomposition leads to a more realistic and substantial pro-poor contribution of GDP to mortality inequality of -0.104. Note that in the breakdown cells of this table, the contribution of each variable is split between the time variant (TV) contribution from the original decomposition, and a time invariant (TI) contribution from the Fixed Effects re-decomposition which is multiplied by the elasticity of the fixed effects. In this table we can see some of the reasons why re-decomposition is necessary. Though GDP leads to a slight increase in the number of deaths in the FE analysis, when the picture is broadened to include the differences between countries, wealth has the effect of reducing the number of deaths in richer countries. Similarly, the fairly low contribution of rich countries' public health spending to health inequality in the initial decomposition is more than doubled when augmented by the auxiliary contribution through the fixed effects.

Dependent variable	Mortality CI -0.055											
Demographic variables	U15 -0.196		O65 0.3									
Breakdown	TV 0.067	TI -0.263	TV 0.108	TI 0.192								
Covariates	GDP -0.104		GHS -0.041		PHS 0.044		Sanitation -0.06		Urban 0.018		HIV -0.017	
Breakdown	TV 0.013	TI -0.117	TV -0.016	TI -0.025	TV 0.023	TI 0.021	TV -0.051	TI -0.008	TV -0.021	TI 0.039	TV -0.013	TI -0.004
Non variable	Period 2 0.002		Period 3 -0.015		Residual 0.013							
Breakdown					TV 0.025	TI -0.012						
Standardised variable	Standardised Mortality CI -0.341											

Table 4.7: Combined results, contributions split into time-variant (TV) and time-invariant (TI) components

4.4.4 Conclusion

The difference between the initial decomposition and subsequent re-decomposition results indicate the extent to which the fixed effects mask the contribution of long term factors to inequality. The nesting of a time-averaged regression in a FE model of inequality allows more of the variation in the data to be used to explain the inequality, and is particularly important to determine the contributions of variables that do not vary much over the time period and are therefore subsumed into the FE. Fundamentally, the problem resides in the derivation and meaning of the elasticities, which measure the effect of a change within a group. The re-decomposition allows some of the inequality in the fixed effects to be apportioned back to the covariates. This is a useful technique when variables of interest either do not vary over time, or do not vary very much over time.

The results of this chapter find no statistically significant effect of Government health spending on mortality, upholding the result in Pritchett and Summers [59]. As discussed in Chapter 2, using international data to measure the effect of health spending is difficult, due to the large degree of heterogeneity between countries. Interestingly, private health spending is positively associated with mortality. A potential explanation of this is that higher levels of private health spending in countries is an indicator that public provision of health care is deficient (though, due to the lack of significance of Government health spending, presumably not due to lack of spending).

As mentioned in Section 4.4.3, it is informative that GDP and the proportion of the population under 15 is significant in the underlying regression but not the initial fixed effects regression. This shows that these factors have not exerted a large influence on mortality in a 15 year time span within countries, but have a larger effect when considering the greater disparities between countries. The subsequent decomposition shows that both of these factors contribute substantially to the concentration index in mortality, overwhelmingly due to the re-decomposition component of the model.

4.4 Demonstration of FE-RD

Though this chapter has demonstrated the use of fixed effects re-decomposition in the context of area-level panel data, a few assumptions and caveats should be noted.

Firstly, due to lack of data, the instrumental variable regression that checked for endogeneity uses a subset of the full dataset (323 observations instead of 409). To appeal to the findings of the endogeneity test, we are assuming that there is no substantial difference between the included and excluded observations.

Secondly, using five year averages for the data reduces the within-group variation available to fixed effects, in a sense exaggerating the relative importance of the between-country variation (and flattering the need for fixed effects re-decomposition). The reason it was important to do this was that data availability was concentrated in more developed, higher GDP, lower mortality countries. As the focus of analysis was the inequality between rich and poor countries, it was crucial to include as full a spectrum of countries as possible.

Overall, this chapter has provided a viable method to extend the analysis of Chapter 3 into a panel model.

Chapter 5

A panel data approach to area-level health inequality

5.1 Introduction

Combining the Concentration Index Redecomposition method in Chapter 4 with the econometric decomposition model developed in Chapter 3, we now move on to analyse area-level health inequality in England between 2004 and 2010. As in Chapter 3, the areas under analysis are PCTs, and our data comes from a plethora of publicly available sources.

In economics as a whole there has been a move towards using panel data. The advantage of repeatedly observing the same statistical units over time is the ability to account for unit-specific heterogeneity, as well as being able to evaluate how variables and relationships evolve over time. For this chapter, broadening the previous analysis to include observations over time gives some indication as to the stability of the relationships identified in Chapter 3.

During the period under consideration, PCT budgets rose year-on-year and mortality fell. In Figure 5.2 all-cause mortality decreased from 9.5 per 1000 to 8.5 per thousand. The main categories of mortality display a similarly downward trend. Figure 5.1 shows the

corresponding trend for spending. Though PCT spending rose more dramatically than mortality fell, the largest budget categories display a much less pronounced trend with Circulatory spending in particular remaining fairly flat over the 6 year period.

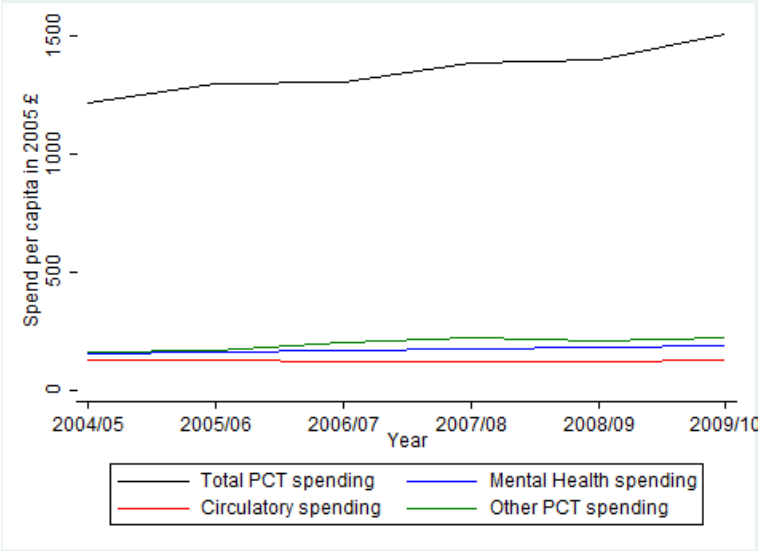

Figure 5.1: PCT spending over time

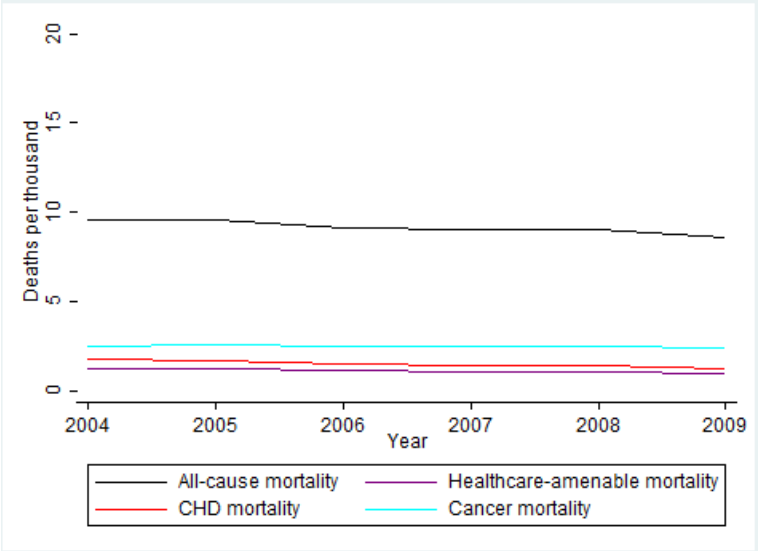

Figure 5.2: All-cause mortality over time

In combining the health model of Chapter 3 with the decomposition technique of Chapter 4, a number of changes had to be made to the data being used. These are detailed in the Methods and Data sections.

Our analysis in this chapter will focus on the relationship between mortality and health care spending at three different levels: deaths due to cancer; deaths due to Coronary Heart Disease (CHD); and deaths amenable to health care. The first of these incorporates all deaths from all cancers at any age, and the cancer spending programme (programme budget 2) provides the relevant disease-specific spending. The second of these incorporates all deaths from CHD, and the CHD spending sub-programme (programme budget 10a) provides the relevant disease-specific spending. The final level of analysis incorporates deaths from causes considered amenable to health care. From the indicator specification document:

"Causes of death are included if there is evidence they are amenable to health-care interventions – given timely, appropriate, and high quality care – death rates should be low among the age groups specified." [14]

The choice of cancer, CHD and 'amenable mortality' is designed to both allow comparison with Chapter 3 and compare the ability of area-level regression to estimate the effect of health spending on mortality at different disease levels, from the (clinically) broad to the narrow. A priori we would expect to see similar results for cancer and CHD to their Chapter 3 analogues, and we would expect accurate estimation of a model incorporating a broader base of disease to be more challenging.

5.2 Methods

As in Chapter 3, the relationship under consideration is between mortality, demographic factors, need and expenditure, modeled using 2SLS with fixed effects:

$$X_{it} = \beta_i + \delta_1 A_{it} + \delta_2 D_{it} + \delta_3 Z_{it} + \tau_{it} \tag{5.1}$$

$$y_{it} = \alpha_i + \gamma_1 A_{it} + \gamma_2 D_{it} + \gamma_3 \hat{X}_{it} + \omega_{it} \tag{5.2}$$

where y is the number of deaths, A is the number of deaths expected when applying national mortality rates for age and sex groups to the areas' specific age and sex profile, D is a measure of deprivation in the area, X is the money spent in the PCT, and τ and ω are error terms. The fixed effects are represented by β and α. The Z variables are instruments for spending and δ and γ are coefficients to be estimated.

Applying the O'Donnell et al. [52] method to decompose inequality into the weighted sum of the determinants' CIs, including the fixed effects (α_i), gives us Equation 5.3 (note given our interest in modelling the determinants of health, this is only applied to the main Equation 5.2 not the first-stage Equation 5.1).

$$CI_y = e_\alpha CI_\alpha + e_A CI_A + e_D CI_D + e_X CI_X + GCI_\omega \tag{5.3}$$

with weights set as the partial elasticities based on the 2SLS FE estimates, calculated by

$$e_x = \frac{\hat{\beta}_{2SLSFE}\bar{x}}{\mu_y} = \frac{[(X - \bar{X})'(X - \bar{X})]^{-1}(X - \bar{X})'(Y - \bar{Y})\bar{x}}{\mu_y} \tag{5.4}$$

The auxiliary regression is defined as a regression of the extracted fixed effects on the averages over time of the covariates, in Equation 5.5.

$$\alpha_i = a + \bar{A}_i'\rho_1 + \bar{D}_i'\rho_2 + \bar{X}_i'\rho_3 + \upsilon_i \tag{5.5}$$

Again, applying the O'Donnell et al. [52] method yields the decomposition:

$$CI_\alpha = \epsilon_{\bar{A}} CI_{\bar{A}} + \epsilon_{\bar{D}} CI_{\bar{D}} + \epsilon_{\bar{X}} CI_{\bar{X}} + GCI_\upsilon \tag{5.6}$$

Combining (5.3) and (5.6) provides the overall decomposition equation

$$\begin{aligned} CI_y &= e_\alpha \left(\epsilon_{\bar{A}} CI_{\bar{A}} + \epsilon_{\bar{D}} CI_{\bar{D}} + \epsilon_{\bar{X}} CI_{\bar{X}} + GCI_\upsilon\right) + e_A CI_A + e_D CI_D + e_X CI_X + GCI_\omega \\ &= \frac{\bar{\alpha}}{\mu_y}\left(\rho_1 \frac{\bar{\bar{A}}}{\bar{\alpha}} CI_{\bar{A}} + \rho_2 \frac{\bar{\bar{D}}}{\bar{\alpha}} CI_{\bar{D}} + \rho_3 \frac{\bar{\bar{X}}}{\bar{\alpha}} CI_{\bar{X}} + GCI_\upsilon\right) \\ &\quad + \gamma_1 \frac{\mu_A}{\mu_y} CI_A + \gamma_2 \frac{\mu_D}{\mu_y} CI_D + \gamma_3 \frac{\mu_X}{\mu_y} CI_X + GCI_\omega \\ &= \frac{\bar{\bar{A}}}{\mu_y}\left[\hat{\gamma_1} CI_A + \hat{\rho_1} CI_{\bar{A}}\right] + \frac{\bar{\bar{D}}}{\mu_y}\left[\hat{\gamma_2} CI_D + \hat{\rho_2} CI_{\bar{D}}\right] \\ &\quad + \frac{\bar{\bar{X}}}{\mu_y}\left[\hat{\gamma_3} CI_X + \hat{\rho_3} CI_{\bar{X}}\right] + \frac{\bar{\alpha}}{\mu_y} GCI_\upsilon + GCI_\varepsilon \end{aligned} \tag{5.7}$$

This equation shows that the covariates' contribution to inequality can be split into their time-varying and non-time-varying components. Notice that standardising CI_y for expected deaths now standardises for both long term and short term contributions.

5.3 Estimation strategy and Data

We base our regression of the determinants of mortality on Chapter 3. Number of deaths is regressed on spending per affected person, expected deaths and deprivation. A few changes were made due to the use of panel data. Firstly, fixed effects were used at the level of PCT, as well as year dummies to control for changes over time. Secondly, as the IMD is not updated annually, deprivation in the time-variant equation was measured by the percentage on Jobseekers' Allowance. Though an obvious indicator for deprivation,

this variable lacks the ability to capture the multiple dimensions of deprivation that the IMD does. Nevertheless it is a suitable substitute for the IMD variable, and has a high correlation of 0.81. It is also worth noting that although changes in deprivation do undoubtedly occur year by year, we would expect most areas' level of deprivation to remain fairly constant over six years, and therefore in the time-variant stage the fixed effects themselves will explain differences in deprivation to a large extent. The final change was the addition of population as a covariate. This was included to control for population size, therefore allowing the effect of the expected number of deaths to more accurately estimate the effect of age and gender. Additionally, this permitted the effect of changes in population over time to be controlled for. Population was considered to be a secondary standardising variable.

As before, the main variable of interest is health care spending. For the cancer and CHD models we use the corresponding programme budgets, divided by the number of people in each PCT who suffer from the illness. For the amenable model, we base the spending variable on the list of conditions covered [14]. Amenable spending is the combination of programme budgets: 1 (infectious diseases), 2 (cancers and tumours), 3 (disorders of blood), 4 (endocrine, nutritional and metabolic problems), 7 (neurological), 10 (problems of circulation), 11 (problems of the respiratory system), 13 (problems of the gastrointestinal system), 17 (problems of the genitourinary system), and 18 (maternity and reproductive health). The programme budgets that were left out are: 5 (mental health), 6 (learning disabilities), 8 (vision), 9 (hearing), 12 (dental), 14 (skin), 15 (musculo skeletal), 16 (trauma and injuries) 19 (neonatal), 20 (poisoning), 21 (health individuals), 22 (social care needs), and 23 (other). No prevalence data was used in the amenable model; the units of spending were simply spending per capita.

Compared to Chapter 4, the estimation of the elastiticities is complicated by the presence of endogeneity, due to health spending being higher in areas of ill health. Elasticities are estimated at both the initial fixed effects regression and the subsequent underlying

regression on the fixed effects, and endogeneity is a potential issue in both regressions. For the first regression, endogeneity would exist because health spending changes depending on the change in health needs across PCTs; for the second, endogeneity would exist because on average health spending is distributed towards areas of long term ill health.

To overcome this we employ instrumental variables. For the initial panel data regression, instruments were chosen that were intuitively likely to affect expenditure but not mortality, except through their effect on expenditure. Instrument sets were excluded if they failed the Hansen J statistic for instrument validity. For the time-variant 2SLS regression we used four instruments; the proportion of individuals on Disability Living Allowance (DLA); the proportion of individuals on Carer's Allowance (CA); the number of people per GP surgery; and number of people per GP surgery squared. The choice of instruments at this stage was constrained by the need to use variables that were updated yearly, thus excluding Census variables. DLA and CA were included as in Chapter 3, with CA replacing the census-derived 'unpaid carers' variable. The terms for claiming CA are more restrictive than those for simply responding to the Census indicating one provides unpaid care, in particular requiring the claimant provides more than 35 hours care per week, which explains the much lower proportions for CA than the census unpaid carer variable. The final instruments, the population per GP surgery and population per GP surgery squared, were used because PCT expenditure on GP services would vary by economies and diseconomies of scale at the surgery level. This variation leaves more or less money for other programmes, and the inclusion of a squared term permits this relationship to be non-linear.

Instruments for the underlying, time-invariant regression on the extracted fixed effects were, however, more difficult to choose. Instrumenting spending for a regression on fixed effects means that the validity of instruments - that they do not affect the fixed effects other than through their effect on spending - is harder to argue. We conceptualise this stage as a regression of the underlying and long-term differences in mortality between areas on the set of time-invariant covariates. Two sets of statistics support this notion.

Firstly, the between-area variation in amenable, cancer and CHD mortality is much larger than within-area/over-time variation: for amenable mortality the between-area standard deviation is 201, the within-area 33; for Cancer mortality the corresponding standard deviations are 538 and 23; for CHD mortality 317 and 54. This that means the fixed effects will capture most of the variation in the regression, as the dependent variable is not changing much over time.

Secondly, time-averaged mortality and the fixed effects are highly correlated, with no correlation less than 0.97 in any model. We therefore use similar instruments for both stages of regression, in particular we use instruments from the static model in Chapter 3. For Cancer, these are the proportion of unpaid carers from the 2001 Census and spending per capita on all programmes other than Cancer. These two instruments are augmented by the average number of people per GP surgery over time, and the square of that variable. For CHD, we use the spending per capita on all programmes other than CHD, alongside the two additional people per GP surgery variables.

Finally, for amenable mortality we use all of the instruments for the Cancer and CHD models, except for the two instruments based on spending. Though some of these instruments have the same name as the instruments in the initial stage, being based on the same variable, it is important to note that their average across time is used in the underlying regression whereas their deviation from their average is used in the first stage of the initial panel data IV regression. In general, there may be instruments we would expect to better predict changes in spending over time than changes in spending between areas, or vice versa. For our example, we might expect the population per GP instruments to exert a more significant effect on spending between areas than within areas, as economies and diseconomies of scale are likely to vary more between area than over time.

If health spending reacts instantly and perfectly to changes in area-level ill health, the problem of simultaneity would arise most starkly in the initial panel data regression. If health spending is distributed towards areas of health merely on average or sluggishly,

the problem of simultaneity is of greater concern in the underlying regression of the fixed effects. To address both possibilities we present four results for each regression set: one where no instruments are used at any stage; one where instruments are used in the underlying regression of the fixed effects; one where instruments are used in the initial panel data regression; and one where instruments are used at both stages. These are labeled 'a' to 'd', while the the three mortality types are labelled '1' (cancer), '2' (CHD) and '3' (amenable mortality), so the models run from '1a' to '3d'.

All regressions use heteroscedastic-robust standard errors and population weights.

The dataset used in this chapter is an amended panel version of the dataset in Chapter 3. The panel covers six years, from 2004 to 2009, and 151 PCTs. Unfortunately the merge of NHS West Hertfordshire (PCT code 5P4) with NHS East and North Hertfordshire (PCT code 5P3) reduced the available data units by one. Summary statistics can be found in Table 5.1. For all mortality data, observed number of deaths exceeds expected number of deaths. This is because the indirect standardisation is based on 2010 mortality rates for age-gender groups.

Variable	Mean	Std. Dev.	Minimum	Maximum	Period	Source
Cancer deaths	3131	12.6	64	135	2004-2009	NHS IC
Expected cancer deaths	2859	531.3	241	3131	2004-2009	NHS IC
Cancer spending per capita	83.4	154	36.8	144.3	2004/05-2009/10	DH
Cancer prevalence	0.0096	0.00364	0.00228	0.0223	2004-2009	NHS IC
CHD deaths	505	320	92	1947	2004-2009	NHS IC
Expected CHD deaths	403	282	97	1782	2004-2009	NHS IC
CHD spending per capita	3.54	1.14	0.521	5.51	2006/07-2009/10	DH
CHD prevalence	0.0354	0.00933	0.0136	0.0551	2004-2009	NHS IC
Amenable deaths	369	203	89	1255	2004-2009	NHS IC
Expected amenable deaths	311	204	87	1304	2004-2009	NHS IC
Amenable deaths spending per capita	603	666.9	269	3895	2004/05-2009/10	DH
Population	334113	172745	98680	1111421	2004/05-2009/10	DH
Proportion on JSA	0.0166	0.00711	0.00482	0.0552	2004-2009	Neighbourhood Statistics
IMD2007	23.8	9.09	8.09	48.3	2007	
Non-cancer spending per capita	1270	111	1030	1800	2004/05-2009/10	DH
Non-CHD spending per capita	1360	105	1100	1840	2006/07-2009/10	DH
Proportion receiving DLA	0.0055	0.00108	0.00203	0.00878	2004-2009	Neighbourhood Statistics
Proportion receiving CA	0.00638	0.00163	0.00205	0.0104	2004-2009	Neighbourhood Statistics
Proportion providing unpaid care	0.099	0.0117	0.0661	0.122	2001	2001 Census
Population per GP surgery	6150	1140	3530	9980	2004/05-2009/10	DH & QOF

Table 5.1: Summary statistics for PCT re-decomposition analysis (n=906 for all except CHD variables n=604)

5.4 Results

For the Cancer results, each regression in each model will be presented separately at first, to demonstrate how the models are different from one another. A table of combined results is then presented to allow ease of comparison. For CHD and amenable mortality only tables of combined results are presented. The sample size is 906 for amenable and cancer models, 604 for CHD and 151 for all underlying regressions on the extracted fixed effects.

Table 5.2 shows the initial panel data regression of Model 1a, regressing cancer mortality on cancer spending, expected mortality, JSA percentage, population and time dummies. Cancer spending is positive and significant, meaning that, even after controlling for covariates, cancer spending was higher in areas with higher cancer mortality. As expected, the coefficient on Expected Mortality is positive and significant, however considering it is based on an indirect standardisation of cancer mortality it might be expected to be closer to one. Neither JSA percentage nor population is significant, which suggests that these variables may not vary particularly over time. None of the time dummies are significant, which fits with the plateau we observe in Figure 5.2 during this period. The correlation between the fixed effects and time-averaged mortality is very high, at 0.994, suggesting that there is a lot of between-area variation that will be modelled in the subsequent underlying stage. The fixed effects are extracted for the next step in Model 1a.

Table 5.3 shows that for Model 1a, in the subsequent, underlying stage of regression on the extracted fixed effects we find that each covariates' coefficient is as expected according to our conceptualisation of the fixed effects as 'long-term cancer mortality'. The (now negative) effect of cancer spending is more significant at this stage than in the previous stage of the model, both in terms of size of coefficient and relatively lower standard error. Likewise, expected mortality is larger and more statistically significant. Though population size is still insignificant, the coefficient of IMD is positive and highly significant.

Model 1a: Cancer mortality		
Variable	Coef	SE
Cancer spending	1.96**	(0.906)
Expected mortality	0.281**	(0.13)
JSA percentage	1460	(1000)
Population (000s)	0.36	(0.358)
Time 2	2.51	(6.74)
Time 3	3.43	(9.09)
Time 4	9.75	(9.72)
Time 5	13.2	(10.6)
Time 6	-11.7	(17.4)
Tests	Value	P value
F test (9, 746)	5.42***	<0.001
Correlation(FE, mortality)	0.994	

Table 5.2: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

Bearing in mind the high degree of positive correlation between the fixed effects and mortality, what these coefficients imply is that differences between areas in cancer spending are negatively correlated with differences in cancer mortality, while differences in deprivation and expected mortality are positively related. These coefficients show the effects of cancer spending, deprivation and expected mortality on cancer mortality using variation between areas; the interpretation of the coefficient on cancer spending is that, based on the between-area variation, an increase in average cancer spending of £1000 per patient will reduce the average number of deaths by 24.

Model 1a: Fixed effects		
Variable	Coef	S.E
Cancer Spending	-24***	(6.06)
Expected mortality	0.516***	(0.0473)
IMD	3.84***	(0.95)
Population (000s)	0.213	(0.143)
Tests	Value	P value
F test (4, 146)	487***	<0.001

Table 5.3: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

In Model 1a no instruments were used. Model 1b includes instruments in the underlying regression on the extracted fixed effects. The results of the initial regression of Model 1b are displayed in Table 5.4. At this stage, the model is identical to 1a.

Model 1b: Cancer mortality		
Variable	Coef	SE
Cancer Spending	1.96**	(0.906)
Expected mortality	0.281**	(0.13)
JSA percentage	1460	(1000)
Population (000s)	0.36	(0.358)
Time 2	2.51	(6.74)
Time 3	3.43	(9.09)
Time 4	9.75	(9.72)
Time 5	13.2	(10.6)
Time 6	-11.7	(17.4)
Tests	Value	P value
F test (9, 746)	5.42***	<0.001
Correlation(FE, mortality)	0.994	

Table 5.4: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

Table 5.5 shows the results of the underlying regression in Model 1b. The instruments used are the proportion of unpaid carers, population per GP practice, population per GP practice squared and non-cancer spending per capita in the PCT. The instruments are jointly significant at the 0.1% level, however the first stage F test does not manage to exceed 10. The Kleibergen-Paap test is only passed at the 10% level, though at 5.95% is not far from significance at the 5% level. The Hansen J statistic does not reject the validity of the instruments and the endogeneity test strongly rejects the null hypothesis that cancer spending is exogenous. The unpaid carers coefficient of -48 aligns very closely with the the equivalent Chapter 3 instrument coefficient of -42, and is similarly highly significant. Population per GP practice displays a significant quadratic relationship with spending; increasing at low levels and falling at higher levels. The final instrument, non-cancer spending, is not significant.

The second stage results in the underlying regression of Model 1b are generally stronger

than the corresponding non-IV estimates of Model 1a. Cancer spending, in particular, is almost four times as effective at reducing cancer mortality as previously predicted; a £1000 per patient increase in spending translating to 80 fewer deaths. The effect of both deprivation and population are larger and, in the case of population, much more statistically significant. The difference between Model 1a and 1b would suggest that the underlying regression of the fixed effects ought to be instrumented.

Model 1b: Fixed Effects		
First stage on Cancer spending		
Variable	Coef	SE
Expected mortality	-0.00175**	(0.00083)
IMD	0.0516**	(0.0205)
Population (000s)	0.00501**	(0.00205)
Unpaid carers	-48***	(0.00083)
Pop (000s) per GP practice	-1.6**	(0.744)
Pop (000s) per GP practice^2	0.114**	(0.0575)
Non-Cancer spending	-1.81	(1.62)
Tests	Value	P value
First stage F(4, 143)	8.21***	<0.001
Hansen J $\chi^2_{(1)}$	2.8	0.423
Kleibergen-Paap $\chi^2_{(4)}$	9.06*	0.0595
Endogeneity test $\chi^2_{(1)}$	17.9***	<0.001
Second stage on Fixed Effects		
Variable	Coef	SE
Cancer Spending	-79.6***	(17.7)
Expected mortality	0.337***	(0.0984)
IMD	4.33***	(1.44)
Population (000s)	0.702***	(0.272)
Tests	Value	P value
F test (4, 146)	365***	<0.001

Table 5.5: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

Results for the initial panel data regression of Model 1c are presented in Table 5.6. This is a within-group IV regression of cancer mortality using the percentage claiming Carer's Allowance and the population per GP practice variables as instruments. The instruments are jointly significant at the 0.1% level, however the first stage F statistic does not quite

exceed 10. The Kleibergen-Paap test is insignificant, suggesting potential problems with instrument strength. Additionally the endogeneity test is only significant at the 10% level, implying that there is not much evidence of endogeneity. The Hansen J test of instrument validity is passed. The CA instrument has a strongly negative coefficient, neither of the other two instruments are significant.

The second stage results, compared with the analogous coefficients in Tables 5.2 and 5.4, are less significant. Both Cancer spending and expected mortality are only significant at the 10% level. The correlation between the fixed effects and time-averaged mortality is very high.

Table 5.7 shows the underlying regression of the fixed effects of Model 1c. Cancer spending and deprivation have slightly larger coefficients than in the corresponding results of Model 1a (Table 5.3).

Table 5.8 presents the results of the initial stage of Model 1d. The results are identical to the initial regression's results of Model 1c.

The results of the underlying regression of the fixed effects in Model 1d are displayed in Table 5.9. The instruments are the same as those used in Model 1b Table 5.5, so the first stage results are identical. The results in the second stage are very similar to those in Model 1b, with all coefficients slightly larger in size.

Results for all the Cancer models are combined in Table 5.10 (instruments are combined in Table 5.11). Across the three instrumented models (1b, 1c and 1d), the results remain qualitatively similar. At the initial panel data stage the significance of spending and expected mortality on mortality is reduced when spending is instrumented (models 1c and 1d). When instruments are employed for spending in the subsequent stage (models 1b and 1d) all coefficients, particularly spending but excluding expected mortality, are larger. All models pass the Hansen J test for instrument validity. The endogeneity Chi

Model 1c: Cancer mortality		
First stage on Cancer spending		
Model	1c	
Variable	Coef	SE
Expected mortality	0.00636***	(0.00242)
JSA percentage	99.4**	(42.1)
Population (000s)	-0.0216***	(0.00832)
Time 2	-3.79***	(0.229)
Time 3	-6.65***	(0.274)
Time 4	-7.23***	(0.264)
Time 5	-8.05***	(0.319)
Time 6	-8.75***	(0.653)
CA percentage	-1470***	(298)
Pop (000s) per GP practice	0.789	(1.4)
Pop(000s) per GP practice^2	-0.071	(0.1)
Tests	Value	P value
First stage F(4, 744)	9.64***	<0.001
Hansen J $\chi^2_{(3)}$	2.24	0.326
Kleibergen-Paap $\chi^2_{(4)}$	0.049	0.997
Endogeneity test $\chi^2_{(1)}$	3.56*	0.0592
Second stage on Cancer Mortality		
Variable	Coef	SE
Cancer Spending	10.3*	(5.82)
Expected mortality	0.236*	(0.123)
JSA percentage	611	(1230)
Population (000s)	0.404	(0.297)
Time 2	35.2	(22.9)
Time 3	61.1	(41)
Time 4	73.5	(45.2)
Time 5	87.2	(53.2)
Time 6	72.1	(63.3)
Tests	Value	P value
F test (9, 746)	6.8***	<0.001
Correlation(FE, mortality)	0.992	

Table 5.6: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

Model 1c: Fixed Effects		
Variable	Coef	S.E
Cancer Spending	-32.8***	(6.06)
Expected mortality	0.559***	(0.0471)
IMD	4.24***	(0.936)
Population (000s)	0.174	(0.143)
Tests	Value	P value
F test (4, 146)	571***	<0.001

Table 5.7: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

squared test is significant at the 1% level for the underlying stage regression but only significant at the 10% level for the initial panel data regression. This, coupled with the more pronounced changes in predicted coefficients in the underlying stage, suggests that the main source of endogeneity is between areas rather than within areas. However, neither regression exceeds 10 in the first stage F test, and the initial panel data 2SLS regression fails the Kleibergen-Paap test, suggestingthat there may be issues with the strength of the instruments, especially at the inital panel data stage.

Table 5.11 shows the results for the first stages of the models. For the inital stage, the only significant instrument is CA; for the subsequent underlying stage the only insignificant instrument is non-cancer spending. This adds to the evidence that the inital stage either lacks instrument strength or does not require instrumentation. The instruments for the underlying stage, however, seem to be sufficiently powerful and significant, pass all diagnostic tests and align very closely with the results from Chapter 3.

Model 1d: Cancer mortality		
First stage on Cancer spending		
Variable	Coef	SE
Expected mortality	0.00636***	(0.00242)
JSA percentage	99.4**	(42.1)
Population (000s)	-0.0216***	(0.00832)
Time 2	-3.79***	(0.229)
Time 3	-6.65***	(0.274)
Time 4	-7.23***	(0.264)
Time 5	-8.05***	(0.319)
Time 6	-8.75***	(0.653)
CA percentage	-1470***	(298)
Pop (000s) per GP practice	0.789	(1.4)
Pop(000s) per GP practice^2	-0.071	(0.1)
Tests	Value	P value
First stage F(4, 744)	9.64***	<0.001
Hansen J $\chi^2_{(3)}$	2.24	0.326
Kleibergen-Paap $\chi^2_{(4)}$	0.049	0.997
Endogeneity test $\chi^2_{(1)}$	3.56*	0.0592
Second stage on Cancer Mortality		
Variable	Coef	SE
Cancer Spending	10.3*	(5.82)
Expected mortality	0.236*	(0.123)
JSA percentage	611	(1230)
Population (000s)	0.404	(0.297)
Time 2	35.2	(22.9)
Time 3	61.1	(41)
Time 4	73.5	(45.2)
Time 5	87.2	(53.2)
Time 6	72.1	(63.3)
Tests	Value	P value
F test (9, 746)	6.8***	<0.001
Correlation(FE, mortality)	0.992	

Table 5.8: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

Model 1d: Fixed Effects		
First stage on Cancer spending		
Variable	Coef	SE
Expected mortality	-0.00175**	(0.00083)
IMD	0.0516**	(0.0205)
Population (000s)	0.00501**	(0.00205)
Unpaid carers	-48***	(0.00083)
Pop (000s) per GP practice	-1.6**	(0.744)
Pop (000s) per GP practice^2	0.114**	(0.0575**)
Non-Cancer spending	-1.81	(1.62)
Tests	Value	P value
First stage F(4, 143)	8.21***	<0.001
Hansen J $\chi^2_{(1)}$	3.01	0.39
Kleibergen-Paap $\chi^2_{(4)}$	9.06***	0.0595
Endogeneity test $\chi^2_{(1)}$	17.4***	<0.001
Second stage on Fixed Effects		
Variable	Coef	S.E
Cancer Spending	-88***	(17.6)
Expected mortality	0.38***	(0.098)
IMD	4.73***	(1.42)
Population (000s)	0.66**	(0.271)
Tests	Value	P value
F test (4, 146)	418***	<0.001

Table 5.9: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

Models for cancer	1a		1b		1c		1d	
Regression on mortality	Coef		SE		Coef		SE	
Cancer Spending	1.96**		(0.906)		10.3*		(5.82)	
Expected mortality	0.281**		(0.13)		0.236*		(0.123)	
JSA percentage	1460		(1000)		611		(1230)	
Population (000s)	0.36		(0.358)		0.404		(0.297)	
Time 2	2.51		(6.74)		35.2		(22.9)	
Time 3	3.43		(9.09)		61.1		(41)	
Time 4	9.75		(9.72)		73.5		(45.2)	
Time 5	13.2		(10.6)		87.2		(53.2)	
Time 6	-11.7		(17.4)		72.1		(63.3)	
Tests	Value		P value		Value		P value	
F test (9, 746)	5.42***		<0.001		6.8***		<0.001	
Correlation(FE, mortality)	0.994				0.992			
Regression on fixed effects	Coef	S.E	Coef	S.E	Coef	S.E	Coef	S.E
Cancer Spending	-24***	(6.06)	-79.6***	(17.7)	-32.8***	(6.06)	-88***	(17.6)
Expected mortality	0.516***	(0.0473)	0.337***	(0.0984)	0.559***	(0.0471)	0.38***	(0.098)
IMD	3.84***	(0.95)	4.33***	(1.44)	4.24***	(0.936)	4.73***	(1.42)
Population (000s)	0.213	(0.143)	0.702***	(0.272)	0.174	(0.143)	0.66**	(0.271)
Tests	Value	P value	Value	P value	Value	P value	Value	P value
F test (4, 146)	487***	<0.001	365***	<0.001	571***	<0.001	418***	<0.001

Table 5.10: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

Models for cancer first stage: spending	1a	1b		1c	1d	
Expected mortality				0.00636***	(0.00242)	
JSA percentage				99.4**	(42.1)	
Population (000s)				-0.0216***	(0.00832)	
Time 2				-3.79***	(0.229)	
Time 3				-6.65***	(0.274)	
Time 4				-7.23***	(0.264)	
Time 5				-8.05***	(0.319)	
Time 6				-8.75***	(0.653)	
CA percentage				-1470***	(298)	
Pop (000s) per GP practice				0.789	(1.4)	
Pop(000s) per GP practice^2				-0.071	(0.1)	
Tests				Value	P value	
First stage F(4, 744)				9.64***	<0.001	
Hansen J $\chi^2_{(3)}$				2.24	0.326	
Kleibergen-Paap $\chi^2_{(4)}$				0.049	0.997	
Endogeneity test $\chi^2_{(1)}$				3.56*	0.0592	
Regression on average cancer spending		Coef	SE		Coef	SE
Expected mortality		-0.00175**	(0.00083)		-0.00175**	(0.00083)
IMD		0.0516**	(0.0205)		0.0516**	(0.0205)
Population (000s)		0.00501**	(0.00205)		0.00501**	(0.00205)
Unpaid carers		-48***	(0.00083)		-48***	(0.00083)
Pop (000s) per GP practice		-1.6**	(0.744)		-1.6**	(0.744)
Pop (000s) per GP practice^2		0.114**	(0.0575**)		0.114**	(0.0575**)
Non-Cancer spending		-1.81	(1.62)		-1.81	(1.62)
Tests		Value	P value		Value	P value
First stage F(4, 143)		8.21***	<0.001		8.21***	<0.001
Hansen J $\chi^2_{(1)}$		2.8	0.423		3.01	0.39
Kleibergen-Paap $\chi^2_{(4)}$		9.06*	0.0595		9.06***	0.0595
Endogeneity test $\chi^2_{(1)}$		17.9***	<0.001		17.4***	<0.001

Table 5.11: *** is significant at the 1% level; ** at the 5% level; * at the 10% level, robust standard errors in parentheses

5.4 Results

The results for the CHD programme of care are presented in Table 5.12. Model 1a shows the results for a regression on CHD mortality and the corresponding regression on the extracted fixed effects. The only significant variable in the initial panel data regression is the expected number of CHD deaths, which is negative. Though this is unexpected, the overall effect of expected deaths when the underlying stage is taken into account is positive. Time dummies are significant and negative across all CHD models and imply that, separate to the influence of the covariates, CHD mortality has fallen year-on-year. In the subsequent, underlying stage of regression on the extracted fixed effects we again find that all covariates accord with expectations.

When instrumented, the initial panel data regression does not change much. The effect of expected CHD mortality is still negative and significant, and the effect of time dummies remains negative, significant, and increases over time. It is therefore no surprise that the endgoeneity test fails to reject the null hypothesis that the endogenous regressor can actually be treated as exogenous. However, as the first stage F test and Kleibergen-Paap test indicate, the instruments used are weak. Table 5.13 shows that of the four instruments, only the two relating to population per GP surgery are significant, whereas the proportion of people on DLA and CA are not significant. In the underlying regression the instruments appear to be stronger: all attain significance at the 5% level, the first stage F test exceeds 10 and the Kleibergen-Paap test is passed. However, the actual effect on the coefficient of spending is very small, merely reducing its size slightly as seen in the changes from models 2a to 2b and, equivalently, 2c to 2d. Both models that instrument for spending at this stage (2b and 2d) pass the Hansen J test of instrument validity. However the Endogeneity test does not reject the null hypothesis that spending can be treated as an exogenous regressor.

Models for CHD	2a		2b		2c		2d	
Regression on mortality	Coef		SE		Coef		SE	
CHD Spending	8.03		(5.61)		6.81		(31)	
Expected mortality	-1.33***		(0.179)		-1.33***		(0.183)	
JSA percentage	-485		(1100)		-487		(1100)	
Population (000s)	-0.0335		(0.254)		-0.0366		(0.251)	
Time 2	-9.82*		(5.11)		-9.78*		(5.2)	
Time 3	-16.3***		(4.77)		-16.3***		(5.08)	
Time 4	-33.9***		(11.8)		-33.8***		(12.1)	
Tests	Value		P value		Value		P value	
F test (7, 446)	60.5***		<0.001		61.2***		<0.001	
Correlation(FE, mortality)	0.985				0.985			
Regression on fixed effects	Coef	S.E	Coef	S.E	Coef	S.E	Coef	S.E
CHD Spending	-183***	(43.4)	-174***	(65.9)	-181***	(43.4)	-172***	(65.9)
Expected mortality	2.21***	(0.0862)	2.22***	(0.0921)	2.22***	(0.0862)	2.22***	(0.0921)
IMD	2.3**	(0.977)	2.38 **	(1.04)	2.31**	(0.977)	2.38**	(1.04)
Population (000s)	0.356***	(0.12)	0.351***	(0.123)	0.359***	(0.12)	0.354***	(0.123)
Tests	Value	P value	Value	P value	Value	P value	Value	P value
F test (4, 146)	1490***	<0.001	1590***	<0.001	1490***	<0.001	1590***	<0.001

Table 5.12: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

Model for CHD first stage: spending	2a	2b		2c	2d	
Expected mortality				-0.000504	(0.000681)	
JSA percentage				-0.848	(7.04)	
Population (000s)				-0.0031*	(0.00159)	
Time 2				0.048	(0.0365)	
Time 3				0.0609	(0.055)	
Time 4				0.102	(0.105)	
DLA percentage				-55.9	(52)	
CA percentage				22.3	(69.8)	
Pop (000s) per GP practice				0.894**	(0.406)	
Pop (000s) per GP practice^2				-0.00652**	(0.003)	
Tests				Value	P value	
First stage F(4, 743)				1.74	0.139	
Hansen J $\chi^2_{(3)}$				1.83	0.608	
Kleibergen-Paap $\chi^2_{(4)}$				4.74	0.315	
Endogeneity test $\chi^2_{(1)}$				0.001	0.973	
Regression on average CHD spending		Coef	S.E		Coef	S.E
Expected mortality		-0.000383**	(0.000151)		-0.000383**	(0.000151)
IMD		-0.00725***	(0.00176)		-0.00725***	(0.00176)
Population (000s)		0.000452**	(0.000221)		0.000452**	(0.000221)
Non-CHD spending		-0.648***	(0.216)		-0.648***	(0.216)
Pop (000s) per GP practice		-0.216**	(-0.0897)		-0.216**	(-0.0897)
Pop (000s) per GP practice^2		0.0131***	(0.00698)		0.0131***	(0.00698)
Tests		Value	P value		Value	P value
First stage F(3, 144)		12***	<0.001		12***	<0.001
Hansen J $\chi^2_{(1)}$		2.19	0.334		2.19	0.334
Kleibergen-Paap $\chi^2_{(4)}$		18.1***	<0.001		18.1***	<0.001
Endogeneity test $\chi^2_{(1)}$		0.014	0.905		0.014	0.905

Table 5.13: *** is significant at the 1% level; ** at the 5% level; * at the 10% level, robust standard errors in parentheses

5.4 Results

The results for the amenable mortality model are presented in Table 5.14. Model 3a shows the results for a regression on amenable mortality and the corresponding regression on the extracted fixed effects. As in the CHD model, the only significant variable in the initial panel data regression is the expected number of amenable deaths, which is negative. Similarly, the time dummies follow the pattern in the CHD model: they are significant, negative and increasing in size. However, unlike the CHD model, this pattern is not repeated when spending is instrumented. In the subsequent, underlying stage of regression on the extracted fixed effects we find that expected mortality, deprivation and population display similar coefficients to their analogues in models 1a and 2a. The coefficient on spending, however, is positive and significant in effect.

When the initial panel data regression is instrumented, the coefficient on spending is negative and significant, as is the coefficient on the size of population. Thought the Hansen J statistic is passed, the first stage F test of 3.98 coupled with the insignificant Kleibergen-Paap statistic suggest that the instruments are weak. The first stage of the IV regressions can be found in Table 5.15. The only significant instrument is the proportion of people on DLA. The endogeneity test for the initial panel data stage is significant, implying that even with the fairly weak instruments available, endogeneity is present. When spending is instrumented in the subsequent underlying regression the coefficient on spending rises to 2550 in Model 1b and 3090 in Model 1d, remaining highly significant in both. The other major change is that the effect of deprivation falls to statistical insignificance. The first stage F test of 6.54 shows that the instruments are explaining more of the variation than in the initial regression, though they still fall short of the rule of thumb value of 10. Though the Hansen J test is passed and the endogeneity test shows endogeneity to be present, the Kleibergen-Paap statistic is not significant at the 10% level, further suggesting that the instruments are weak. Of the instruments themselves, only the proportion of people providing unpaid care is significant.

There are two unexpected features in the amenable mortality models: firstly, the positive

effect of spending on mortality in the underlying stage where the fixed effects are analysed; secondly, that when instrumented, this coefficient increases in size. Regarding the former, as the amenable mortality set of models is a collection of a broader and more diverse range of health conditions than cancer and CHD, it is likely that it is relationships between non-cancer and non-CHD conditions' health spending and mortality that is causing these results. Regarding the latter, it is possible that there is some issue with the use of the proportion of unpaid carers as an instrument, as its sign is opposite to what we would expect and what is found in the cancer models of this chapter and Chapter 3.

Models for amenable mortality	3a		3b		3c		3d	
Regression on mortality	Coef		SE		Coef		SE	
Amenable Spending	-25.6		(22.9)		-472**		(203)	
Expected mortality	-0.977***		(0.268)		-0.991***		(0.279)	
JSA percentage	-303		(659)		776		(883)	
Population (000s)	-0.284		(0.186)		-0.441**		(0.214)	
Time 2	-16.3***		(4.1)		-0.536		(8.3)	
Time 3	-34.7***		(3.75)		-28.1***		(5.34)	
Time 4	-44.8***		(3.85)		-29.3***		(8.23)	
Time 5	-44.5***		(4.47)		-21.9**		(11.1)	
Time 6	-52.5***		(8.75)		-15.4		(19.6)	
Tests	Value		P value		Value		P value	
F test (9, 746)	100***		<0.001		83.1***		<0.001	
Correlation(FE, mortality)	0.976				0.976			
Regression on fixed effects	Coef	S.E	Coef	S.E	Coef	S.E	Coef	S.E
Amenable Spending	704***	(148)	2740***	(557)	1180***	(147)	3270***	(569)
Expected mortality	1.59***	(0.115)	1.27***	(0.179)	1.61***	(0.115)	1.28***	(0.18)
IMD	3.47***	(0.851)	0.943	(1.45)	2.99***	(0.844)	0.4	(1.47)
Population (000s)	0.73***	(0.116)	1.06***	(0.197)	0.884***	(0.115)	1.22***	(0.198)
Tests	Value	P value	Value	P value	Value	P value	Value	P value
F test (4, 146)	2140***	<0.001	2450***	<0.001	2580***	<0.001	2660 ***	<0.001

Table 5.14: *** is significant at the 1% level; ** at the 5% level; * at the 10% level

Models for amenable mortality first stage: spending	3a	3b		3c	3d	
Expected mortality				-0.000212	(0.000225)	
JSA percentage				2.18***	(0.831)	
Population (000s)				-0.000122	(0.00024)	
Time 2				0.0323***	(0.00443)	
Time 3				0.0108**	(0.00592)	
Time 4				0.0265***	(0.0067)	
Time 5				0.0419***	(0.00808)	
Time 6				0.0773***	(0.0151)	
DLA percentage				19.3**	(6.17)	
CA percentage				-8.95	(5.88)	
Pop (000s) per GP practice				-0.00261	(0.0326)	
Pop (000s) per GP practice^2				0.000995	(0.00219)	
Tests				Value	P value	
First stage F(4, 743)				3.93***	<0.001	
Hansen J $\chi^2_{(3)}$				4.95	0.176	
Kleibergen-Paap $\chi^2_{(4)}$				7.51	0.112	
Endogeneity test $\chi^2_{(1)}$				5.41**	0.02	
Regression on average spending		Coef	S.E		Coef	S.E
Expected mortality		0.0000246	(0.0000792)		0.0000246	(0.0000792)
IMD		0.000286	(0.000559)		0.000286	(0.000559)
Population (000s)		-0.0000288	(0.0000819)		-0.0000288	(0.0000819)
Unpaid Carers percentage		1.07***	(0.264)		1.07***	(0.264)
Pop (000s) per GP practice		0.0157	(0.0186)		0.0157	(0.0186)
Pop (000s) per GP practice^2		-0.00228	(0.00139)		-0.00228	(0.00139)
Tests		Value	P value		Value	P value
First stage F(3, 143)		8.89***	<0.001		8.89***	<0.001
Hansen J $\chi^2_{(1)}$		2.06	0.357		1.68	0.43
Kleibergen-Paap $\chi^2_{(4)}$		1.47	0.689		1.56	0.816
Endogeneity test $\chi^2_{(1)}$		21.8***	<0.001		23***	<0.001

Table 5.15: *** is significant at the 1% level; ** at the 5% level; * at the 10% level, robust standard errors in parentheses

5.4 Results

For the inequality analysis we present the re-decomposition based on the fully instrumented models 1d, 2d and 3d. Using the same model specification across all mortality groups aids comparison of the results. Additionally, there is only one IV regression that fails to attain significance in the endogeneity test, namely the initial regression in model 2d, and for this the results are quantitively very similar whether instrumented or not.

The unadjusted Concentration Index for Cancer is 0.165, which is strongly pro-rich (Table 5.16). The main contributions to this inequality are from population (9.3%), expected mortality (26.5%) and the fixed effects (67.3%). Each of these three positive contributions is made up of a positive effect on cancer mortality (elasticity greater than zero) and a pro-rich distribution. The contribution of the residual is small, at 0.23%, which indicates that most of the inequality is being explained within the model.

Dependent variable	Cancer Mortality CI 0.165									
Variables	Spending		Expected mortality		JSA percentage		Population			
	0.000468		0.0437		-0.00216		0.0154			
Breakdown	Elas	CI	Elast	CI	Elast	CI	Elast	CI		
	0.906	0.00516	0.231	0.19	0.00881	-0.245	0.155	0.0997		
Time dummies	Time 2		Time 3		Time 4		Time 5		Time 6	
	0.000619		0.000209		0.00299		0.00105		-0.00717	
Breakdown	Elast	CI	Elast	CI	Elast	CI	Elast	CI	Elas	CI
	0.0051748	0.12	0.00912	0.0227	0.0111	0.205	0.0133	0.0788	0.0112	-0.64
Non variables	Fixed Effects		Residual υ							
	0.111		-0.000382							
Breakdown	Elast	CI								
	0.466	0.239								
Standardised variable	Standardised Mortality CI 0.106									

Table 5.16: Cancer decomposition results

Dependent variable	Fixed Effects in Cancer model CI 0.239							
Variables	Spending		Expected mortality		IMD		Population	
	0.0809		0.196		-0.0419		0.0712	
Breakdown	Elas	CI	Elast	CI	Elast	CI	Elast	CI
	-1.52	-0.533	0.798	0.245	0.209	-0.2	0.539	0.132
Non variables	Residual ε							
	-0.01							
Breakdown								

Table 5.17: Cancer re-decomposition results

The fixed effects are extracted and re-decomposed in Table 5.17. These results accord closely with Chapter 3 and our a priori expectations. Spending reduces mortality and displays a pro-poor distribution, therefore its overall contribution is pro-rich. Expected mortality and population increase the number of deaths, and are distributed more towards richer areas. These two variables, as features of the population in the area, are treated as standardising variables. Deprivation, as measured by the IMD2007, increases the number of deaths and has a pro-poor distribution. The residual at this stage is larger, at 4.2%, than in the initial stage. This suggests that less variation is being explained by the model at this stage, though the model still captures 95.8% of the CI inequality.

Table 5.18 shows the combined results from the decomposition and re-decomposition. The most important thing to note is that, when long term factors are taken into account through the re-decomposition of the fixed effects, the standardised mortality switches from strongly pro-rich to strongly pro-poor. The standardising variables, expected mortality and population, increase mortality and have a pro-rich distribution at both stages. For both variables the time-invariant contribution is approximately twice as great as the time-varying contribution. The effect of deprivation (combining the JSA contribution in the initial stage with the IMD contribution in the underlying stage) is pro-poor; throughout the model deprivation increases mortality and is distributed in poorer areas. The time-invariant contribution is around nine times greater than the time-varying contribution, however this comparison is only approximate as the two variables measure slightly different things. Spending exhibits a strong pro-rich contribution to mortality, dominated by the contribution of the underlying, time-invariant stage. Spending on cancer reduces mortality and has a pro-poor distribution. However, the contribution at the time-varying stage, although small, is pro-rich for the inverse reason; spending increases mortality and is pro-rich distributed. The positive elasticity at the time-varying stage is based on the initial stage's spending coefficient in Model 1d that is insignificant at the 5% level (and exceeded by a negative coefficient in the underlying stage). The small positive concentration index for spending is at odds with the large negative concentration index for spending when

averaged over time. This characteristic is due to spending increasing over time as JSA claimants per capita fell, so although between areas there is more cancer spending in areas with more JSA claimants per capita, when each area-time observation is treated separately the concentration index is dominated by the effect of observations towards the end of the period having higher spending and lower JSA per capita figures overall. The residuals' joint contribution is small, at 3.1%, suggesting that nearly all of the inequality is being captured in the model. When fully standardised, the CI of cancer mortality is pro-poor, at -0.018, and close to the estimation in Chapter 3.

Dependent variable	Cancer Mortality CI 0.165				
Variables	Spending 0.0381	Expected mortality 0.134	Deprivation -0.0216	Population 0.0486	
Breakdown	TV TI 0.000468 0.0376	TV TI 0.0437 0.0912	TV TI -0.00216 -0.0195	TV TI 0.0154 0.0332	
Time dummies	Time 2 0.000619	Time 3 0.000209	Time 4 0.00299	Time 5 0.00105	Time 6 -0.00717
Breakdown					
Non variables	Residual v -0.00507				
Breakdown	TV TI -0.000382 -0.00469				
Standardised variable	Standardised Mortality CI -0.018				

Table 5.18: Cancer combined re-decomposition results, contributions split into time-variant (TV) and time-invariant (TI) components

5.4 Results

The unadjusted Concentration Index for CHD mortality is 0.178, which is strongly pro-rich (Table 5.19) and similar to the the unadjusted CI for cancer. The contribution of the fixed effects in this model are dominant, at 204% of the unadjusted CI. In contrast to the cancer model the expected mortality contribution is pro-poor, which is a feature of the negative coefficients in the initial stage of the CHD regression models. The contribution of the residuals is fairly large, at 11%, suggesting that the model is not capturing all sources of inequality well.

Dependent variable	CHD Mortality CI 0.178							
Variables	Spending -0.00007		Expected mortality -0.21		JSA percentage 0.00289		Population -0.00235	
Breakdown	Elas 0.01146	CI -0.00612	Elast -1.09	CI 0.193	Elast -0.01181	CI -0.245	Elast -0.0236	CI 0.0997
Time dummies	Time 4 -0.000512		Time 5 -0.00033		Time 6 0.00565			
Breakdown	Elast -0.002491	CI 0.205	Elast -0.00419	CI 0.0788	Elas -0.00883	CI -0.64		
Non variables	Fixed Effects 0.363		Residual v 0.0195					
Breakdown	Elast 2.12	CI 0.171						
Standardised variable	Standardised Mortality CI 0.39							

Table 5.19: CHD decomposition results

Dependent variable	Fixed Effects in CHD model CI 0.171							
Variables	Spending 0.000585		Expected mortality 0.216		IMD -0.00776		Population 0.0142	
Breakdown	Elas -0.0915	CI -0.00377	Elast 0.858	CI 0.251	Elast 0.0388	CI -0.2	Elast 0.108	CI 0.132
Non variables Breakdown	Residual ε -0.000104							

Table 5.20: CHD re-decomposition results

The fixed effects are extracted and re-decomposed in Table 5.20. These results are not as similar to those in Chapter 3 as the analogous results for the cancer model. In particular, the effect of spending is far lower than in Chapter 3. Expected mortality and population increase the number of deaths, and are distributed more towards richer areas. Deprivation also increases the number of deaths, but is distributed towards poorer areas. Spending reduces mortality and its CI is very close to zero, indicating that cancer spending is fairly equal between rich and poor areas. The residual at this stage is small, at 0.1% of the fixed effects.

Table 5.21 shows the combined results from the decomposition and re-decomposition. As before, the inclusion of the fixed effects re-decomposition causes the standardised mortality to switch from strongly pro-rich to strongly pro-poor. The standardising variables, expected mortality and population, make pro-rich contributions overall. When combined, the effect of deprivation is pro-poor and small. Spending is pro-rich and small. However in CHD, unlike cancer, the CI of spending does not switch from pro-poor to pro-rich when spending is averaged over time. Indeed, the two CIs for spending are very close together. The residuals joint contribution is moderate, at 10.8%, which means that the model is not performing particularly well at capturing the inequality in CHD deaths.

Dependent variable	CHD Mortality CI 0.178							
Variables	Spending 0.00117		Expected mortality 0.247		Deprivation -0.0135		Population 0.0278	
Breakdown	TV -0.00007	TI 0.00124	TV -0.21	TI 0.456	TV 0.00289	TI -0.0164	TV -0.00235	TI 0.0301
Time dummies	Time 4 -0.000512		Time 5 -0.00033		Time 6 0.00565			
Breakdown								
Non variables	Residual υ 0.0187							
Breakdown	TV 0.0195	TI -0.000799						
Standardised variable	Standardised Mortality CI -0.0965							

Table 5.21: CHD combined re-decomposition results, contributions split into time-variant (TV) and time-invariant (TI) components

5.4 Results

The unadjusted Concentration Index for amenable mortality is 0.125, which is strongly pro-rich (Table 5.22) and which is slightly less pro-poor than the corresponding CIs for cancer and CHD. As in the CHD model, the contribution of the fixed effects is dominant, at 254% of the unadjusted CI. The expected mortality contribution is pro-rich, again made up of a negative elasticity and positive CI. The contribution of the residuals is small, at 0.3%.

Dependent variable	Amenable Mortality CI 0.125									
Variables	Spending 0.00795		Expected mortality -0.153		JSA percentage -0.00647		Population -0.0397			
Breakdown	Elas	CI	Elast	CI	Elast	CI	Elast	CI		
	-0.61	-0.013	-0.869	0.176	0.0264	-0.245	-0.399	0.0997		
Time dummies	Time 2 -0.0000223		Time 3 -0.000227		Time 4 -0.00216		Time 5 -0.000623		Time 6 0.00361	
Breakdown	Elast	CI	Elast	CI	Elast	CI	Elast	CI	Elas	CI
	-0.000186	0.12	-0.01	0.0227	-0.0105	0.205	-0.00791	0.0788	-.00564	-0.64
Non variables	Fixed Effects 0.317		Residual υ -0.000381							
Breakdown	Elast	CI								
	2.87	0.11								
Standardised variable	Standardised Mortality CI 0.318									

Table 5.22: Amenable decomposition results

Dependent variable	Fixed Effects in amenable model CI 0.11							
Variables	Spending 0.00327		Expected mortality 0.0886		IMD -0.00135		Population 0.05086	
Breakdown	Elas 1.47	CI 0.00223	Elast 0.39	CI 0.227	Elast 0.00675	CI -0.2	Elast 0.384	CI 0.132
Non variables	Residual ε -0.00393							
Breakdown								

Table 5.23: Amenable re-decomposition results

The fixed effects are extracted and re-decomposed in Table 5.23. Expected mortality and population both display positive CI and elasticity. Deprivation has a small positive elasticity and negative CI, leading to a small pro-poor contribution. Spending is very weakly pro-rich, but displays a large elasticity greater than one. The residual at this stage is small, at 3.6% of the fixed effects.

Table 5.24 shows the combined results from the decomposition and re-decomposition. As in both prior cases, the inclusion of the fixed effects re-decomposition causes the standardised mortality to switch from strongly pro-rich, to strongly pro-poor. The standardising variables, expected mortality and population, make pro-rich contributions overall. The contribution of deprivation is pro-poor and small. Spending is pro-rich, combining a a negative elasticity negative CI contribution at the first stage with a positive elasticity positive CI contribution at the second stage. The residuals' joint contribution is small, at 9.4%.

Dependent variable	Amenable Mortality CI 0.125				
Variables	Spending 0.0174		Expected mortality 0.102	Deprivation -0.0103	Population 0.106	
Breakdown	TV 0.00795	TI 0.00941	TV TI -0.153 0.255	TV TI -0.00647 -0.00388	TV TI -0.0397 0.146	
Time dummies	Time 2 0.000619		Time 3 0.000209	Time 4 0.00299	Time 5 0.00105	Time 6 -0.00717
Breakdown						
Non variables	Residual υ -0.0117					
Breakdown	TV -0.000381	TI -0.0113				
Standardised variable	Standardised Mortality CI -0.0827					

Table 5.24: Amenable combined Re-Decomposition results, contributions split into time-variant (TV) and time-invariant (TI) components

5.5 Conclusion

By combining the elements of Chapters 3 and 4, this chapter has analysed the effect of spending on NHS health and health inequality between 2004 and 2009. Using a wide range of data from various data sources we have been able to see how fixed effects re-decomposition works within three different levels of NHS mortality data. The results are somewhat mixed: the cancer programme and CHD sub-programme models perform well but the amenable mortality model performs poorly.

The cancer model performs well in all diagnostic tests, and the results accord with previous work both in Chapter 3 and in the literature. It is through the fixed effects re-decomposition that the important contribution of expected mortality and the important negative elasticity of health spending are found. A few caveats to note within the cancer model are that the specific treatments that cancer spending is buying will have changed over the course of the six years, and changed to differing extents for the various cancers that the spending data covers. Though the time dummies control for changes in mortality over time, changes in the relationship between health spending and mortality are less easy to test with only six years of data. Additionally, the scope of severities and prognoses suggest that, on a micro level, this data may be based on some of the same individuals across multiple year observations, as people live with cancer over the financial year boundaries. These types of issues are inherent when using macro area-level data.

The CHD model, while passing most diagnostic tests, does not appear on the face of it to require instrumentation. This may be because issues of endogeneity are less severe in the CHD sub-programme, or it may be because suitably powerful instruments have not been identified. The collection of spending data on CHD was initiated more recently than for the other two models and potentially issues of data quality may be more severe than for the more established programmes of spending. A major caveat to note with the CHD model (and the amenable model) is the significant negative coefficient on expected deaths at the

initial, panel data stage of the model. This negative coefficient appeared whenever fixed effects were employed. It is not clear why. One possible explanation is that the expected mortality figures were derived through indirect standardisation of 2010 mortality rates. This means the national mortality rate for each age group in 2010 is applied to the PCT's population for each age group for every year. If mortality rates for age groups change over time, and particularly if the change is part of an upward trend as lifestyle choices and medical interventions delay the burden of mortality to later years, an increase in the 40-45 age group between 2005 and 2006 might increase CHD mortality but be expected to reduce CHD mortality according to the 2010 national mortality rates.

The model for mortality amenable to health care performs the worst. It was included to provide a model with a wider variety of conditions. Considering the variety of illnesses included in the definition of amenable mortality, it is perhaps not surprising that a clear effect of health spending could not be identified. Additionally, the spending variable itself could not be distilled down to include only that spending which would affect the relevant illnesses. Despite these two drawbacks, in the initial stage of the instrumented models, the coefficient on spending is negative and significant, albeit with significant concerns about the strengths of the instruments. In the underlying stage, there also appear to be issues with the instruments.

While the relationship between spending and amenable mortality does not appear to be accurately captured in any of the amenable mortality models, with the inclusion of the fixed effects re-decomposition the standardising variables fit with expectations, leading to a pro-poor standardised mortality.

Two general caveats should be noted for all models. Firstly, the large contributions of the fixed effects, and their high correlation with mortality, show that these panel data sets do not vary substantially over time. Within the three models considered, the fixed effects contribute 67.3% (cancer), 204% (CHD) and 254% (amenable) of the inequalities in mortality. By contrast, the time dummies (the fixed effects' analogues) contribute

1.4%, 2.7% and 0.5% respectively. Though fixed effects still provide consistent estimation, lack of dynamic variation reduces the relevance of the initial regressions' estimates. The second general caveat is the difficulty in appealing to intuition for the instruments in the underlying regression on the fixed effects. The fixed effects themselves are a composite term, seemingly dominated by mortality, and as such we use the instruments that make sense for the amenable mortality.

In the future, this analysis could be extended to cover further conditions and a longer time period. However, given the change in NHS commissioning this will depend on the availability and suitability of new data. A more direct extension would be to find a way to incorporate Jones and Nicolás' mobility index into the inequality analysis [39]. This would permit more detailed examination of how inequality is varying over time.

Overall, however, this chapter provides further support for the findings in Chapter 3, as well as exploring how the fixed effect re-decomposition of Chapter 4 can be applied within England. The difficulties in finding an appropriate model for amenable mortality are indicative of the challenges macro-level analysis faces when a wide range of illnesses or a diverse set of programme budgets are combined. For cancer the panel model results fit quite well with the results from Chapter 3. The contribution of cancer spending to cancer mortality is pro-rich, at 0.0381, and against the standardised cancer mortality CI of 0.018 appears to reduce health inequality by approximately three quarters. The CHD results show greater inequality in CHD deaths, once standardised. It is not clear if this is due to the longer time scale - this could be a consequence of the necessary change of ranking variable from IMD to JSA. The effect of CHD spending on health inequality is very small, at 0.00117 it only reduces the standardised CHD CI by 1%. This lack of effect is caused by the relatively even distribution of CHD spending between rich and poor areas.

What we have seen in Chapter 3 and confirmed in this chapter, is that substantial if not dominant contributions to inequality are made by health spending in the case of cancer, but in the case of CHD, though spending seems to be effective, its distribution is even.

Chapter 6

The effect of health care on individual-level health inequality

6.1 Introduction

In this chapter we analyse the effect of health care on health inequality at the individual level. We use wave 18 of the British Household Panel Survey (BHPS) which took place between September 2008 and April 2009. The households were linked to their geographical Primary Care Trust (PCT) to allow the effect of PCT-level spending to be studied. The following analysis is split into three parts: first, we look at the effect of PCT-level spending on the utilisation of health care at the individual level; next, we estimate the effect of health care utilisation on health while controlling for a wide range of the determinants of health described in Chapter 1; finally, we conduct concentration index analysis at each stage to consider the effect of spending on inequality in health care utilisation and the effect of health care utilisation on inequality in health.

A number of papers have used individual-level surveys to examine determinants of health [70] [24] [39] [41] [64] [65], and a number have used surveys to identify the factors that influence health care utilisation [49] [66] [11]. The intersection, however, is fairly small. As explored in Chapter 2, Mangalore [44] constructs a three equation model of health,

health care and income. However, she does not estimate an effect of health care on health, instead she splits the sample into the sub-group that accesses health care and the sub-group who that does not, before estimating what effect the covariates have on health. A more relevant analysis is presented in Gravelle et al. [31], which looks at the effect of GP supply on self-assessed health. Using Health Survey for England (HSE) data for 1998, 1999 and 2000, linked with area-level data on local and GP supply characterstics, Gravelle et al. exploit two instruments - house prices and capitation payments - to control for endogeneity in the relationship between health care and health. The effect of GP supply is positive but insignificant when endogeneity is not accounted for, and becomes positive and significant when instruments are used. This highlights the need to correct for endogeneity in the relationship between health and health care. The exact mechanism by which GP supply affects health care is not defined, but factors such as reducing access costs and longer consultations are noted as possible explanations.

Based on the model in Figure 1.1, the analysis in this chapter focuses on the relationship between PCT spending, health care utilisation and health. We assume that PCT spending affects health care utilisation directly, and health care utilisation affects health but that this latter relationship is bi-directional. Health care utilisation is measured by number of uses. There may (and probably will) be effects of health spending on the overall quality, and therefore efficaciousness, of health care utilisation but we do not address these due to lack of data.

At each stage we control for additional confounding factors. The initial stage of health care utilisation determination for this chapter's analysis given by

$$U_i = f(S_a, X_i, I_i) \tag{6.1}$$

where U_i is the measure of health care utilisation, which is a function of area-level health

spending (S_a), personal characteristics (X_i) and interview features (I_I). This last variable is necessary to control for variable lengths of reference time within which health care utilisation is measured.

The determination of health is given by

$$H_i = g(U_i, X_i, I_i, lH_i) \tag{6.2}$$

where U_I is self-assessed health, and we include an additional term (lH_i) for the previous year's self-assessed health. The variable lH_i is included to control for factors, either health-related or due to personal perspective, that might influence reported self-assessed health. By controlling for previous health, the model also implicitly treats the effect of health care change in the level of health - this is attractive because the benefits of health care are in general going to be enjoyed by people whose health is not perfect but was worse, rather than individuals with currently perfect health.

To keep this exploratory analysis simple, the dataset used is a cross-section representing one wave of the BHPS. In the future the models used here can be scaled up to a panel dataset in a similar manner to the scaling up of Chapter 3 to Chapter 5.

We focus on two health care utilisation variables, the number of GP appointments and number of inpatient days in hospital. These were chosen due to their availability in the BHPS. Furthermore, as GPs act as the gatekeepers to the NHS and inpatient stays include the most intensive forms of health care, these two instances of health care utisilation cover a wide range of examples of patients' interaction with the NHS.

6.2 Methods

6.2.1 Regression of health care utilisation on health care spending

The models used are to some extent dictated by the format of data available in the BHPS.

The BHPS question pertaining to GP contacts allows for responses within five categories: no visits, 1-2 visits, 3-5 visits, 6-10 visits and 10+ visits. As responses are grouped we use interval regression. The spending variable related to GPs is the programme budget containing GP budgets (23).

The variable for hospital days takes integer values, is strictly positive and has a long tailed distribution. The sample mean is 0.704 and the sample variance is 41.3, with a minimum of zero and a maximum of 280. Our analysis of the effect of spending on hospital days therefore uses negative binomial regression, as the dependent variable is over-dispersed. There is no hospital spending variable available, so we use all spending minus programme budgets for public health, social care, GPs and strategic health authorities. Obviously this money is not all being spent in hospitals, let alone on inpatient stays, but it is only appropriate to exclude spending that will not have a contemporaneous effect on hospital inpatient stay and health.

For both models, a wide range of controls were included. To control for seasonal and time effects, we included the season of interview as well as the number of days between the interview and the reference point (1st September 2007) used for number of GP visits and inpatient days. Dummies for women and pregnant women were included. Age was included in linear and squared form, as well as multiplied by the dummy for women. Dummies were included for marital status, with divorce combined with separation. Social class of most recent job was included, as well as highest educational qualification. Last month's household income and usual number of daily cigarettes were included. Ethnicity was not included as a control due to lack of observations (<1%). The reference respondent

was interviewed in winter, not pregnant, male, single, has/had an unskilled job, and has no qualifications.

6.2.2 Regression of health on health care utilisation

For the analysis of health we are faced with a number of problems. Firstly, health utilisation is predominantly a result of ill health, meaning endogeneity through reverse causality biases simple analysis. To combat this, we use instrumental variables in a two-step procedure. For both GP and hospital models we use one common instrument: a dummy that indicates whether the respondent believes local health services to be poor or only fair (as against good or excellent). The instrument works on the intuition that on an individual basis the benefits of health care utilisation are balanced against the cost (in terms of time and effort) in acquiring it. Believing medical services are only fair or poor suggests perceived benefits are lower than for those who thought services were good or excellent, and we would therefore expect less health care utilisation, and for the instrument to have a negative coefficient. A counterargument to the use of this instrument is that if these beliefs are founded and certain areas do have poor health services, it would be a predictor of both the level of utilisation and the effect of utilisation. In this case the variable should probably be used to separate the areas of high-quality service provision from the areas of low-quality service provision and separate models run. However, we do not think it likely that beliefs regarding local medical service provision are based on a comprehensive understanding of local medical service quality, instead we contend that they are most likely based on personal experience or anecdotal evidence. In support of this view, the within-PCT variance is almost three times bigger than the between-PCT variance (0.446 vs 0.166). For GP services there is an additional instrument: the spending per capita on GP services. This instrument is an obvious candidate, as any effect on health must come through actual GP utilisation. We expect GP spending per capita to be positively correlated with the number of GP visits. For hospital days, a different additional instrument is use: a dummy indicating whether the individual uses alternative medicine. The intuition

behind this is that those who trust alternative medicine are less likely on average to rely on inpatient services, and so this instrument is expected to have a negative coefficient.

The second problem we face is the choice of health variable, because the BHPS does not contain a health index, only self-assessed health for the past 12 months on the Likert scale. Normally, this would mean the only option is to use an ordered probit or logit, which would be highly problematic for the subsequent inequality analysis as Concentration Indices require variables to be measurable on a ratio scale. However, as wave 14 of the BHPS includes the SF-6D, we follow Doorslaer and Jones (2003) [65] and Gravelle et al. [31] in using the cut-off values of the SF-6D for self-assessed health in wave 14 as the cut-off values for self-assessed health in our dataset. This transforms the ordinal groups of self-assessed health into interval groups of predicted SF-6D. From there, we use interval regression on these intervals. Further, this means the underlying linear prediction of the interval regression is an estimation of the SF-6D health index. Lacking a more objective measure of health, the terms 'health' and 'self-assessed health' are used interchangeably from this point.

The set of controls used in the regressions of health care utilisation on health care spending is retained and augmented with the previous year's self-assessed health. Previous year's self-assessed health is included to control for self-assessed health prior to health care utilisation.

Normally, instrument validity would be checked with a Sargan-Hansen test of overidentifying restrictions. This is calculated from a regression of the second stage residuals on the instruments and all covariates. In our analysis of self-assessed health, the observations are intervals, the cut-off points of which are imputed from the SF-6D. To attempt to provide a similar test of overidentifying restrictions, we create residuals by subtracting from the observed intervals the predicted underlying SF-6D index. These residuals are themselves intervals. We regress these residuals on the instruments and covariates using interval regression. The pseudo-Hansen test reported is the overall significance of this regression.

6.2.3 Inequality analysis

Concentration index decomposition is used to explore the inequality of health spending, health care utilisation and health. For both inpatient stays and GP visits, the determinants of self-assessed health and the determinants of health care utilisation are estimated according to the previously described regressions. As GP visits are recorded as intervals, the concentration index for GP visits is based on the underlying index of the interval regression of GP visit intervals on GP spending and our set of controls. As the underlying index of the interval regression is the prediction of the number of GP visits, it is a suitable variable for concentration index analysis.

Equations 6.3 and 6.4 show the decomposition for the effect of spending on utilisation and the effect of utilisation on health respectively.

$$CI_U = e_{S,U}CI_S + \sum e_{X,U}CI_X + \sum e_{I,U}CI_I + GCI_\varepsilon \tag{6.3}$$

$$CI_H = e_{U,H}CI_U + \sum e_{X,H}CI_X + \sum e_{I,H}CI_I + \sum e_{lH,H}CI_{lH} + GCI_\upsilon \tag{6.4}$$

The concentration index of health care utilisation (CI_U) is decomposed into the contribution of the variables in Equation 6.1, which are combinations of the elasticity of the variable with respect to U multiplied by the concentration index of the variable. The analogous decomposition of health (CI_H) uses the variables of Equation 6.2. Both decompositions also include a term for the contribution of the residuals.

6.3 Data

The data are described in Table 6.1. All data are from wave 18 of the BHPS except for health spending data (which is from the Department of Health programme budgeting

6.3 Data

Variable	Mean/prop.	Std. Dev.	Min.	Max.
SA health: excellent (1 in SF-6D)	0.21	0.408		
SA health: good (0.6664 to 1 in SF-6D)	0.491	0.5		
SA health: fair (0.5241 to 0.6664 in SF-6D)	0.216	0.411		
SA health: poor (0.4347 to 0.5241 in SF-6D)	0.069	0.254		
SA health: very poor (0 to 0.4347 in SF-6D)	0.0136	0.116		
No GP visits	0.235	0.424		
1-2 GP visits	0.378	0.485		
3-5 GP visits	0.213	0.41		
6-10 GP visits	0.107	0.309		
10+ GP visits	0.0663	0.249		
Hospital days	0.704	6.42	0	280
GP spending per capita (£000s)	0.232	0.0426	0.133	0.345
Hospital spending per capita (£000s)	1.31	0.0455	1.12	1.6
Previous year's SAH: excellent	0.226	0.418		
Previous year's SAH: good	0.489	0.5		
Previous year's SAH: fair	0.208	0.406		
Previous year's SAH: poor	0.0619	0.241		
Previous year's SAH: very poor	0.0148	0.121		
Days	398	26.2	366	576
Spring	0.00414	0.0642		
Winter	0.122	0.328		
Autumn	0.873	0.333		
Maternity	0.0176	0.131		
Female	0.546	0.498		
Age	48.2	18.15	18	98
Age squared	2650	1870	324	9604
Age × Female	26.3	27.6		
Married	0.564	0.496		
Cohabiting	0.13	0.336		
Separated	0.1	0.3		
Widowed	0.0661	0.248		
Professional	0.0371	0.189		
Managerial	0.27	0.444		
Skilled Manual	0.121	0.326		
Skilled non-Manual	0.187	0.39		
Lower skilled	0.117	0.321		
Never worked	0.0122	0.11		
GCSEs or equivalent	0.141	0.348		
A levels or equivalent	0.121	0.326		
Degree or other higher qualification	0.526	0.499		
Last month's HH income (£)	1604	1703	0	56900
Cigarettes	2.74	6.57		
Medical services not good	0.3	0.458		
Alternative medicine	0.0471	0.212		

Table 6.1: Summary statistics (n=5070)

project); the previous year's self-assessed health (which is from wave 17 of the BHPS); and the cut-off points in the SF-6D (which were extracted from wave 14 of the BHPS). Individuals for whom there was no data on any of the required variables of interest were dropped, as were those under the age of 18, which left 5070 observations.

6.4 Results

6.4.1 Regression of health care utilisation on health care spending

Table 6.2 contains the results of the regressions of health spending on health utilisation. For GP visits, the interval regression finds a positive effect of GP spending on GP visits, though only significant at the 10% level. There is no signifcant effect of time of interview. Being female increases the average number of GP visits, though this effect diminishes with age. Age squared is positive and significant at the 10% level, suggesting age increases the number of GP visits at an increasing rate. Those who work at the professional and managerial levels, as well as those who have never worked, go to the GPs less often. Finally, those who smoke have slightly fewer GP visits.

The effect of spending on hospital days is much greater in statistical significance and positive. The number of days since 1st September 2007 has a counter-intuitive statistically significant negative coefficient, however this is counter-balanced by the statistically significant negative coefficient of an 'early' interview in Autumn and the slightly positive coefficient of a 'late' interview in Spring. When either days or season of interview is dropped, the other is not statistically significant, implying that there is not a strong overall effect of early or late interview. Pregnancy increases hospital stays. Being separated or divorced increases the number of hospital days. Having a professional job slightly reduces the number of hospital days, but is only significant at the 10% level. Smoking slightly increases the number of hospital days, but again is only significant at the 10% level.

	Int. reg.: GP visits		Neg. bin. reg.: Hospital days	
	Coef	S.E	Coef	S.E
GP spending	2.24*	1.27		
Hospital Spending			5.25***	1.9
Days	0.00377	0.00335	-0.0203***	0.00615
Spring	1.03	0.821	2.5*	1.44
Autumn	0.178	0.218	-1.23***	0.442
Pregnant	0.695	0.612	2.97***	0.269
Female	1.97***	0.335	.523	0.565
Age	-0.0228	0.0232	-0.0117	0.0301
Age squared	0.000475*	0.000247	0.00371	0.00027
Female × Age	-0.0194***	0.00704	-0.00422	0.00981
Married	0.182	0.168	-0.0033	0.292
Cohabiting	-0.123	0.225	-0.527	0.415
Separated	-0.102	0.255	0.8**	0.367
Widowed	-0.00532	0.351	0.0384	0.434
Professional	-0.543**	0.274	-0.843*	0.477
Managerial	-0.452**	0.204	-0.382	0.32
Skilled	0.013	0.245	-0.55	0.382
Skilled non-manual	-0.234	0.209	-0.0709	0.335
Lower skilled	0.0204	0.272	-0.407	0.363
Never worked	-1.03*	0.547	-0.467	0.685
GCSEs	-0.253	0.204	0.0206	0.294
A levels	-0.107	0.223	-0.462	0.334
Degree	-0.0814	0.174	-0.222	0.24
HH income	0.0167	0.0185	0.0447	0.0346
Cigarettes	-0.0146*	0.00764	0.0264*	0.0112
Tests	Value	P value	Value	P value
Wald $\chi_{(24)}$	261***	<0.001	388***	<0.001

Table 6.2: Standard errors clustered at the PCT level, cross-sectional weights used. *** is significant at the 1% level; ** at the 5% level; * at the 10% level

6.4.2 Regression of health on health care utilisation

The results of the IV regression of GP visits on self-assessed health are presented in Table 6.3. In the first stage, both instruments take the expected sign but suffer from lack of significance at the 5% level. Perceived lack of quality in local medical services reduces the number of GP visits, while spending on GP services increases GP visits. The instruments are jointly significant at the 5% level. The instruments' validity is confirmed by the Hansen

J test's lack of significance.

In the second stage, instrumented GP visits have a positive but insignificant effect on health. Previous good health is the most statstically significant important predictor of current health, though working a professional job does have positive effect on health and is significant at the 5% level. No other variables are significant at the 5% level, however at the 10% level pregnancy lowers self-assessed health, working at a managerial level or having GCSE or A level qualification increases health. Finally, higher household income increases health.

The results of the IV regression of hospital days on self-assessed health are presented in Table 6.4. In the first stage, both instruments take the expected sign and are significant at the 1% level. Perceived lack of quality in local medical services significantly reduces the number of hospital days and while using alternative medicine reduces hospital stays, fulfilling its expected role as a substitute of medical care. The instruments are jointly significant at the 1% level and their validity is confirmed by the Hansen test's lack of significance.

In the second stage instrumented hospital days have positive and significant effect on health. Previous good health is still the most statstically significant important predictor of current health. The inclusion of instrumented hospital days in place of instrumented GP visits changes the results for some of the covariates. Coefficients on pregnancy, A levels and income are larger in size and all now significant at the 5% level. Coefficients on GCSEs, and having mangerial or professional jobs are now insignificant at the 10% level. Finally, there is a negative and statistically significant effect of the number of cigarettes smoked on health.

	1st Stage: GP visits		2ns Stage: Health	
	Coef	S.E	Coef	S.E
Instrumented GP visits			0.038	0.0526
Prev V good SAH	-0.739	0.698	0.410***	0.0616
Prev good SAH	-0.00559	0.694	0.273***	0.0497
Prev fair SAH	1.25*	0.692	0.0801	0.088
Prev bad good SAH	0.974	0.706	-0.00163	0.0713
Days	0.00369	0.00333	-0.000397	0.000327
Spring	0.984	0.857	-0.0322	0.0962
Autumn	0.174	0.219	-0.00672	0.0195
Pregnant	0.674	0.631	-0.0784*	0.054
Female	1.82***	0.34	-0.0672	0.0941
Age	-0.032	0.023	0.000633	0.00361
Age squared	0.000509**	0.000242	-0.0000186	0.0000435
Female × Age	-0.0173**	0.00687	0.000616	0.000968
Married	0.172	0.169	-0.013	0.0184
Cohabiting	-0.137	0.217	-0.00267	0.0176
Separated	-0.104	0.254	-0.01	0.0208
Widowed	-0.0663	0.355	-0.00376	0.0269
Professional	-0.472*	0.249	0.0638**	0.0281
Managerial	-0.318	0.196	0.0321*	0.02
Skilled	0.0393	0.235	0.00612	0.016
Skilled non-manual	-0.164	0.2501	0.018	0.0233
Lower skilled	-0.228	0.26	0.00723	0.0188
Never worked	-1.3**	0.543	0.0485	0.0794
GCSEs	-0.136	0.201	0.0281*	0.0189
A levels	-0.0131	0.227	0.0261*	0.0176
Degree	0.0324	0.175	0.0133	0.0156
HH income	0.00625	0.0187	0.00278*	0.00207
Cigarettes	-0.0233***	0.00755	-0.000689	0.00137
Medical services not good	-0.228*	0.128		
GP spending	2.41*	1.31		
Tests	Value	P value	Value	P value
Wald $\chi_{(29)}$	451***	<0.001		
First stage Wald $\chi_{(2)}$	6.39**	0.0409		
Wald $\chi_{(28)}$			4222***	<0.001
Pseudo-Hansen test $\chi_{(29)}$			10.7	0.9992

Table 6.3: Standard errors in first stage clustered at the PCT level, standard errors in second stage based on 2000 boostrap replications, cross-sectional weights used. *** is significant at the 1% level; ** at the 5% level; * at the 10% level

6.4 Results

	1st Stage : Hospital days		2nd Stage: Health	
	Coef	S.E	Coef	S.E
Instrumented hospital days			0.0486**	0.0234
Prev V good SAH	-3.95***	1.4	0.573***	0.131
Prev good SAH	-3.7***	1.42	0.451***	0.129
Prev fair SAH	-3.46**	1.42	0.296**	0.127
Prev bad good SAH	-1.88	1.64	0.127	0.121
Days	-0.00563	0.00568	0.0000438	0.00036
Spring	0.0883	0.917	-0.0017	0.0724
Autumn	-0.735	0.464	0.036	0.0245
Pregnant	4***	0.364	-0.246**	0.101
Female	-1.19	0.771	0.0593	0.0473
Age	-0.124	0.0856	0.00559	0.00546
Age squared	0.00128*	0.000838	-0.0000629	0.0000552
Female × Age	0.0284	0.0182	-0.00141	0.00109
Married	-0.0371	0.267	-.0056	0.0173
Cohabiting	-0.361	0.231	0.0098	0.0164
Separated	0.427	0.513	-0.0353	0.0286
Widowed	0.698	0.809	-0.0421	0.0492
Professional	0.259	0.508	0.033	0.0324
Managerial	0.328	0.519	0.00424	0.0323
Skilled	-0.0123	0.496	0.00758	0.029
Skilled non-manual	0.213	0.515	0.000748	0.0313
Lower skilled	-0.0922	0.52	0.00185	0.0296
Never worked	0.023	0.66	-0.0025	0.0427
GCSEs	0.205	0.427	0.0133	0.0253
A levels	-0.36	0.269	0.0434**	0.018
Degree	-0.136	0.251	0.0215	0.0144
HH income	-0.00537	0.015	0.00341**	0.00138
Cigarettes	0.0196	0.0171	-0.00254**	0.00123
Medical services not good	-0.378***	0.14		
Alternative medicine	-0.519***	0.146		
Tests	Value	P value	Value	P value
F(29, 149)	9.48	<0.001		
First stage F(2, 149)	6.94	0.0013		
Wald $\chi_{(28)}$			4190***	<0.001
Pseudo-Hansen test $\chi_{(29)}$			0.03	1

Table 6.4: Standard errors in first stage clustered at the PCT level, standard errors in second stage based on 2000 boostrap replications, cross-sectional weights used. *** is significant at the 1% level; ** at the 5% level; * at the 10% level

6.4.3 Inequality analysis

Table 6.5 presents the results of the inequality analysis based on the GP results of Table 6.3. Predicted GP visits show a statistically significant pro-poor distribution. GP spending is significantly pro-rich, the elasticity of GP spending is positive but only significant at the 10% level, and the resulting contribution is pro-rich but not statistically significant. The elasticity suggests that a 10% increase in GP spending only translates to a 1% increase in GP visits. As in Table 6.2 , there is no effect of day or season of interview on GP visits, but the concentration indices for these variables are all significant and show that households with higher household income are interviewed later in the interviewing window. The women in the sample tend to be in significantly poorer households and, combined with the increased GP visits which women make, this leads to a pro-poor contribution of gender to the concentration index of GP visits. Age, both in its linear and squared terms, has a pro-poor distribution. In its squared term this distribution combines with a significantly positive elasticity to make a pro-poor contribution to GP visits.

The concentration indices for being married or cohabiting are positive and significant, whilst for being separated or widowed they are negative and significant. However, as the ranking variable is household income (which should increase with the number of potential employed people) this result is trivial. The concentration indices for type of job are significant and pro-rich for skilled, managerial and professional jobs and negative but insignificant for the 'lower skilled' or 'never worked' categories. The significant negative elasticities of managerial and professional jobs lead to significant overall pro-poor contributions for these two variables. That is, being in a managerial or professional job means an individual is richer and goes to the GP less often. The concentration indices for education show owners of degrees, and to a lesser extent A levels, have pro-rich distribution while GCSE holders show a pro-poor distribution. As expected, household income shows a pro-rich distribution. The number of cigarettes smoked reduces the number of GP visits, and as the variable has a pro-poor distribution, this leads to a pro-rich contribution

- however, this contribution is not statistically significant. The generalised concentration index of errors is 9.1% of the size of the concentration index of GP visits and is statistically significantly different from zero.

Variable	Elasticity	S.E	CI	S.E	Cont.	S.E
GP visits			-0.0303***	(0.0051)		
GP Spending	0.103*	(0.0626)	0.00465***	(0.00157)	0.000481	(0.000354)
Days	0.296	(0.275)	0.00726***	(0.000572)	0.00215	(0.002)
Spring	0.00742	(0.0007)	0.33**	(0.145)	0.00245	(0.000303)
Autumn	0.0314	(0.0444)	-0.0207***	(0.00295)	-0.000652	(0.000935)
Pregnant	0.00161	(0.00139)	0.11	0.0768	0.000177	(0.000226)
Female	0.208***	(0.0347)	-0.0433***	(0.00847)	-0.009***	(0.00235)
Age	-0.228	(0.24)	-0.0891***	(0.00308)	0.0204	(0.0213)
Age squared	0.275**	(0.138)	-0.177***	(0.00536)	-0.0485**	(0.0242)
Female × Age	-0.104***	(0.036)	-0.138***	(0.00969)	0.0144***	(0.0051)
Married	0.0202	(0.0215)	0.0928***	(0.00795)	0.00187	(0.002)
Cohabiting	-0.00222	(0.00343)	0.172***	(0.0224)	-0.000382	(0.000588)
Separated	-0.00193	(0.00504)	-0.218***	(0.0267)	0.00042	(0.00111)
Widowed	-0.000862	(0.00552)	-0.576***	(0.0229)	0.0000497	(0.00318)
Professional	-0.00371*	(0.00197)	0.422***	(0.0369)	-0.00157*	(0.000845)
Managerial	-0.0221**	(0.0105)	0.0343***	(0.0138)	-0.00759**	(0.00361)
Skilled	0.000306	(0.00542)	0.0737***	(0.0214)	0.0000226	(0.000416)
Skilled non-manual	-0.00824	(0.00791)	0.118***	(0.0184)	-0.000969	(0.00095)
Lower skilled	-0.00455	(0.00558)	-0.00335	(0.0234)	0.000152	(0.000259)
Never worked	–0.00275*	(0.00167)	-0.0252	(0.0819)	0.0000692	(0.000258)
GCSEs	-0.00702	(0.00601)	-0.0903***	(0.0215)	0.000634	(0.000592)
A levels	-0.00244	(0.00514)	0.117***	(0.024)	-0.000285	(0.000612)
Degree	-0.00816	(0.0176)	0.172***	(0.00874)	-0.0014	(0.00303)
HH income	-0.0112	(0.0139)	0.381***	(0.00495)	-0.00425	(0.00529)
Cigarettes	-0.00756*	(0.00456)	-0.0788***	(0.021)	0.000596	(0.00042)
Generalised CI_ϵ					0.00277***	(0.000472)

Table 6.5: Results from GP visit decomposition; all standard errors are bootstrapped based on 2000 replications, cross-sectional weights used. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=5070)

Table 6.6 presents the results of the inequality analysis based on the hospital days results of Table 6.3. Hospital spending has a significantly positive elasticity and displays a significantly pro-poor distribution, so its contribution to the concentration index of hospital days is pro-poor. The effect of spending is large, suggesting that a 10% increase in spending translates to a 69% increase in hospital days. The concentration indices for all covari-

ates are the same as in Table 6.5. The significant effects of day and season of interview observed in Table 6.3 reappear here. The effects conflict: according to number of days since 1st September 2007, those interviewed later went to hospital less, whilst according to the seasonal dummies those interviewed earlier went to hospital less. Combining the contributions of day and season of interview results in an overall contribution of -0.0328. Pregnancy increases the the number of hospital days, but makes no statistically significant contribution to the concentration index of hospital days as the concentration index of pregnancy is not statistically significant. Being separated or divorced increases the number of hospital days but is only significant at the 10% level. This leads to a contribution, also significant at the 10% level, of -0.0165. The generalised concentration index of errors is larger than in the model for GP visits, but is not statistically significant.

Table 6.7 presents the results of the inequality analysis based on the GP results of Table 6.3. Health has a pro-rich distribution. The elasticity and contribution of GP visits if not statistically significant. The effect of previous health status is strong and statistically significant for 'very good' and 'good' previous health. These two health stati were also pro-rich, leading to pro-rich contributions to current health. A previous health status worse than 'good' shows pro-poor distribution but has an insigificant effect on current health. Only three covariates have statistically significant elasticities: having a professional job, having GCSEs, and income. Of those, only the former leads to significant contribution at the 5% level, as the positive effect on health of having a professional job combines with the pro-rich distribution of professional jobs to make a pro-rich contribution. Income also increases health and makes a pro-rich contribution at the 10% level. The generalised concentration index of errors is statistically insignificant, but still contributes 12.2% of the concentration index of health.

Table 6.8 presents the results of the inequality analysis based on the GP results of Table 6.4. Health has a pro-rich distribution. Hospital days increase health, with a 10% increase in hospital days translating to a 4.9% increase in predicted SF-6D health. As hospital

Variable	Elasticity	S.E	CI	S.E	Cont.	S.E
Hospital days			-0.413***	(0.0617)		
Hospital Spending	6.88**	(2.88)	-0.00178***	(0.000344)	-0.0122**	(0.00584)
Days	-8.07***	(2.78)	0.00726***	(0.000561)	-0.0585***	(0.0205)
Spring	0.00909	(0.035)	0.33**	(0.144)	0.003	(0.0134)
Autumn	-1.094**	(0.454)	-0.0207***	(0.00299)	0.0227**	(0.0097)
Pregnant	0.0347***	(0.00635)	0.11	(0.079)	0.00382	(0.00316)
Female	0.279	(0.37)	-0.0433***	(0.00856)	-0.0121	(0.0166)
Age	-0.591	(1.78)	-0.0891***	(0.00303)	0.0527	(0.159)
Age squared	01.08	(0.922)	-0.177***	(0.0053)	-0.191	(0.163)
Female × Age	-0.114	(0.321)	-0.138***	(0.00974)	0.0157	(0.0444)
Married	-0.00185	(0.184)	0.0928***	(0.00799)	-0.00172	(0.0171)
Cohabiting	-0.0482	(0.045)	0.172***	(0.023)	–0.00826	(0.00783)
Separated	0.0761*	(0.0403)	-0.218***	(0.0266)	-0.0165*	(0.00921)
Widowed	0.00314	(0.0403)	-0.576***	(0.0225)	-0.00181	(0.0232)
Professional	-0.0291	(0.0194)	0.422***	(0.0367)	-0.0123	(0.00831)
Managerial	-0.0944	(0.0899)	0.343***	(0.0138)	-0.0324	(0.0308)
Skilled	-0.0655	(0.0519)	0.0737***	(0.0215)	0-0.00483	(0.00422)
Skilled non-manual	-0.0126	(0.0703)	0.118***	(0.0177)	-0.00148	(0.00831)
Lower skilled	-0.0458	(0.048)	-0.0335	(0.0237)	0.00153	(0.00221)
Never worked	-0.00626	(0.0144)	-0.0252	(0.0807)	0.000158	(0.00122)
GCSEs	0.00288	(0.0489)	-0.0903***	(0.0217)	-0.000261	(0.00451)
A levels	-0.0528	(0.044)	0.117***	(0.0257)	-0.00617	(0.00538)
Degree	-0.112	(0.143)	0.172***	(0.00881)	-0.0193	(0.0245)
HH income	-0.15	(0.163)	0.381***	(0.00482)	-0.0573	(0.0618)
Cigarettes	0.0688*	(0.0391)	-0.0788***	(0.0211)	-0.00542	(0.00364)
Generalised CI_ϵ					-0.0728	(0.0552)

Table 6.6: Results from hospital day decomposition; all standard errors are bootstrapped based on 2000 replications, cross-sectional weights used. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=5070)

days are significantly distributed towards the poor, the contribution of hospital days is pro-poor.

As before, the effect of previous health status is strong and statistically significant for 'very good' and'good' previous health, but in addition 'fair' health increases current predicted SF-6D health. Pregnancy increases the number of hospital days, but is not itself distributed in a significantly pro-poor or pro-rich manner, so makes no contribution to the distribution of health. Of the rest of the covariates, only A levels, household income

6.4 Results

Variable	Elasticity	S.E	CI	S.E	Cont.	S.E
Health			0.0288***	(0.00187)		
GP visits	0.249	(0.438)	-0.0303***	(0.00501)	-0.00754	(0.0155)
Prev V good SAH	0.116***	(0.0151)	0.172***	(0.0168)	0.0198***	(0.00306)
Prev good SAH	0.172***	(0.0258)	0.0338***	(0.00941)	0.00582***	(0.00186)
Prev fair SAH	0.0225	(0.0191)	-0.142***	(0.0169)	-0.00319	(0.00272)
Prev bad good SAH	-0.000137	(0.00509)	-0.273***	(0.0313)	0.0000373	(0.00142)
Days	-0.205	(0.158)	0.00726***	(0.000573)	-0.00148	(0.00115)
Spring	-0.000152	(0.000431)	0.33**	(0.146)	-0.0000502	(0.000176)
Autumn	-0.00776	(0.0248)	-0.0207***	(0.00304)	0.000161	(0.000512)
Pregnant	-0.00119	(0.000835)	0.11	(0.0793)	-0.000131	(0.000152)
Female	-0.0465	(0.0596)	-0.0433***	(0.00855)	0.00201	(0.0027)
Age	0.0416	(0.143)	-0.0891***	(0.00312)	-0.00371	(0.0128)
Age squared	-0.0704	(0.103)	-0.177***	(0.00546)	0.0124	(0.0181)
Female × Age	0.0217	(0.0334)	-0.138***	(0.00983)	-0.00298	(0.00463)
Married	-0.0095	(0.0114)	0.0928***	(0.00819)	-0.00088	(0.00106)
Cohabiting	-0.000317	(0.00191)	0.172***	(0.023)	-0.0000543	(0.000335)
Separated	-0.00125	(0.00237)	-0.218***	(0.0268)	0.000271	(0.000501)
Widowed	-0.000399	(0.00227)	-0.576***	(0.0225)	0.00023	(0.0013)
Professional	0.00286**	(0.00131)	0.422***	(0.037)	0.00121**	(0.000571)
Managerial	0.0103	(0.00747)	0.343***	(0.0139)	0.00354	(0.00256)
Skilled	0.000947	(0.00264)	0.0737***	(0.0218)	0.0000698	(0.000219)
Skilled non-manual	0.00415	(0.00443)	0.118***	(0.0173)	0.000488	(0.000528)
Lower skilled	0.00106	(0.00343)	-0.0335	(0.0241)	-0.0000354	(0.000123)
Never worked	0.000844	(0.00129)	-0.0252	(0.0825)	-0.0000213	(0.000114)
GCSEs	0.00511*	(0.00297)	-0.0903***	(0.0209)	0.000462	(0.000283)
A levels	0.00388	(0.00245)	0.117***	(0.0242)	0.000454	(0.000302)
Degree	0.00876	(0.00768)	0.172***	(0.0085)	0.0015	(0.00132)
HH income	0.0122*	(0.00704)	0.381***	(0.00474)	0.00464*	(0.00267)
Cigarettes	-0.00234	(0.00393)	-0.0788***	(0.0211)	0.000184	(0.000319)
Generalised CI_ϵ					-0.00351	(0.00268)

Table 6.7: Results from health decomposition; all standard errors are bootstrapped based on 2000 replications, cross-sectional weights used. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=5070)

and number of cigarettes smoked show a statistically significant effect on health. A levels and household income increase health and are pro-rich, leading to a pro-rich contribution. The number of cigarettes, though significant at the 5% level, does not make a statistically significant contribution to the distribution of health. The generalised concentration index of errors is small and statistically insignificant at only 2.7% of the concentration index of

health.

Variable	Elasticity	S.E	CI	S.E	Cont.	S.E
Health			0.0288***	(0.00194)		
Hospital days	0.0491**	(0.021)	-0.413***	(0.0607)	-0.0203**	(0.00865)
Prev V good SAH	0.161***	(0.0372)	0.172***	(0.0166)	0.0277***	(0.00694)
Prev good SAH	0.285***	(0.0812)	0.0338***	(0.00938)	0.00964**	(0.00389)
Prev fair SAH	0.083**	(0.0352)	-0.142***	(0.0168)	-0.0118**	(0.00516)
Prev bad good SAH	0.0107	(0.0101)	-0.273***	(0.0322)	-0.00291	(0.00281)
Days	0.0226	(0.187)	0.00726***	(0.000557)	0.000164	(0.00136)
Spring	-0.00000803	(0.00035)	0.33**	(0.143)	-0.00000265	(0.000129)
Autumn	0.0416	(0.0297)	-0.0207***	(0.00299)	-0.000862	(0.000636)
Pregnant	-0.00373**	(0.00168)	0.11	(0.0754)	-0.000411	(0.000381)
Female	0.0411	(0.0331)	-0.0433***	(0.00828)	-0.00178	(0.00151)
Age	0.367	(0.371)	-0.0891***	(0.00305)	-0.0327	(0.0332)
Age squared	-0.238	(0.217)	-0.177***	(0.00537)	0.0421	(0.0384)
Female × Age	-0.0497	(0.0383)	-0.138***	(0.00963)	0.00683	(0.00532)
Married	-0.00408	(0.0128)	0.0928***	(0.00809)	-0.000379	(0.00119)
Cohabiting	0.00116	(0.00197)	0.172***	(0.0227)	0.0002	(0.000342)
Separated	-0.00436	(0.00362)	-0.218***	(0.0267)	0.00095	(0.000806)
Widowed	-0.00447	(0.00534)	-0.576***	(0.0228)	0.00257	(0.00308)
Professional	0.00148	(0.00152)	0.422***	(0.0373)	0.000625	(0.000645)
Managerial	0.00136	(0.0108)	0.343***	(0.0138)	0.000467	(0.0037)
Skilled	0.00117	(0.00465)	0.0737***	(0.0218)	0.000086	(0.000362)
Skilled non-manual	0.000173	(0.00747)	0.118***	(0.0178)	0.0000203	(0.000885)
Lower skilled	0.00027	(0.00445)	-0.0335	(0.0236)	-0.00000905	(0.000185)
Never worked	-0.0000436	(0.000752)	-0.0252	(0.0831)	0.0000011	(0.0000652)
GCSEs	0.00242	(0.00459)	-0.0903***	(0.021)	-0.000219	(0.000431)
A levels	0.00646**	(0.00268)	0.117***	(0.0243)	0.000754**	(0.00034)
Degree	0.0141	(0.00969)	0.172	(0.00869)	0.00242	(0.00166)
HH income	0.0149**	(0.00633)	0.381***	(0.00476)	0.0057**	(0.00238)
Cigarettes	-0.0086**	(0.00439)	-0.0788***	(0.0211)	0.000678	(0.000424)
Generalised CI_ϵ					-0.000775	(0.000977)

Table 6.8: Results from health decomposition; all standard errors are bootstrapped based on 2000 replications, cross-sectional weights used. *** is significant at the 1% level; ** at the 5% level; * at the 10% level (n=5070)

6.5 Conclusion

In this chapter we have looked in more detail at the effect of health care on health and health inequality by using individual level data. Both GP spending and hospital spend-

ing affect health care utilisation, although the effect of GP spending is less statistically significant and much smaller. For a 10% reduction in GP spending, average GP visits are expected to fall by 1%; for a 10% reduction in hospital spending, the average number of hospital visits is expected to fall by 69%. As the distribution of GP spending is pro-rich, this means that the contribution of GP spending is weakly pro-rich. However it is insignificant at the 10% level due to the weak effect of GP spending. Hospital spending is pro-poor, and so the contribution of hospital spending to hospital days is pro-poor overall. The effect of utilisation on health is statistically significant only in the model for hospital days. Here, a 10% reduction in hospital days translated to a 5% reduction in health, where health is a prediction based on the SF-6D. As the distribution of hospital days is pro-poor, the contribution of hospital care to health inequality is pro-poor also.

The models for GP visits lack significance. This may be because the number of GP visits are not affected by GP spending, or GP visits themselves may not make a large impact on health. It would seem likely that patients suffering ill health, and with potential for large health improvements due to medical care, are more likely to realise that health gain in specialist services within a hospital than during a relatively brief GP appointment. The other possibility is that the models are not accurately capturing the effects of GP spending on GP visits on health. This may be due to the requirement to use interval level data (which is less precise) to measure these effects. For the regression of GP visits on health, the lack of significance of the instruments is a possible problem, as this suggests that GP visits is not being instrumented properly. Better instruments might lead to a significant effect of GP visits on health.

The models for inpatient hospital days appear to perform better and both instruments exceed the 1% significance level. This may be because the variable is measured more precisely. The main concern for the regression regarding the effect of hospital spending on hospital days is the odd significance of day and season of interview. As previously noted, these effects seem to cancel out. It is also odd that within the interview period, people

in lower income households were interviewed earlier. Repeating the analysis for different BHPS waves and with different surveys might show this to be a consistent pattern or a unique artifact.

More generally, it would be fruitful to explore whether the results from these models were similar for different surveys. In particular, the Health Survey for England has a large set of health and health use variables. Additionally, linking individuals to the PCT in which they reside for more recent dates would allow more detailed health spending data to be incorporated into the model, as the DH national programme budgeting project now includes more specific health spending data. Expanding the number of years covered would also allow dynamics of health spending, health utilisation and health to be studied.

With more accurate usage data and more specific spending categories, it would be possible to explore how and if health care has a differential impact on different groups of people, particularly by socioeconomic group. As a preliminary check of this possibility, for all regressions in this chapter the main independent variable of interest (GP and hospital spending in Table 6.2, GP visits in Table 6.3 and hospital days in Table 6.4) was multiplied by dummies representing quartiles according to household income. The resulting four coefficients were not significantly different from each other, suggesting that there is little differential impact by income group, at least at the level of quartiles.

In the future, as Clinical Commissioning takes over commissioning, it is not clear exactly what data will be available to link individuals to specific health care budgets. This chapter suggests that there was an effect of health spending on health utilisation, and that for hospital inpatients there is an effect of utilisation on health. Comparing such results before and after the implementation of the Health and Social Care Act 2010 would allow some evaluation of the effect of clinical commissioning to be made.

Focusing on the model for hospital days (because we did not find that GP visits had a significant effect on health), it is informative to take a broad view of the pathway from

health spending to health inequality. The concentration index of health spending is -0.00178. Though this is statistically significant at the 1% level, relative to the size of the CIs of other variables this is quite small. The elasticity of health spending is 6.88, which is both large and statistically significant. Because it is so large, the contribution of health spending to health care utilisation inequality is greater than the inequality in health spending (the elasticity acts as a multiplier). Nevertheless, this contribution makes up only a small part (3%) of the inequality in hospital days, which at -0.414 is one of the bigger inequalities in the data. The elasticity of hospital days with respect to health is 0.0491, so the contribution of hospital days to health inequality is -0.0203 which, compared to the CI of health of 0.0288, represents a substantial reduction in health inequalities. Compared with the case where hospital days were either entirely evenly distributed or entirely ineffective at improving health (either case nullifying any contribution of hospital days to health), the predicted effect of the inequality in hospital days reduces health inequalities by 41%. Thus, health spending reduces self-assessed health inequality by just over 1% through its effect on the number of inpatient days.

Chapter 7

Conclusion

7.1 Review

This thesis has explored the effect of health care on health inequality using both individual and area level data. Due to constraints of data we have focused on types of health care that are easier to link to health outcomes. To meet the aims and objectives, this thesis has analysed the effect of health spending on two of the biggest causes of mortality, as well as the effect of GP and inpatient visits on self-assessed health. In this section we will describe how the objectives set out in Chapter 1 have been met.

Objective 1. To use concentration index methods to explore area-level inequality in health spending

In Chapter 3, inequality in NHS spending was analysed, using programme budgeting data to explore how specific disease areas were resourced. Through this analysis, we saw that spending by disease programme varied between richer and poorer areas, with greater spending for conditions associated with older people in richer areas and greater spending for conditions associated with younger people in poorer areas. This suggests that PCTs are not commissioning some generic set of services - their commissioning is

done with their local population's needs in mind and therefore it reacts to differences in demography. Overall spending was neither pro-rich nor pro-poor. Therefore, PCTs face a similar budget constraint whether located in rich areas or poor areas. The decision they face is how they divide this budget between all the possible services their population would like.

This analysis fulfilled Objective 1. Three points of further work could extend this analysis.

Firstly, there is a question as to how inequality in spending changes over time, and how this could best be explored. As discussed in Chapter 5, techniques for looking at inequality over time have been developed, but have generally been applied to individual-level variables. A key challenge in this analysis would be to identify a suitable ranking variable, one that changes over time and measures the socioeconomic status of an area. This would be insightful on two counts; the effect of changes in budget (either due to changes in the budget allocation formula or in reductions in the Distance from Target) on health spending inequality could be examined, additionally the changing priorities of commissioners in richer and poorer areas could be studied.

Secondly, the Health and Social Care Act 2012 abolished PCTs, and created new Clinical Commissioning Groups (CCGs) to commission health care. The idea behind CCGs was to give clinicians, and particularly GPs, a greater say in how services are commissioned in a local area. Being closer to patients, the hope was that clinicians would have a better grasp of what should be commissioned. However, commissioning services and writing contracts is a skill that few clinicians are trained in. Furthermore, due to increasing financial pressure the overarching challenge in commissioning is less 'what should be commissioned' and more 'what can be cut'. Analysis of the inequalities in health spending before and after the Act would make for an interesting evaluation of the effects of the introduction of CCGs.

Finally, the spending variable in this analysis was pounds per capita. As such there was minimal evaluation of what level of health care need this was being spent on. In the

subsequent analysis this was extended to standardise for disease prevalence, but it should be possible to try to account for need in a more advanced manner, taking account of demography, severity of disease and utilisation.

Objective 2. To estimate the effect of health spending on area-level health

The relationship between health spending and area-level health was explored for two major illnesses, cancer and CHD, in order to meet Objective 2. The work of Martin et al. [47] was replicated and updated, using similar instruments and an additional functional form. We found that a decrease in spending of 10% would suggest an increase in mortality of 6.6% for cancer and 6.1% for CHD, thus achieving Objective 2 for two major causes of mortality.

Cancer and CHD were chosen as disease categories because there was data available on them and they represented a major focus of the NHS - to avoid unnecessary mortality. Though they cover only 9% of total spending, they account for 46% of all deaths. Because there is no data on area-level health-related quality of life, the best indicator of outcomes at the area level is mortality, and cancer and CHD are two large causes of death for which there are well-established health care treatments.

Increasingly, however, the pressures on the NHS are due to an aging population with an increasing number of co-morbidities. The focus in the coming years, therefore, will likely move to supporting ill, aging patients live as well as possible, as independently as possible. In this context, it seems there should be a broad stream of work that looks at how health spending can best be used to support this focus. In particular, figuring out a way to measure how successful the NHS is in helping people live with multiple co-morbidities is important, as it will allow best practice to be identified.

Objective 3. To estimate the contribution of health spending to area-level health in-

equality

In Chapter 3 we estimated the contribution of health spending to area-level health inequality for cancer and CHD, accomplishing Objective 3. Inequality was measured through the concentration index, and the contribution of spending to mortality inequality was estimated using concentration index decomposition. The results revealed that cancer spending was pro-poor, therefore reducing inequality in cancer mortality by approximately half. CHD spending was neither pro-poor nor pro-rich, so it did not alter inequality in CHD mortality.

Future work could explore a broader variety of health spending or outcome categories, potentially based on the work in Martin et al [48], to describe in better detail the relative trade-offs commissioners in different areas are making. Similar work on how health care can affect disease prevalence would be another logical extension.

Objective 4. To explore how area-level health inequality can be studied in a panel data context

A method to use concentration index decomposition analysis with area-level panel datasets was introduced in Chapter 4, using an international dataset uncomplicated with issues of endogeneity. The problem was that when using fixed effects estimation on datasets with little time-varying heterogeneity, normal CI decomposition is dominated by the contribution of the fixed effects. Contributions of covariates to inequality that do not vary substantially over time are not measured, leading to difficulties in standardising inequality, for instance for age and population of areas. The solution we proposed was to re-decompose the fixed effects onto the time-averaged variables.

Applying the technique to a non-IV model of international mortality determination provided an example of the clarity that this approach could bring to an otherwise unclear

model. In particular, the effect of GDP at the initial decomposition was to increase deaths. This result would contradict historical data, expert opinion and common sense. Fixed effects re-decomposition allowed the sizable differences between countries to be included in the analysis, leading to a more reasonable pro-poor contribution of GDP. This innovation allowed us to tackle Objective 4 in the subsequent chapter.

In Chapter 5 this technique was applied to the cancer and CHD models of Chapter 3, as well as a broader model of mortality amenable to health care. These three models were complicated by the presence of endogeneity, so multiple combinations of instruments are run. Overall, we found strong evidence that cancer and CHD spending reduces mortality, and reduces mortality in line with the estimates from Chapter 3. The model for amenable mortality did not provide clear results, likely this was a result of poor mapping from spending to mortality, the wide range of conditions included or poor instrumentation. In all three models, mortality was pro-rich after standardisation at the initial stage, and only when long term effects are included in the re-decomposition did mortality become pro-poor. In both cancer and CHD we found that health spending was pro-rich overall due to spending reducing mortality and being distributed in poorer areas.

There were a number of advantages in using this technique. Firstly, it allowed analysis of area-level health inequality, as health at the area level varies very little over time. It also partitions inequality into long-term and short-term components, which is a useful distinction. The disadvantages are that the estimated fixed effects are estimated on a fairly small number of observations, and that the second stage is liable to suffer bias.

Fixed effect redecomposition offers a way to analyse area-level inequality, fulfilling Objective 4. Further work needs to be done to refine the technique, particularly in how to incorporate changes in ranking over time.

Objective 5. To use concentration index methods to estimate individual-level inequality in health care utilisation

7.1 Review

In Chapter 6, our analysis moved away from area-level mortality to individual self-assessed health. Using BHPS data, we found that individuals with greater household income had significantly lower levels of health care utilisation, in terms of both GP visits and hospital days. This accomplished Objective 5. As GP visits were reported in interval form, we used the underlying predicted number of GP visits from an interval regression to come to the GP concentration index estimate. This objective was a means to an end, namely an estimation of the distributional effect of spending on health through health care utilisation.

Objective 6. To estimate the effect of area-level health spending on individual-level health care utilisation

The link between area-level health spending and mortality, as explored in Chapters 3 and 5, depends on health care utilisation at the individual level increasing with spending. This is explored in Chapter 6. Increasing GP spending by 10% leads to an increase in GP utilisation of 1%, however this result is only statistically significant at the 10% level. Increasing hospital spending by 10%, however, increases the number of hospital days by 69%, and this relationship is statistically significant at the 5% level. These estimates fulfil Objective 6, however they are subject to a few caveats. Firstly, there is no specific hospital spending category in the Programme Budgeting data, so the spending we have used in Chapter 6 will include spending on non-hospital health care. As previously mentioned, with more detailed programme budgeting data more specific analysis can be done. Secondly, the GP result had to be estimated through interval regression. With data on the specific number of GP visits, we would be able to use more efficient techniques, such as negative binomial regression, and generate a more precise and accurate estimate.

Objective 7. To estimate the effect of individual-level health care utilisation on individual-level health

The link between area-level health spending and mortality, as explored in Chapters 3 and 5, also depends on health care utilisation at the individual level reducing mortality. As the BHPS does not have mortality data, we instead look at the effect of health care utilisation on self-assessed health. Self-assessed health is measured on the Likert scale in the BHPS, so we analyse it using interval regression, fixing the cut-off points at the SF-6D score of a previous BHPS wave. In this analysis we find that GP visits have no affect on predicted health, whereas a 10% increase in hospital visits increases predicted SF-6D health by 4.9%. There are a couple of reasons why the GP estimate may not be significant. GPs act as gatekeepers to the rest of the NHS, therefore a large number of GP visits might suggest the GP is no making specialist referrals (where we might expect big health gains to be made). Additionally, as previously mentioned the number of GP visits is imprecisely measured by interval data.

Objective 8. To estimate the contribution of individual-level health care utilisation on individual-level health inequality

In Chapter 6, concentration index decomposition was used to estimate the contribution of individual-level health care utilisation on individual-level health inequality. Health inequality is pro-rich, with a CI of 0.029. The number of GP visits has no statistically significant effect on health, so inequality in GP visits has no effect on health inequality. Number of hospital days does increase health, and as hospital days are pro-poor this means that hospital days have a pro-poor contribution to health - we estimate they reduce the inherent pro-rich inequality by 41%. This accomplishes Objective 8 for GP and hospital days. Clearly there are many other ways individuals can utilise health care, such as through community services, allied health services or drugs. An obvious extension of this analysis would be to use a more detailed survey with a greater number of health use variables, and try to estimate a more comprehensive set of health care utilisation effects.

7.2 Contribution

All the results in this thesis are, to our knowledge, original and new except for the regression models which underpin Chapters 3 and 4 which are based on Martin et al. [47], and Pritchett and Summers [59] and Filmer and Pritchett [30] respectively.

The central academic innovation in this thesis is the technique of decomposing the fixed effects in the concentration index analysis of area-level panal data. As is clear from Chapters 4 and 5, this method shows promise in better describing inequality and its causes when area-level panel data is used. Previously, the only options were to dispense either with panel data or any interest in time-invariant variables. Now fixed effect re-decomposition allows researchers a third choice. There is still work to be done refining this technique, amending it to take more sophisticated account of changes in rank over time, and comparing it to well-established techniques for analysing individual longitudinal studies.

The most important message of this thesis is that health care improves health and therefore its distribution affects health inequality. These findings apply to cancer and CHD programmes of care, as well as inpatient hospital stays more broadly. Through Chapters 3, 5 and 6 we have seen how the distribution of cancer spending and inpatient care have significantly reduced health inequalities, whereas the even distribution of CHD spending has precluded this effect. Though these applications cover only a portion of total NHS health care it is most likely that the reason more sweeping results have not been found is due to issues of data quality or inherent statistical difficulty.

A number of more minor contributions have also been made. The systematic literature review of the effect of health care on health inequality found very little research had been done in the area. This is an important finding as, if reductions in health inequality are a political aim, decision-makers need to know what policy levers are available to them and

what their relative effectivness is. This is particularly crucial when one considers the range of recommended interventions in the Marmot Review [45], and that for a decision-maker with limited resources and constrained scope of power there will inevitably need to be evidence-based prioritisation.

Another contribution was made in the analysis of spending in Chapter 3. Though overall spending was neither pro-poor nor pro-rich, the specific programme budgets found that spending on services for younger people, for instance neonates, infectious diseases and maternity and reproductive health, were pro-poor whereas services for conditions related to older people, for instance cancer, vision and muscoskeletal problems, were pro-rich. Later in the same chapter, the pro-rich CI for both cancer and CHD expected deaths supports this, as expected deaths increase with age.

At the individual level, the estimate of the effect of hospital days on health is an interesting finding because it covers a wide range of illnesses, and because it shows an effect on a health measure based on self-assessed health. The predicted SF-6D ranges from zero to one, so the coefficient of 0.0486 implies that a day in hospital increases a person's health by 4.86 percentage points on average. This seems to us a plausible figure considering the scope of health care available in hospitals. The advantage of the measure of health used is that it implies hospital stays can improve health-related quality of life.

7.3 General caveats

Though our analysis has successfully identified contributions of health care to health inequality, there are a number of caveats to note. More specific caveats regarding specific analyses were discussed at the end of each chapter.

Firstly, as previously stated, analysis has focused on the more tractable aspects of health care. Whether the findings can be extrapolated to all of health care mainly depends on

whether specific types of health care positively impact on health. As so many health interventions are now the subject of cost-effectiveness analyses, it is likely that this is the case. The difficulty is that, at a macro level, insufficient data and the wide variety of health conditions hampers an overall measurement of the contribution of health care factors to health inequality.

Data issues have constrained a number of the analyses in this thesis. Primarily, this has been due to the difficulty of mapping specific health care (however defined) to specific health outcomes. For instance, it would have been interesting to see the effect of area-level health spending on non-mortality health data that included health-related quality of life. Using mortality rates is in some ways a crude way to measure the effectiveness of health spending, considering how many interventions focus on improving health-related quality of life. Similarly, it would have been useful to have specific data on hospital spending for the individual-level analysis in Chapter 6, as the broad collection of programme budgets covers an unknown quantity of non-hospital spending (this has recently become available as programme budgeting data for 2011/12 is decomposed into care setting, including inpatient).

There were additionally a number of data issues within the BHPS. It is possible that integer data on the number of GP visits would result in greater statistical significance in both the regressions of health spending on health care utilisation and the regressions of health care utilisation on health. Similarly, having a good, validated measure of health such as the SF-6D or EQ-5D might improve the performance of the estimated effects of health care utilisation on health.

Though non-linearity in the relationship between health care utilisation and health was explored briefly in Chapter 6, it is possible that in other areas of health care different socioeconomic groups extract substantially different benefits though health care utilisation.

It should also be noted that, due to the endogenous nature of the relationship between

health care and health, instrumental variables have been required in most regressions. Though the intuition behind these instruments is strong, and the statistical tests of validity are always passed, at times some of the instruments' predictive power is not as great as we would hope. It may be the case that as more data becomes available, more accurate results will be found.

For the inequality analysis, concentration index techniques were used. This is a measure of relative inequality and, as such, this thesis does not look at absolute inequality.

7.4 Future research

The data used in this thesis predates the 2010 Health and Social Care Act. As commissioning power transfers to Clincial Commissioning Groups the kind of analysis conducted in this thesis should be replicated to examine how a change in commissioning structure, along with an increase in competition, changes the efficiency and equity of health care.

There are a number of areas in which the Fixed Effects re-decomposition technique could be used. For instance, expanding the analysis in Chapter 6 of the BHPS into a panel dataset would allow inequality in health to be partitioned into long term and short term influences. Finding a way to combine this with Jones and Nicolás health-related income mobility index [39] would allow a highly complex modelling of the dynamics of health and health inequality to be explored.

As previously mentioned, the 2011/12 programme budgeting data includes a breakdown by care setting. Mapping such specific data to individual health conditions would allow for more accurate estimates of the costs of reducing mortality. This would allow a more nuanced description of the effect of health spending on both health and health inequality.

The possibilities of future work are mainly bounded by data availability. As the new

structure of the NHS takes form, with the aim of transparency and choice taking centre stage, there should be much scope to explore how these changes affect health inequality.

7.5 Summary

Overall, this thesis has investigated the effect of health care on health inequality in a variety of ways using a range of techniques. The literature has been surveyed, and the sparsity of research reinforces the importance of the contributions of this thesis. The headline quantitative results are that: cancer spending reduces inequality in cancer mortality by between a half and three quarters; CHD spending, though efficacious, has no effect on health inequality due to its even distribution; inpatient hospital days reduce health inequality by 40%. These three findings indicate the importance, albeit not dominance, of health care to health inequality in England.

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