
Article:
Howdon, D orcid.org/0000-0001-8052-2893 and Rice, N (2018) Health care expenditures, age, proximity to death and morbidity: Implications for an ageing population. Journal of Health Economics, 57. pp. 60-74. ISSN 1879-1646

https://doi.org/10.1016/j.jhealeco.2017.11.001

Health care expenditures, age, proximity to death and morbidity: implications for an ageing population.

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November 1, 2017

Abstract

This paper uses Hospital Episode Statistics, English administrative data, to investigate the growth in admitted patient health care expenditures and the implications of an ageing population. We use two samples of around 40,000 individuals who a) used inpatient health care in the financial year 2005/06 and died by 2011/12 and b) died in 2011/12 and had some hospital utilisation since 2005/06. We use a panel structure to follow individuals over seven years of this administrative data, containing estimates of inpatient health care expenditures (HCE), information regarding individuals' age, time-to-death (TTD), morbidities at the time of an admission, as well as the hospital provider, year and season of admission. We show that HCE is principally determined by proximity to death rather than age, and that proximity to death is itself a proxy for morbidity.

JEL codes: H51; J11; I19.

Keywords: health care expenditures, ageing, time-to-death, morbidity.

Acknowledgements: This is an independent report commissioned and funded by the Policy Research Programme in the Department of Health from the Economics of Social and Health Care Research Unit (ESHCRU). ESHCRU is a joint collaboration between the University of York, London School of Economics and University of Kent. The views expressed are those of the authors and may not reflect those of the funders. Daniel Howdon acknowledges PhD funding from the Economic and Social Research Council under the Large Grant Scheme (RES-060-25-0045). The authors gratefully acknowledge the highly constructive remarks of two anonymous peer reviewers.

1 Introduction

There is concern that the demographic pressures of population ageing will lead to an unprecedented rise in public expenditures to levels unsustainable under current financing arrangements. In the UK in 2013 approximately 17% of the population (11 million individuals) were aged 65 years or over. This represents a rise of 17.3% in this age group on a decade earlier. Projections suggest that by 2050 this group will have increased disproportionately to younger age groups accounting for approximately 25% of the population (Cracknell, 2010)). The growth in the proportion of older individuals is partly due to increased longevity and partly due to the age structure of the population, particularly ageing of the generation of baby boomers of the post war period to the early 1970s. Health care expenditures in the UK have also risen substantially over time both in real terms and proportional to economic growth. Close to the inception of the National Health Service (NHS) net expenditure (net of patient charges and receipts) on the UK NHS in 2050/51 was £11.7b (GBP, in 2010/11 prices); representing 3.5% of Gross Domestic product (GDP). This rose to £121.3b in 2010/11; approximately 8.2% of GDP. Over the twenty-five year period from 1999/00 to 2014/15 expenditure in England has almost doubled to £103.7b (2010/11 prices) with an average expenditure per head of population of £1,900 (Harker, 2012). Abstracting from issues such as technological innovation, the concern is that as the share of the population at older ages rises, the economic burden of providing healthcare will become increasingly unsupportable.

Interest in the link between ageing populations and health care expenditures can be traced back 25 years when the International Monetary Fund (IMF) asserted that 'demographic pressures [in the UK] of an aging population will be associated with increased demand for medical services', and presented descriptive statistics from various countries, showing that older patients, on average, had greater health care costs than younger patients (Heller et al., 1986). A report by the Organisation for Economic Co-operation and Development (OECD) predicted that across Europe population ageing will create a rise in age-related social expenditures from around 19% of GDP in 2000 to around 26% by 2050. Old-age pension payments and expenditure on health and long-term care was deemed responsible for approximately half this increase (Dang et al., 2001). Approaches to predicting expenditure growth vary, but in a simplistic form consists of computing observed expenditures per head for different age-sex groups and multiplying by

projections of the number of people expected to fall into each group. This approach, however, fails to consider the underlying drivers of heath care expenditures and the relative role of age, or, as has been suggested, proximity to death, or underlying levels of disability and ill-health, in determining expenditures and its likely growth (see Gray (2005)).

Additional to projections of population ageing is the potential change in the health profile of the population over time. An 'expansion of morbidity' hypothesis has proposed that the 'net contribution of our successes has actually been to worsen the people's health', as improvements in health care tend to lengthen the lives of those living with illness disproportionately to the effect of such improvements on the lifespan of those living without (Gruenberg, 2005). Should population ageing occur alongside a deterioration of health at older ages, then this will exacerbate impacts on public expenditures. While subsequent academic research into these claims – notably, research in the 'compression of morbidity' and 'red herring' strands of literature – have given reason to suggest that such concerns may have been misplaced or exaggerated, concern over the impact of an ageing population on HCE has persisted. Indeed, even in 2012, the UK's then-Secretary of State for Health claimed that the fact that 'the number of people aged over 85 in this country will double in the next 20 years' was one of two factors in 'costs... rising at an unaffordable rate' (Lansley, 2012). He further argued that 'age is *the* principal determinant of health need'[1], and that local NHS budgets should be recalibrated to be based on this, as a result (Williams, 2012).

This paper uses UK administrative data from Hospital Episode Statistics (HES), and deaths data from the Office for National Statistics (ONS), to consider two related research areas. The first, in line with the 'red herring' thesis advanced by Zweifel et al. (1999), is to explore the determinants of inpatient health care expenditures, with particular attention to the role played by age, time-to-death (TTD), and morbidity. We do this in a unique way by following samples of individuals who died in England, over seven years of HES data from 2005/06 to 2011/12, and constructing a panel on individual health care expenditures and morbidity over this period. We show that TTD dominates age as a key driver of health care expenditures and morbidity characteristics dominate TTD. This finding extends the 'red herring' literature by showing that TTD is itself a 'red herring' and acts as a proxy for morbidity. This links to a second area of research by locating the modelling of health care expenditures for individuals close to

[1]Emphasis ours.

death within the broader literature on prospective prediction of hospital use to inform resource allocation, particularly those based on individual level data and which incorporate information on morbidity (for example, see Iezzoni et al. (1998); van de Ven et al. (2003); Pope et al. (2004); Dixon et al. (2011)).

2 Literature review

2.1 Compression of morbidity

The 'compression of morbidity' strand of literature beginning with Fries (1980) suggests that, '[i]n its simplest form, "the age at first appearance of symptoms of aging and chronic disease can increase more rapidly than life expectancy"' (Fries et al., 2011). Fries (2005) identifies three separate 'eras' of illness and well-being experienced during the 20th Century and beyond: an era of infectious disease, followed by an era of chronic disease, followed by an era described by the author as 'directly related to the process of senescence, where the aging process itself, independent of specific disease, will constitute a major burden of disease'. Senescence – the process of ageing – is characterised by the 'decline of maximal function of [all] vital organs', beginning before any chronic disease takes hold: deaths where this function declines below a level necessary to sustain life, in the absence of any disease occasioning this, may be termed 'natural deaths' (Fries, 2005).

The implications for HCE of an ageing population become less clear in the light of compression of morbidity, and there are two aspects to this which deserve attention. First, as the "age at first appearance of symptoms of aging and chronic disease" increases, individuals can be said to age more healthily: the implications of this for HCE are considered below. Second, the compression of morbidity thesis takes for granted an increase in life expectancy. The implications of this for HCE can be considered at a population level for any given year of spending. Setting aside the causal process for this health ageing (again, considered below), as the average person ages more healthily, they require lower HCE at any given age. As more people live to very old age – for instance, 90 years old – each individual requires lower health spending at that age. The overall picture for HCE is however ambiguous: a larger number of people requiring lower HCE may require greater overall costs at a population level than a smaller number of people requiring higher HCE. Similarly, an individual, who dies at age 90 and requires lower HCE at

any given age than they would had they been born into an earlier cohort, may require greater cumulative HCE over their lifespan than they would had they aged less heathily and died at the age of 70. The implications for HCE in the presence of healthy ageing *and* increased lifespan may differ at an individual level to a population level.

Freedman et al. (2002), in a systematic review covering research that had been conducted between 1990 and 2002 found that many measures of disability and limitations in old age had seen declines in recent years: in particular, a change of -1.55% to -0.92% per year in those reporting any disability during the late 1980s and 1990s. Romeu Gordo (2011) observes a cohort-on-cohort fall in the number of individuals with high levels of disability-related functional problems in their everyday life for those born between 1924 and 1947 in the US. Cutler et al. (2013), using Medicare records from the US, present evidence of an increase in disability-free life between 1991 and 2009. The authors conclude that 'The major question raised by our results is why this has occurred. How much of this trend is a result of medical care versus other social and environmental factors?'.

Cross-country international evidence on the changing patterns of disability rates across nine OECD countries is provide by Jacobzone et al. (2000). Consistent with the above literature, they report evidence of significant falls in severe disability rates. The importance of this issue for forecasting HCE depends upon how changes in mortality, changes in morbidity, and changes in disability occur and interact with each other. If the onset of chronic conditions – those imposing large costs on health systems – can be postponed out of an individual's lifetime, then health care costs may fall as later cohorts enjoy a longer lifespan, with a reduced level of necessary treatment for chronic conditions. Dormont et al. (2006), for instance, find that improvements in morbidity profiles in France between 1992 and 2000 have caused reductions in HCE that more than offset the rise in HCE induced by an ageing population.

The morbidity and disability profile of individuals, according to this research, at any given age has improved over time, leading to health problems being experienced later in life and more closely to death. In the illustrated case (Figures 1 and 2[2]), individuals live up to a longer observed maximum age (indicated by the shift out of the survival curve from S_1 to S_2 in Figure 1), and have a higher observed level of health at all ages (indicated by the shift out of the health status curve from H_1 to H_2 in Figure 2). Both survival curves and health status

[2]Adapted from Fries (1980) and `http://www.aei.org/files/2008/06/27/20080626_WashingtonAEI.pdf`.

curves have become increasingly rectangular. The effect on health care expenditure (HCE) is ambiguous, given that generally more healthy ageing – a decrease in morbidity at any given age – puts downward pressure on HCE, while an increase in life expectancy, *ceteris paribus*, puts upward pressure on HCE. The actual relationship between health care costs and changes in morbidity and mortality profiles at every given age depends upon the changing shape of these two curves, and also the extent to which the changes in each are *due to* or *caused by* the healthcare that creates these HCE. The use of age *per se* in predicting future health care costs should be approached with caution, as a result.

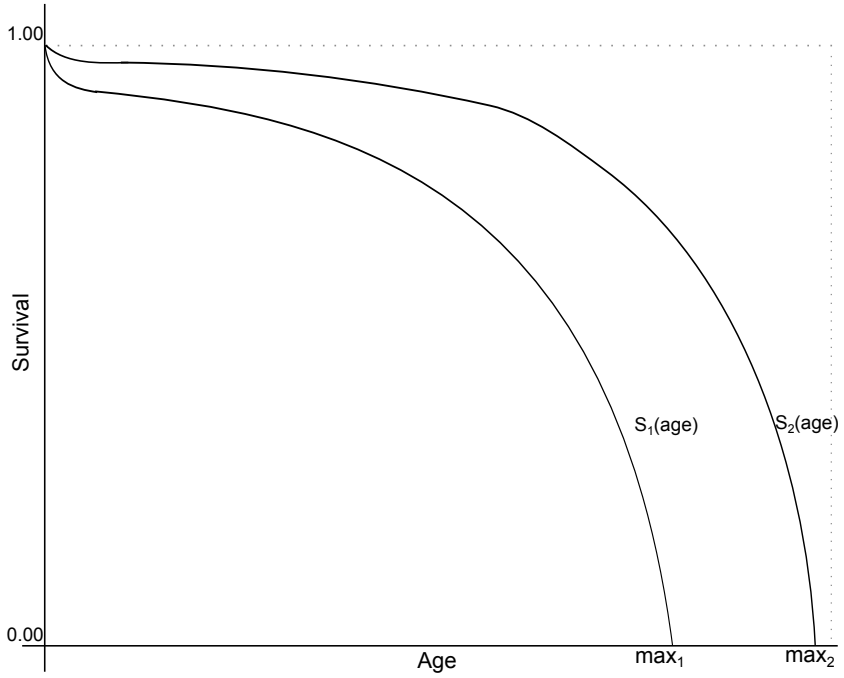

Figure 1: Stylised change in survival curves

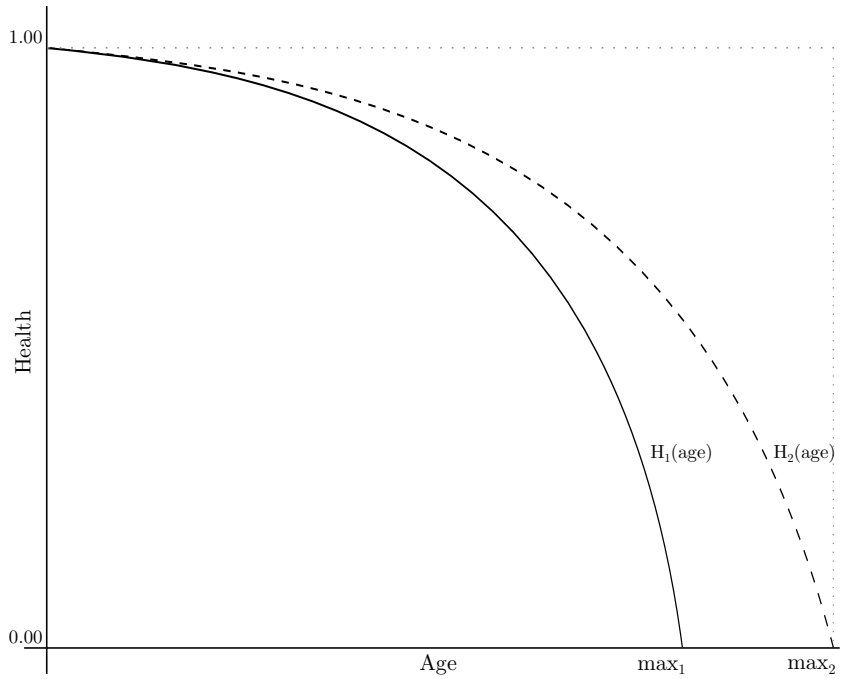

Figure 2: Stylised change in health profiles

2.2 Age, time-to-death and healthcare expenditures

The 'red herring' strand of literature further gives empirical reason to suggest that claims of steeply-rising future HCE due to population ageing[3] may have been exaggerated, potentially owing to morbidity being concentrated in later years of life. Zweifel et al. (1999), using Swiss sickness fund data, find that no effect of age on health care expenditures existed after controlling for TTD, i.e. the time from any given point of observation to death for an individual. Owing to the number of individuals with zero HCE, a two-step model (with a probit first stage and OLS second stage) was employed, with only deceased patients included in the model. Such work was criticised on the grounds of potential endogeneity, with time-to-death affected by both present, previous (and, due to the nature of how TTD must be measured) future HCE. In a subsequent paper, Zweifel et al. (2004) seek to test for such problems, finding that while TTD is endogenous, their results were 'fairly robust' to the error this induces. Werblow et al. (2007) find that age is a small (but statistically significant) determinant of HCE after controlling for TTD for patients using long-term care (LTC), such as those in care homes, and is not associated with HCE for non-LTC patients. More complicated methods, such as those employing generalised linear

[3]HCE may rise due to technological change brought about by new expensive innovations in health care treatments, or due to shifting patterns of morbidity.

models, have since been used, for example by Werblow et al. (2007), in order to deal with the non-normal properties (such as positive skewness) exhibited in the distribution of HCE. These papers have corroborated results obtained using probit and OLS two-step models. Felder et al. (2010), in a recent paper in this series, first predict individuals' survival based on observed HCE and socioeconomic characteristics (in early waves), before using predicted values based on this as an instrument for TTD in explaining HCE in later waves. The authors find that, while TTD cannot be deemed exogenous, any effect of age on HCE becomes insignificant when TTD (or instrumented TTD) is included in the model. Furthermore, results regarding the relative importance of TTD compared to age have also been corroborated in a disease-specific study carried out by Wong et al. (2011).

While use has been made of morbidity markers in models of long-term care expenditures (LTCE) (see de Meijer et al. (2011)), such use has not been made in models explicitly investigating the link between HCE and population ageing. One possibility is that TTD is itself a red herring, in that it is simply a proxy for morbidity, unobserved in existing HCE models in the red herring strand of literature. Such a theory has been provisionally borne out empirically in literature related to economic evaluation of healthcare, with Gheorghe et al. (2015) finding that quality of life (as measured by SF-6D scores) declines with proximity to death, and also by biological and medical literature. Indeed, Dalgaard and Strulik (2014), proposing an alternative life cycle model of ageing, note that previous work in the 'red herring' strand of literature is consistent with biological and medical research (see, *inter alia* Mitnitski et al. (2002*a*,*b*, 2005); Rockwood and Mitnitski (2006, 2007)), showing that conceptions of ageing focusing on time-from-birth (such as that inherent in Grossman (1972)) are erroneous. This model conceptualises the human body as a system which has substantial inbuilt redundancy (that is, an ability to function at a level well over and above that required to sustain life) in youth, but redundancy which declines as 'deficits' (a decline in function of individual parts of the body) are accumulated. Ageing depends not upon a 'biological clock', but is a process of increasing frailty which is the outcome of investments in health, available health technology, the lived environment, and a physiological 'force of aging' parameter. According to such a model, health is predicted to decline at an increasing rate when the individual's health status is lower. The authors note that existing research in the 'red herring' strand of literature, in line with this, 'suggests that health

status (e.g., frailty), and not the year on the birth certificate, is what matters to health investments'. The latent assumption here is that a TTD variable proxies for this health status, which declines as individuals become more morbid as they approach death, and with ever-increasing levels of health investment to (partly) offset this decline in health status and postpone death.

This seems intuitively plausible: in the years before death, it is likely that morbidity will increase, leading to more treatment, and that comorbidities complicating the treatment of the disease bringing about the hospital episode will also increase. Shwartz et al. (1996), in work predating the original red herring hypothesis, note that the inclusion of variables for comorbidities increase substantially the explanatory power of models. It seems likely that, as Dalgaard and Strulik (2014) suggest, variables incorporating 'time-to-death' in more recent models of HCE are picking up, in large part, these comorbidities, which are not included in existing HCE models in the red herring literature. Indeed, de Meijer et al. (2011) conclude that time-to-death 'largely approximates disability' in models of LTCE. Dixon et al. (2011), in proposing individual-level formulae for resource allocation in the UK's National Health Service (often termed 'Person-Based Resource Allocation', or PBRA) include individual level morbidity markers, finding that these have a 'powerful effect... in predicting individual level expenditure'.

The process generating HCE is clearly not a simple function of those explanatory variables used in existing 'red herring' research: the actual data-generating process behind these health care expenditures is unlikely to be characterised accurately by a simple use of age, historical time and time-to-death. In addition to the aforementioned problems surrounding TTD and age as a proxy for morbidity, as Breyer et al. (2014) note, many existing models are likely to be characterised with substantial endogeneity problems, which lead to potential bias in the estimation of the change in HCE as an individual ages or approaches death. The authors control for potential endogeneity introduced by differential treatment based on a physician's view of the patient's expected health benefits from treatment, proxied by actuarial tables of life expectancy conditional on age. If physicians expect individuals to respond differently to treatment, this may cause those who are more likely to respond to treatment to be treated more intensely than those who are not, thus increasing expected HCE for individuals who are younger, further-from-death or with fewer comorbidities because of physician selection. Conversely, HCE for older individuals – or, more likely, individuals in the final years of life – may rise as intensity of

treatment becomes stronger with heroic efforts to save an individual's life, possibly motivated by ethical 'rule of rescue' concerns when faced with an identifiable, gravely sick individual (Jonsen, 1986). Breyer et al. (2014) jointly estimate this possible physician selection based on life expectancy alongside a model for health care expenditures, incorporating both age and time-to-death as explanatory variables. They find that increasing survival rates for the elderly in Germany have positive impacts on HCE, arguing that this is explained by physician selection: treating patients more intensively if they expect positive results from treatment over a longer time span.

Datasets used within the 'red herring' literature are, in general, sickness fund datasets, with only Seshamani and Gray (2004) using population-level (for users of NHS treatment) data, the Oxford Record Linkage Study, a longitudinal dataset of all individuals within an area of Oxfordshire, England. We believe our paper to be the first to use a sample of individuals from a comprehensive national-level dataset of health care users.

The extent to which 'red herring' and related issues are of interest depends upon the intended use of such research. Much existing literature focuses on projections of future health care costs given an ageing population, with the headline results of some papers (such as Stearns and Norton (2004) and Seshamani and Gray (2004)) being the overestimation of expected costs for a given future year when TTD is an omitted variable. This is due to the collinearity between TTD and age for a given individual: an individual who gets one year closer to death also gets one year older, and so the impact of TTD is picked up by age in such models. The inclusion of morbidity markers in addition to, or replacing, TTD would allow greater precision of future estimates where reliable estimates of morbidity prevalence, and the cost of treatments, conditional on age and TTD were known. Certainly, if the compression of morbidity hypothesis holds, and individuals are able to postpone the onset of chronic diseases – with associated higher HCE – to a time period closer to their death, or even indefinitely, explicitly considering morbidity rather than proxying this by age or TTD becomes ever more important.

We build upon the compression of morbidity and red herring strands of existing literature, seeking to further examine the relationship between ageing, time-to-death and health care expenditures. The original red herring hypothesis is that, once time-to-death is included in models of HCE, age *per se* does not explain changes in HCE. While models intended for resource allo-

cation (Dixon et al., 2011) have already included morbidity as an explanatory variable in HCE for the general population, other applications of models of HCE have not – in particular, those focusing explicitly on ageing populations, or costs in the years approaching death.

While hospital inpatient care forms only one part of health and social care incurred later in life, it is responsible for a large proportion of such expenditures. While it may seem that expenditures from other categories of health-related expenditures such as that arising from general practice, prescriptions, and even long-term care should be considered in the aggregate, much richer, more universal and more reliable individual-level administrative data is at our disposal for the UK for hospital expenditures than for other types of expenditure, and information regarding the relationships examined in this paper are likely to be informative for specific avenues of NHS budget-setting. Furthermore, conflicting evidence exists regarding end-of-life long-term care expenditures and their functional relationships with age and TTD (de Meijer et al., 2011; Karlsson and Klohn, 2014), and the UK's institutional structure and its resultant incentives regarding (predominantly privately-financed) long-term care and (predominantly socially-financed) hospital care is such that different relationships for each that are particular to the UK may well be expected. Finally, hospital expenditure is a major contributor to end-of-life health care costs: French et al. (2017) find that hospital expenditure is dominant in the final year of life with non-hospital expenditure, of which long-term care is a major component, playing a greater role in periods prior to this. Indeed, they estimate that for England 11.6% of hospital expenditure occurs in the last year of life.

This paper seeks to bridge the gap between the red herring strand of literature and models of resource allocation, treating morbidity measures as omitted variables in models of current health care expenditure, and examining what the relationship between age, TTD and HCE is once morbidity is included in these models (see, for instance, Aragon et al. (2016)).

3 Data

3.1 Data sources

Information on patient-level hospital use and associated reference costs for treatment are derived from the Hospital Episodes Statistics (HES) dataset, published by the Health and Social Care Information Centre (HSCIC). This is complemented with small-area data on years of potential

life lost (YPLL) published by the ONS, and individual level mortality information, jointly published by the HSCIC and the ONS.

We use successive years (financial years 2005/06 to 2011/12) of the HES dataset, which has been published for each financial year[4] since 1989/90 and is available for admitted patient care, outpatient, accident and emergency and maternity cases. The admitted patient (commonly, 'inpatient') care HES dataset that we use provides information on individual-level patient characteristics and diagnoses and procedures undergone for all patients admitted to hospitals in England.[5]

Information regarding inpatient spells is used to associate reference costs to each spell. Reference costs are based on each NHS provider's estimates of their own costs for each patient spell, categorised by Healthcare Resource Group (HRG, the NHS's system of grouping clinically-similar events with comparable resource use.). These reference costs are derived from accounting costs for each HRG, submitted by each organisation providing secondary care in England (Department of Health, 2012). The NHS Costing Manual provides guidance to all providers to support the calculation of reference costs and to enforce more uniform standards for costing methodologies. We use the estimate provided by the hospital providing treatment as our estimated cost for the patient's episode. The DH's Reference Cost data is submitted on a full absorption basis – that is, taking account of all direct and indirect costs relating to the activities in question, as well as a proportion of an estimate of all overhead costs relating to the overall running of the provider. Further, to account for the fact that costs will vary even within HRGs, hospitals are required to provide *per diem* costs for longer admissions that exceed a given 'trim point', which differs by each HRG. This trim point is defined as the upper quartile of length of stay, plus 1.5 times the inter-quartile range for length of stay for that HRG (Department of Health, 2012). Moreover, we augment the standard costs incurred in each episode with the 'unbundled' costs where recorded for the episode. This represents one or more extra fixed costs associated with the episode where additional, unusual, high-cost treatment or procedures were involved. Even within the same primary HRG, costs are not identical but differ according to the patient's length of stay. An estimate of costs for each inpatient spell is obtained by matching data on costs for that provider in the Reference Costs database to HRG

[4]In the UK, the financial year runs from April to March.

[5]This dataset includes both daycases (patients without an overnight stay) as well as patients who have at least one night's stay in hospital. Our use of 'inpatient' throughout this text includes both types of patient.

for each episode in the relevant year's HES data.

HES contains diagnostic data, categorised (since 1995/96) according to the tenth revision of the World Health Organization's International Classification of Diseases (ICD-10). Details of procedures and interventions are recorded according to the fourth revision of the Office of Population, Censuses and Surveys' Classification of Intervention and Procedures (OPCS-4) (Health & Social Care Information Centre, 2013).

HES is broken down by completed "episode" – each record consists of a continuous period of care at a single provider of treatment under the same consultant. A new record is generated when a patient is either transferred to the care of either a new consultant, transferred to a new provider, or is discharged from hospital. Although individuals are not identifiable, individuals can be tracked across episodes by an anonymised identification number. The costing of a patient's time in hospital and the recording of their diagnoses and procedures undergone are made at the episode level.

Patients can be tracked across different years of the HES dataset, which enables the creation of a panel structure for the data. Information within the HES dataset – most commonly, information regarding diagnosis, treatment and age of the patient – is used to apply the most appropriate Healthcare Resource Group (HRG) categorisation to the dataset. We use the Health and Social Care Information Centre's Consultation 'Grouper' software in order to carry out this first step. We use the most recent version of this Grouper – for the 2011/12 financial year – for all seven of the years we use, to categorise patients into HRGs. HRGs are used to categorise patient spells not only by broad diagnosis, but by the type and complexity of the patient's spell, into one of over 1,400 groupings. This allows us to apply the current best-practice methods for grouping patients into HRGs based on the information available. We apply available estimates of hospital costs for each inpatient spell, using reference costs data for the relevant financial year.

We add information regarding an individual's death from linked HES-ONS mortality data. The latest version of this data provides information on deaths to the end of the 2012 calendar year, and therefore provides information on some individuals whose deaths are known to have occurred after the end of the final wave in our dataset. Where individuals are known to have died, they are included up to and including the final quarter of their life, and not included

in the panel in following years. TTD can only be measured – for decedents – retrospectively, using information available at the time of the individual's death. We observe individuals for a maximum of seven years (from 2005/06 to 2011/12) or 28 quarters and code TTD from 1 to 28, with $TTD = 1$ denoting the final quarter in which death occurs.[6]

We adopt a strategy that employs two complementary sampling procedures, each incorporating approximately 40,000 individuals. The first draws a sample of individuals who died in 2011/12, the final year of our analysis, and who had at least one quarter of recorded positive HCE in the 28 quarters of our data. The second draws a sample of individuals who had at least one quarter of recorded positive HCE in 2005/06, and died in or before 2011/12. We believe that each of these sampling procedures has advantages and disadvantages but that, together, they can be used to establish a clear conclusion on our research question.

Our first sample for analysis consists of a random sample of 39,381 individuals (18,690 men and 20,691 women) aged 50 years and older, taken from those with at least one inpatient episode between 2005/06 and 2011/12, and whose death was recorded by the ONS in the financial year 2011/12. Our second sample consists of a random sample of 39,796 individuals (19,673 men and 20,123 women) aged 50 years and older, taken from those with at least one inpatient episode in 2005/06, and whose death was recorded by the ONS after this point, and by the end of the financial year 2011/12. Sample size was selected to enable computations not to become burdensome, and the age cut-off was selected to ensure sufficient deaths were observed in the data to make meaningful inference. We follow all sampled individuals across all quarters until their death to observe their subsequent inpatient health care use and associated morbidity characteristics.

We collapse all inpatient episodes for each individual from HES for a given quarter into a single observation in our data. This observation contains the sum of all hospital costs incurred in all episodes finishing in that quarter, as well as diagnostic information contained in the ICD-10 codes for those episodes in that quarter. In principle, the ICD-10 classification allows for up to 14,400 different diagnoses. To make these more manageable for analysis, however, we collapse this information using the US Agency for Healthcare Research and Quality's Clinical Classifications Software (CCS) method to convert ICD-10 codes to CCS codes (US Agency for Healthcare Research and Quality, 2009). This reduces the number of different groupings

[6]Coding TTD in this way is akin to assuming all deaths occur at the end of a quarter.

to a more manageable 260 mutually-exclusive, and clinically meaningful, categories[7]. Where individuals do not have any episodes in a given quarter, we separately adopt two distinct methods in order to deal with such cases. In one approach, they are recorded as having zero hospital costs, and as having zero observed morbidities arising from diagnostic information. In the absence of additional information on the gravity of any residual health problem, this assumes that such health issues are insignificant relative to those leading to a hospitalisation. In a second approach, we recognise that the recording of zero morbidities might be unrealistic for patients observed to have hospitalisations in recent periods and for whom there is likely to exist an underlying, albeit less grave, health problem. Consequently, we model these cases in our second approach under the assumption that episodes for which no information is available represent non-informative, missing data.

While we include a sum of all hospital costs for episodes ending in the quarter in question, we include only a maximum of three diagnoses for each individual, for a maximum of five episodes ending in that quarter. Using the merged mortality data, we are able to add a variable for the individual's time-to-death, measured in number of quarters to death.

In addition, we make use of the Office for National Statistics' Indices of Multiple Deprivation (IMD), by Lower Super Output Area (LSOA) in order to construct an instrument for TTD. LSOAs are defined at the time of the UK's decennial Census and are made up of similarly-sized small areas of the country. HES data, for the years used in our dataset, provides information on the individual's LSOA of residence at the time of the 2001 Census. At this time, LSOAs in England consisted of 32,482 areas of populations between 1,000 and 3,000, with between 400 and 1,200 households (Office for National Statistics, 2011).

Indices of Multiple Deprivation, at this LSOA level, are measures of the levels of deprivation in those small areas. Although made up of seven domains (income, employment, health and disability, education, housing, living environment and crime (Department for Communities and Local Government, 2011)), we primarily make use of one of the indicators that forms part of the health and disability IMD score: years of potential life lost per 1000 people. This consists of a standardised measure of premature mortality calculated using information for all individuals

[7] A full list of these CCS groupings is provided in Appendix A

to have died before the age of 75, as described in Blane and Drever (1998)[89]. Although the LSOAs themselves are defined every ten years at the time of the UK's census, statistics for each domain are collected and published for these areas more regularly: we make use of those published in 2007 (produced using data from 2001-2005 inclusive), and 2010 (produced using data from 2004-2008 inclusive) (Department for Communities and Local Government, 2008, 2011). For each of these years, we use LSOAs as defined in the 2001 UK Census. While these figures are comparable within years, the data collector (the UK's Department for Communities and Local Government) caution against using this data for trend analysis. These measures are highly correlated with TTD and, by virtue of being calculated at an aggregate level, exogenous in a model of HCE. That is, while the level of YPLL at an LSOA level is a strong predictor of an individual's TTD, this YPLL level is not influenced by the HCE for a given individual. We therefore include at least one wave of this measure separately as instruments.

Tables 1 to 2 present descriptive statistics for the sample of decedents from the first wave of data, under our strategy of sampling from the first year of observations (2005/06). Tables 3 to 4 present descriptive statistics from the first wave of data, under our strategy of sampling from the final year of observations (2011/12).

Table 1: Summary statistics (Quarter 1, men, first year sample.)

Variable	Mean	Std. Dev.	Min	Max
HCE [missing treated as zero]	475.60	1740.26	0	82901.09
log(HCE) [missing treated as zero]	1.57	3.00	0	11.32
log(HCE) [missing treated as missing]	7.19	1.01	3.42	11.32
Quarters to death (QTD)	9.53	7.77	0	27
log(QTD)	2.02	0.88	0	3.33
Age	75.03	10.24	50	105.66
YPLL (IMD 2007)	65.50	15.71	33.80	180.8

[8]The Office for National Statistics, however, use 75 rather than 65 years, in their implementation of this method, as the age at which mortality is considered to be premature (Department for Communities and Local Government, 2011).

[9]Details of the method employed by the ONS were obtained in personal communication with the study's author, Chris Dibben.

Table 2: Summary statistics (Quarter 1, women, first year sample.)

Variable	Mean	Std. Dev.	Min	Max
HCE [missing treated as zero]	504.42	1629.34	0	45095.81
log(HCE) [missing treated as zero]	1.56	3.03	0	10.71
log(HCE) [missing treated as missing]	7.30	0.99	3.39	10.71
Quarters to death (QTD)	9.86	7.89	0	27
log(QTD)	2.05	0.89	0	3.33
Age	78.11	10.93	50	111.15
YPLL (IMD 2007)	65.85	15.54	33.30	191.5

Table 3: Summary statistics (Quarter 1, men, final year sample.)

Variable	Mean	Std. Dev.	Min	Max
HCE [missing treated as zero]	220.74	1339.96	0	66770.92
log(HCE) [missing treated as zero]	0.61	2.05	0	11.11
log(HCE) [missing treated as missing]	7.28	1.08	3.85	11.11
Quarters to death (QTD)	25.57	1.13	24.00	27.00
log(QTD)	3.28	0.04	3.22	3.33
Age	72.93	9.82	50	100.83
YPLL (IMD 2007)	64.14	15.09	33.80	162.90

Table 4: Summary statistics (Quarter 1, women, final year sample.)

Variable	Mean	Std. Dev.	Min	Max
HCE [missing treated as zero]	213.89	1310.72	0	64392.08
log(HCE) [missing treated as zero]	0.56	1.98	0	11.07
log(HCE) [missing treated as missing]	7.36	1.08	3.34	11.07
Quarters to death (QTD)	25.59	1.13	24.00	27.00
log(QTD)	3.28	0.04	3.22	3.33
Age	76.80	10.02	50	105.58
YPLL (IMD 2007)	64.78	15.07	33.80	180.80

As is usual, the distribution of HCE is positively skewed, with this skewness reduced somewhat when we take a logarithmic transformation.[10] As would be expected due to their longer lifespan, on average, the average age of women in the sample is somewhat higher than that for men. Similarly, women are observed for, on average, slightly more waves. HCE, with missing waves treated as zero-(log)-cost observations, is on average higher when sampling from the first financial year of data than when sampling from those who died in the final year of analysis. This is as expected: the former is drawn from those with an inpatient episode in 2005/06, whereas the latter is drawn from those with an inpatient episode in any of the seven financial years of analysis. Indeed, HCE is approximately similar when missing waves are treated as missing observations.

Diagrams, presented in Figures 3 and 4, based on descriptive statistics from a sample of 9,957,084 individual quarterly observations in HES, provide some illustration of the existing red herring thesis. HCE appear to increase with age (top-left panel): this is the usual age-expenditure curve that is used to infer rising costs with population ageing, with the assumption being that as the population ages, the curve continues to rise as an extrapolation of the observed trend[11]. The observation that expenditures rise with age, however, is an artefact of a compositional effect. The naïve age-expenditure curve is composed of individuals who are known to have died during the period of observation (the sample used in estimation) – who have, on average, high expenditures for this period (top-right panel) – and individuals who are known to have survived to at least the end of the period of observation who have, on average, lower

[10] Due to log(0) being undefined, we add a value of one to such observations in our modelling strategies that include zero-cost quarters.

[11] We set aside here the drop in expenditures at very high ages, as this is likely to be due to the substantially lower sample sizes observed here.

expenditures for this period (bottom-left panel)[12]. The average expenditures for individuals observed to have died during the sample period are far greater than for individuals who survive. This suggests an important role for time-to-death in explaining HCE. As the proportion of the full population who are decedents increases with age, the näive observed relationship between age and expenditure displays an increasing trend. Note, however, that average expenditures for both decedents and survivors display a flatter profile than that depicted for the full population suggesting a less important role for age. Indeed, expenditure on decedents generally decrease, with this decrease particularly pronounced for women. Expenditure on survivors generally increase, but with a shallower gradient than observed for the full population, and at a lower average cost.

When we focus on decedents, and consider average HCE by proximity to death, we observe a large increase in costs in terminal quarters – particularly in the year immediately before death. Figure 7 in the Appendix shows a similar relationship between expenditures and TTD for men at selected ages. In general, expenditure in quarters preceding the final three average around £500 (although there is variation). In the final three quarters, and particularly the final quarter, we observed a large increase in expenditure. With the exception of 50 year olds, there is a clear gradient of health expenditures rising most dramatically in the final quarter of life with average increases over the penultimate quarter ranging from £460 for 55 year olds to £1,099 for 90 year olds.

[12]While some of these survivors will be closer to death than other and therefore would be classed as decedents over a longer observation period, such an effect would bias us against finding a visual difference in these graphs. We therefore consider this to be strong evidence of a different age profile of HCE for decedents and survivors.

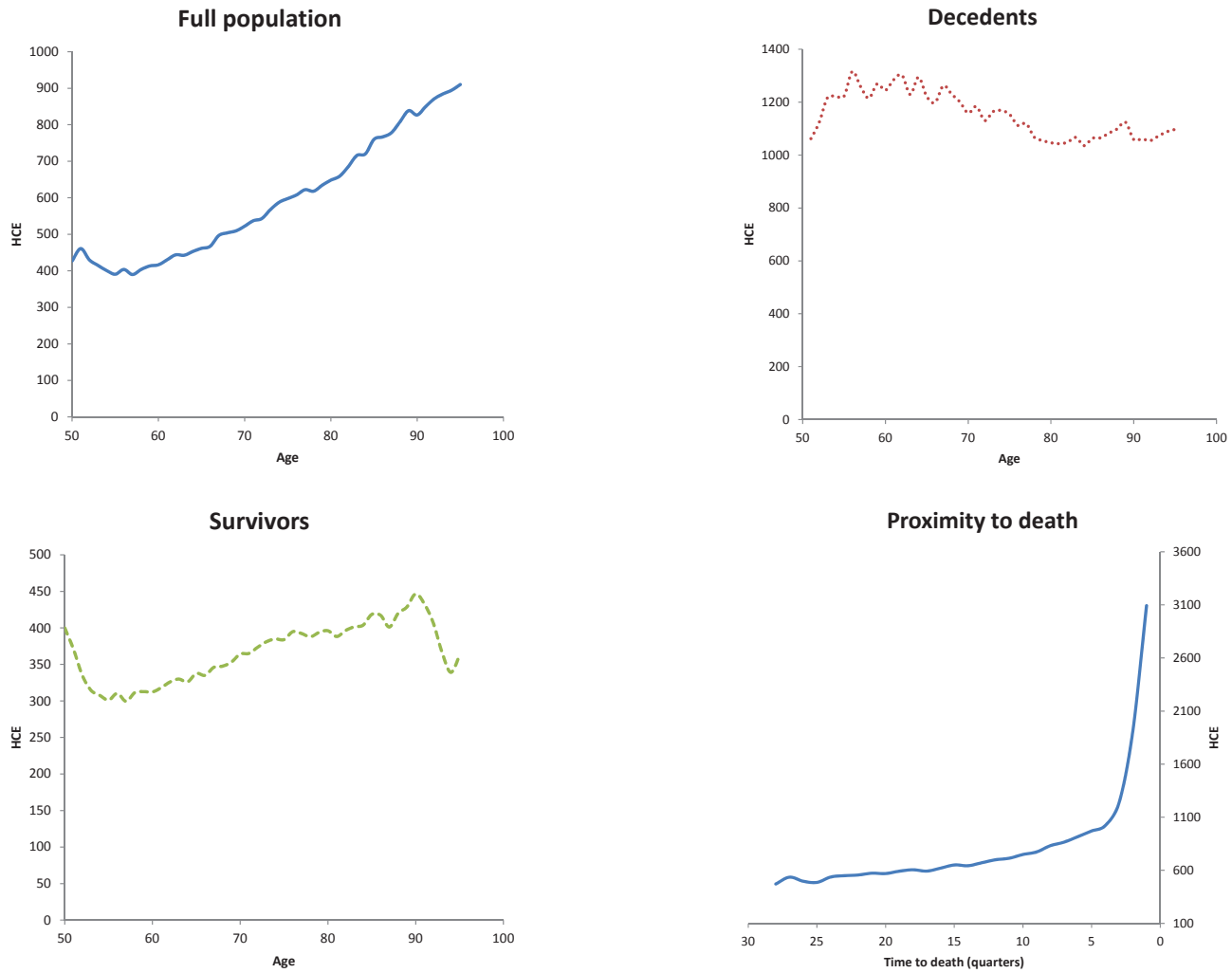

Figure 3: Healthcare expenditures by age and proximity to death, males

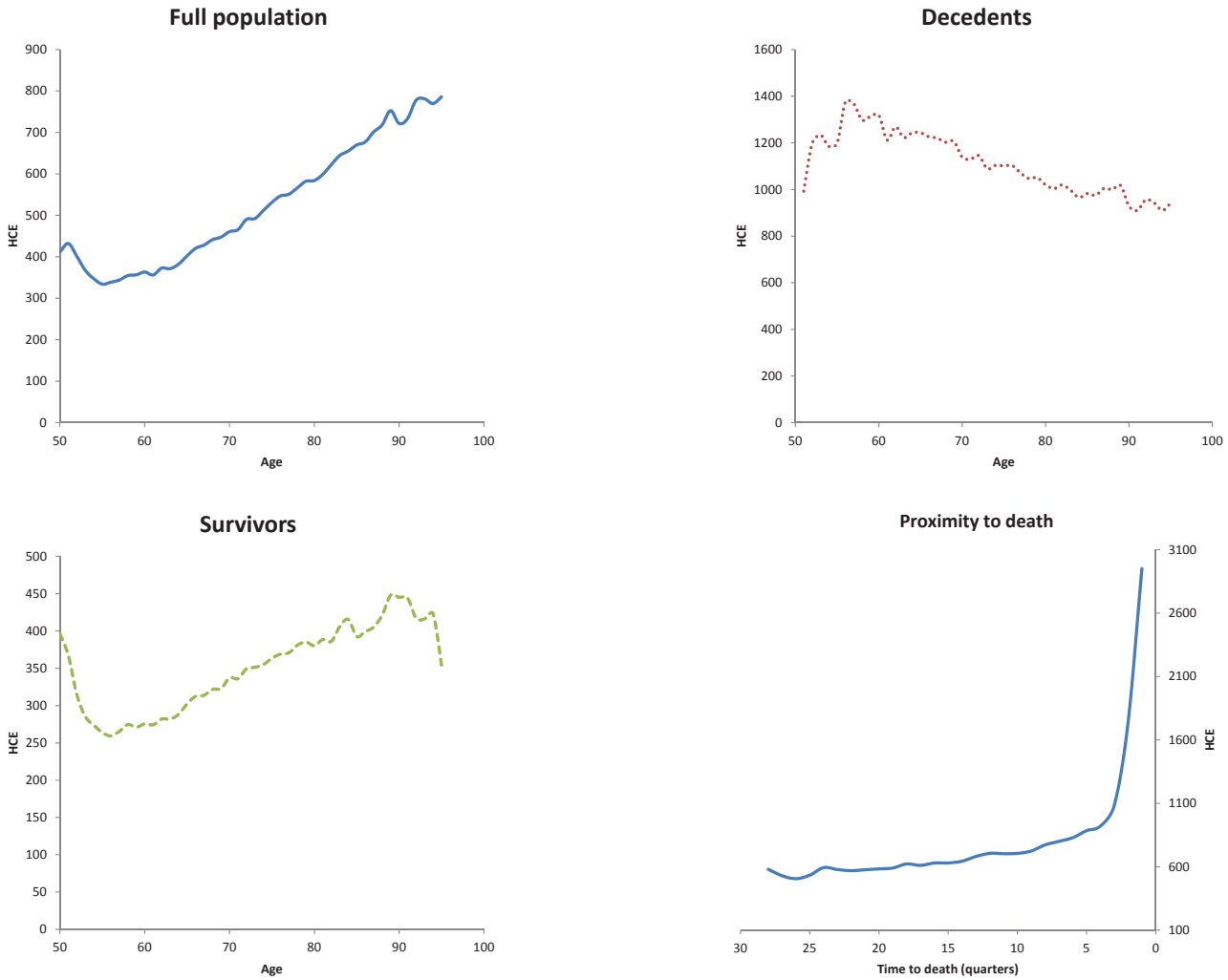

Figure 4: Healthcare expenditures by age and proximity to death, females

The relationship between HCE and TTD in levels is nonlinear. Figure 5 shows that the relationship is approximately linear on the logarithmic scale and in the modelling that follows logarithms of both HCE and TTD are used throughout.

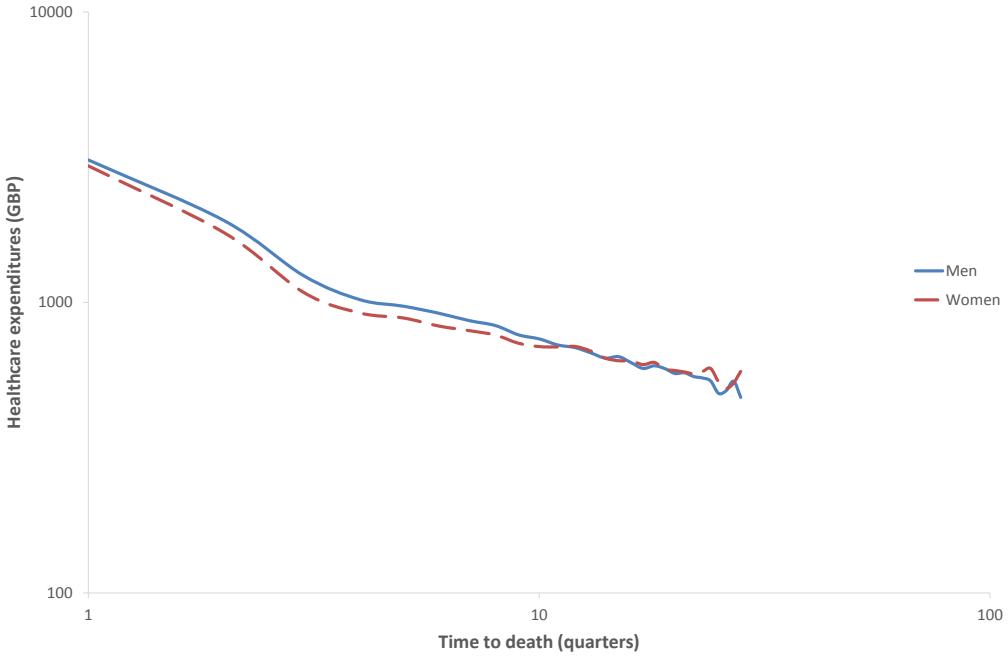

Figure 5: Average health care expenditures according to quarters to death (log scale for x- and y- axes)

4 Econometric model

We follow the general strand of the red herring literature and specify a baseline model of HCE, including only age as an explanatory variable.

$$log\left(HCE_{it}\right) = \alpha + \beta_{age} age_{it} + \tau_{it} + \mu_i + \varepsilon_{it}, \qquad i = 1, ..., N, t = 1, ..., T_i, \quad (1)$$

where τ_{it} is a vector of control variables (year and season of admission, and hospital provider dummies) μ_i is an individual-specific unobserved effect and ε_{it} is an idiosyncratic error term. Although this model is not estimated in existing papers, it is claimed that such a model would not adequately explain HCE. TTD is claimed to be an omitted variable in these models, giving

rise to models such as:

$$log\left(HCE_{it}\right) = \alpha + \beta_{age} age_{it} + \beta_{TTD} log\left(TTD_{it}\right) + \tau_{it} + \mu_i + \varepsilon_{it}. \quad (2)$$

We argue that individual morbidity is an omitted variable in this type of model, where TTD functions as a proxy for such morbidity[13]. Accordingly, we augment the model as follows:

$$log\left(HCE_{it}\right) = \alpha + \beta_{age} age_{it} + \beta_{TTD} log\left(TTD_{it}\right) + \sum_{j=1}^{260} \beta_{CCS_j} CCS_{jit} + \tau_{it} + \mu_i + \varepsilon_{it}, \quad (3)$$

where CCS_n represents a recorded morbidity of CCS type n (n = 1...260). We exploit the available data in HES to include detailed information about a patient's morbidities at the time of their hospital stay. We estimate each of these models with random effects, representing unobserved heterogeneity.

Modelling HCE as a function of TTD suffers from potential problems of endogeneity. Existing literature suggests that conditional on other covariates, being further from death – i.e. having a high TTD – in time period t is likely to lead to lower levels of HCE in t. Higher levels of HCE_{it}, however, are likely to lead to high levels of TTD_{it}: if the hospital activity that generates health care expenditures is effective in improving health then the individual is likely to enjoy a longer remaining lifespan as a result. We therefore posit that actual TTD at time period t has been determined in part by HCE in that time period as well as other time periods. Consequently, if endogeneity does pose problems in this analysis, the coefficient estimate on TTD (when treated as exogenous) is likely to be an underestimate of the true 'effect' of TTD.

Other models in the red herring strand of literature model HCE, using TTD and age as explanatory variables, but highlighting this endogeneity problem. Various attempts are made to purge TTD of its endogeneity in HCE (Zweifel et al., 2004; Werblow et al., 2007; Felder et al., 2010). We propose the use of a component of the Health and Disability Index of Multiple Deprivation by Lower Super Output Area – years of potential life lost (YPLL) – as an instrument for TTD under the assumption that such measures are exogenous in a model of HCE but highly

[13] And, furthermore, that such a proxy relationship may change over time in the presence of a compression of morbidity.

correlated with TTD. That is, while the level of YPLL at an LSOA level is a strong predictor of an individual's TTD, this YPLL level is not influenced by the HCE for a given individual. Accordingly, where possible, we reestimate models (2) and (3) instrumenting TTD by YPLL[14]. Such an instrumented approach is, however, possible only in the case of our second sampling procedure, where TTD is not pre-determined by the construction of the sample. In our former sampling procedure, all individuals die in the final four quarters (i.e., final financial year) of the sample, and thus any relevance of variation across areas in deprivation would not be expected.

5 Results

All versions of our different sampling and modelling strategies lead to qualitatively similar results. In short, a weak (and often statistically insignificant) relationship is observed when costs are modelled as a function of age alone. Confirming the overall red herring results, a strongly significant relationship is observed between TTD and HCE, when TTD is added as an explanatory variable. This is in line with our descriptive diagrams (Figures 3 & 4), demonstrating that the naïvely-estimated relationship between age and HCE is muted when conditioning on TTD. When morbidities are included as explanatory variables, the relationship between TTD and HCE is reduced (in all cases, the coefficient is reduced by approximately two-thirds). When, where possible, instrumenting TTD, the relationship between TTD and age becomes larger, with the addition of morbidities again reducing the size of the TTD cofficient.[15]

Table 5 presents the results of various specification of a random effects panel data model of log(HCE) on age, log(TTD) and morbidity characteristics for the sub-sample of decedents, when a sample is drawn from those who died in 2011/12. The first column of results (model 1) shows a weak and generally non-significant relationship between age and inpatient costs. These results represent, as far as we are aware, the first reported results in the red herring strand of literature of whether hospital costs increase with age in the aggregate, even before control

[14]While HCE is a function of morbidity, morbidity itself will be a function of age, and TTD is likely to be a function of morbidity. Indeed, our hypothesis is that TTD is a proxy for morbidity. Accordingly, we expect supplementing (3) with information on morbidity will temper the effect of both age (remaining after conditioning on TTD) and TTD on HCE.

[15]In the interests of consistency, all results presented here employ one wave of the YPLL instrument. Where both instruments appear as relevant at the first stage, we estimated the models using both YPLL waves in order to carry out a Hansen J test of the validity of overidentifying restrictions. In all cases, we observe large p-values consistent with failing to reject the null-hypothesis (between 0.3354 and 0.6317), suggesting evidence in favour of the exogeneity of our chosen instruments. Furthermore, our second stage results suggest very similar coefficients and confidence levels, such that none of our conclusions drawn below are affected.

is made for other factors such as TTD and morbidities. Existing research broadly states that this is the case, but refer merely to population-level descriptive statistics. In a random effects model (2) including TTD and age, we observe a highly significant relationship with TTD. This result is in line with those in the red herring strand of existing research. As an individual gets 1% closer to death, HCE increases by between 0.34% and 0.42% for men (between 0.28% and 0.34% for women), depending on the modelling strategy adopted[16].

[16]Because we aggregate costs by quarter and consequently use discrete values of TTD for each individual in each wave, this elasticity can only be considered as an approximation.

Table 5: Results, final wave sampling.

	Missing observations treated as missing		
Model	(1)	(2)	(3)
	AGE_ONLY	AGE_TTD	AGE_TTD_MORBS
Men			
Age	-.01459**	-.01274*	-.00518
	(.00654)	(.00652)	(.00526)
Age^2	.00010**	.00009**	.00003
	(.00004)	(.00004)	(.00003)
log(TTD)		-.42375***	-.14454***
		(.01467)	(.01206)
Morbidities			included
Women			
Age	-.00068	.00081	-.00038
	(.00588)	(.00585)	(.00474)
Age^2	.00004	.00003	.00001
	(.00004)	(.00004)	(.00003)
log(TTD)		-.34305***	-.13276***
		(.01458)	(.01218)
Morbidities			included
	Missing observations treated as zeros		
Model	(1)	(2)	(3)
Age	.00130	.00204	.00289*
	(.00180)	(.00181)	(.00156)
Age^2	0.00000	-.00001	-.00002**
	(.00001)	(.00001)	(.00001)
log(TTD)		-.33712***	-.10645***
		(.00679)	(.00560)
Morbidities			included
Women			
Age	.00983***	.01087***	.00559***
	(.00189)	(.00189)	(.00154)
Age^2	-.00005***	-.00006***	-.00003***

Continued on next page

	(.00001)	(.00001)	(.00000)
log(TTD)		-.27927***	-.09789***
		(.00604)	(.00520)
Morbidities			included
			** p<0.05 ** p<0.01 *** p<01*

Table 6: Results, first wave sampling.

	Missing observations treated as missing				
Model	(1)	(2)	(3)	(4)	(5)
Men					
	AGE_ONLY	AGE_TTD	AGE_TTD_MORBS	AGE_TTDIV	AGE_TTDIV_MORBS
Age	-.01800*	-.00454	.00290	-.00953	-.01124
	(.00932)	(.00896)	(.00746)	(.02617)	(.01087)
Age^2	.00013**	.00003	-.00002	.00007	.00007
	(.00006)	(0.00006)	(.00005)	(.00018)	(.00008)
log(TTD)		-.31565***	-.10098***	-.35626	-.13120
		(.00655)	(.00616)	(.36994)	(.30968)
Relevance F-statistic				16.38	13.24
Morbidities			included		included
Women					
Age	-.00269	.01198	.00065	.08939	-.01118
	(.00832)	(.00813)	(.00696)	(.06773)	(.01124)
Age^2	.00004	-.00005	-.00001	-.00059	.00007
	(.00005)	(.00005)	(.00004)	(.00047)	(.00009)
log(TTD)		-.26423***	-.09307***	-1.82773	-.12894
		(.00663)	(.00612)	(1.2711)	(.3239383)
Relevance F-statistic				66.95	11.67
Morbidities			included		included

Continued on next page

	Missing observations treated as zeros				
Model	(1)	(2)	(3)	(4)	(5)
Men					
Age	-.06500***	.10191***	-.00691	.28057***	.03547
	(.02465)	(.02265)	(.01462)	(.09001)	(.06062)
Age^2	.00049***	-.00088***	.00001	-.00363***	-.00036
	(.00016)	(.00015)	(.00010)	(.00113)	(.00058)
log(TTD)		-1.19842***	-.18669***	-2.05176***	-.31020
		(.01205)	(.00745)	(.38267)	(.23856)
Relevance F-statistic				80.44	71.30
Morbidities			included		included
Women					
Age	.06863***	.08155***	.02635***	.61590	.05830
	(.00928)	(.00934)	(.00806)	(.40329)	(.05937)
Age^2	-.00039***	-.00048***	-.00016***	-.00393	-.00038
	(.00928)	(.00006)	(.00005)	(.00261)	(.00040)
log(TTD)		-.15705***	-.03723***	-6.43066	-.64920
		(.00801)	(.00716)	(4.95179)	(.8581702)
Relevance F-statistic				13.09	11.81
Morbidities			included		included
					** p<0.05 ** p<0.01 *** p<01*

Conditioning on morbidity markers, we find a reduced role for TTD in explaining HCE, using both sampling strategies. Our estimate of the TTD elasticity of HCE falls by approximately two-thirds in almost all (non-IV) cases when we condition on the individual's observed morbidity in the current time period (i.e., when we move from model 2 to model 3). In all models, in excess of 90% of the estimated coefficients for the morbidity indicators are significant at the 1% level, yielding a p-value of 0.0000. We interpret this as indicating that TTD does indeed serve as a proxy for unobserved morbidity. The estimated coefficients for age when morbidity markers are included see similar falls. This is illustrated in Figure 6 which shows the difference in $log\,(HCE)$ from the quarter of death to preceding quarters for an individual who dies at age 75 for the alternative specifications of the model[17]. The combined relationship of time-to-death and age is severely muted when we condition on current morbidity markers as seen by the lines representing RE_AGE_TTD_MORBS and RE_AGE_TTD.

We anticipate hospital costs to rise as individuals approach death, and as such expect a negative relationship between TTD and HCE. For the sampling strategy where this is possible – sampling from the first calendar year – we instrument for TTD in order to deal with the potential endogeneity of TTD in HCE, which would mean that a naïve estimate of the 'effect' of TTD on HCE was likely to be biased towards zero (i.e. that naïve estimates would be expected to be less negative). In a further pair of models, we instrument TTD with LSOA-level YPLL measures, our small-area measure of premature mortality.

When we instrument using YPLL measures – model (4) – the estimated coefficient of log(TTD) rises (in absolute terms) in all cases. While we confirm the findings of Zweifel et al. (2004) that 'the proximity of death rather than age [being] a main determinant of HCE is fairly robust to endogeneity error,' our results also suggest that failing to account for the endogeneity of TTD in these models may lead to a large underestimate of the true 'effect' of TTD in models that do not include morbidity markers[18]. This is also illustrated in Figure 6, which shows the large divergence in estimated costs for these two models for an individual who dies at the age of 75. As expected, first-stage regressions show a negative and significant relationship between

[17]While our instrumented model including morbidity markers appears to show a mildly negative relationship between age and HCE, this arises from the use of a small and non-significant negative age coefficient in construction of this graph.

[18]While our point estimates rise, in most cases, however, the TTD coefficients are no longer significant when we carried out our instrumented regressions. We do not rely on these results in our conclusions regarding the relationship between TTD, morbidity, and HCE, and present these results to demonstrate its consistency with existing research.

YPLL and TTD and an F-test of these instruments suggests their relevance as a predictor of TTD according to the commonly used Stock-Yogo 'rule of thumb' of an F-statistic of 10 in all cases.

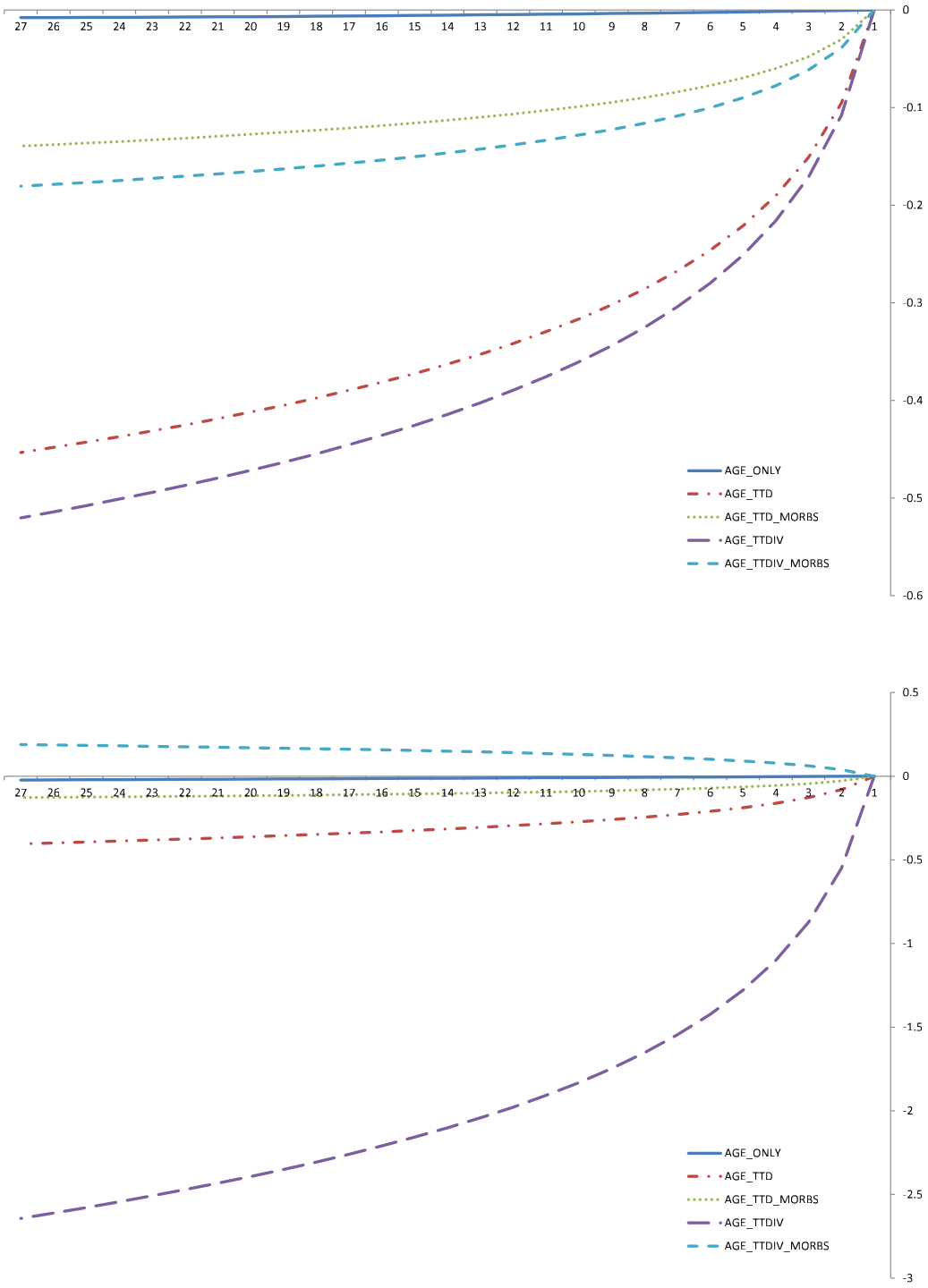

Figure 6: Change in HCE according to time-to-death and age, hypothetical individual dying at 75 (top – men, bottom – women; log HCE on y-axis)

6 Conclusions

Ageing populations pose a substantial problem for public service provision, particularly for health and social care. Estimates of how an ageing population will impact HCEs vary considerably. Developing credible predictions is a core component of health systems planning as is allocating resources efficiently and equitably to meet the health care needs of the population. Whilst it is undeniable that health care costs will rise as the baby-boomers age, the impact might not be quite as large as models based on a simple extrapolation of a crude age-expenditure curve suggests. As individuals live longer, all other things equal, they may generate larger cumulative life-time costs. The extent to which this becomes a burden on the health care sector will depend on how morbidity profiles of cohorts change over time. Should a compression of morbidity thesis hold, Fries (1980), Freedman et al. (2002) & Romeu Gordo (2011), on average individuals can expect to live longer and delay the onset of morbidity into later years. This will have the effect of moving the age-expenditure curve to the right as populations age. An expansion of morbidty would have more severe consequences for HCEs with individuals living longer, but also experiencing a greater number of years in ill-health.

Our findings support other literature that it is not age per se, but time-to-death (TTD), particularly the final year of life, that is a strong driver of HCEs. Our results regarding the relationship between age, TTD and HCE are in line with existing results in the 'red herring' strange of literature. We extend this existing literature to show that, in line with the economic implications of biological models of ageing as drawn out by Dalgaard and Strulik (2014), TTD in large part proxies for morbidity in explaining HCE. Our results – showing a weak relationship between HCE and age when TTD is included – fall in line with existing research into the determinants of HCE for ageing populations. However, while TTD clearly plays an important role in explaining HCEs, it is unhelpful in forecasting future expenditure needs. At an individual level TTD is unknown and hence to forecast future expenditure growth assumptions about the proportions of decedents and survivors together with projections of populations within age groups is required. By extending the modelling of HCE to include morbidity characteristics we show that the impact of TTD is diminished indicating that it acts as a proxy for underlying health status. This is important to allow the planning of future resource requirements and in developing appropriate models for budgets to be allocated equitably across providers of care

in response to population health care need. Our results are robust to problems of endogeneity that exist between HCE and TTD.

Our results strengthen the need to include measures of morbidity in models of HCE. Merely including TTD is insufficient in predicting future HCE. To accurately forecast future expenditure needs, information on changes to profiles of morbidity are required. The existence of a compression of morbidity, along with a tendency for increased life expectancy, suggests competing and opposing pressures on HCE. While increases in life expectancy suggests that a greater number of individuals will be alive at any given age, with associated upward pressure on HCE, a compression of morbidity will tend to, on average, provide downward pressure on HCE for any given individual at any given age.

This work has focused on determinants of the demand for inpatient health care services at an individual level via age, time-to-death and morbidity characteristics. Clearly there is also a substantial role for supply-side impacts on expenditure growth notably through technological advances in health care interventions and the way in which health care services are organized and delivered. We do not address these issues here, but are areas that warrant further investigation at an aggregate level. Inpatient hospital care is one of a number of services provided by the National Health Service in England and other expenditure should also be taken into account when assessing the overall impact of an ageing population, as should costs placed on the Government by long-term care services predominantly accessed by older age groups. The increasing ability to link administrative sources of data provides a potentially valuable resource for future research in this area.

A Appendix

Table A1: Clinical Classifications Software (CCS) groupings

CCS code	Description
1	Tuberculosis
2	Septicemia (except in labor)
3	Bacterial infection; unspecified site
4	Mycoses
5	HIV infection
6	Hepatitis
7	Viral infection
8	Other infections; including parasitic
9	Sexually transmitted infections (not HIV or hepatitis)
10	Immunizations and screening for infectious disease
11	Cancer of head and neck
12	Cancer of esophagus
13	Cancer of stomach
14	Cancer of colon
15	Cancer of rectum and anus
16	Cancer of liver and intrahepatic bile duct
17	Cancer of pancreas
18	Cancer of other GI organs; peritoneum
19	Cancer of bronchus; lung
20	Cancer; other respiratory and intrathoracic
21	Cancer of bone and connective tissue
22	Melanomas of skin
23	Other non-epithelial cancer of skin
24	Cancer of breast
25	Cancer of uterus
26	Cancer of cervix
27	Cancer of ovary
28	Cancer of other female genital organs
29	Cancer of prostate
30	Cancer of testis
31	Cancer of other male genital organs
32	Cancer of bladder
33	Cancer of kidney and renal pelvis
34	Cancer of other urinary organs
35	Cancer of brain and nervous system
36	Cancer of thyroid
37	Hodgkin‘s disease
38	Non-Hodgkin‘s lymphoma
39	Leukemias
40	Multiple myeloma
41	Cancer; other and unspecified primary
42	Secondary malignancies
43	Malignant neoplasm without specification of site
44	Neoplasms of unspecified nature or uncertain behavior
45	Maintenance chemotherapy; radiotherapy
46	Benign neoplasm of uterus
47	Other and unspecified benign neoplasm
48	Thyroid disorders

Continued on next page

49	Diabetes mellitus without complication
50	Diabetes mellitus with complications
51	Other endocrine disorders
52	Nutritional deficiencies
53	Disorders of lipid metabolism
54	Gout and other crystal arthropathies
55	Fluid and electrolyte disorders
56	Cystic fibrosis
57	Immunity disorders
58	Other nutritional; endocrine; and metabolic disorders
59	Deficiency and other anemia
60	Acute posthemorrhagic anemia
61	Sickle cell anemia
62	Coagulation and hemorrhagic disorders
63	Diseases of white blood cells
64	Other hematologic conditions
65	Mental retardation
66	Alcohol-related mental disorders
67	Substance-related mental disorders
68	Senility and organic mental disorders
69	Affective disorders
70	Schizophrenia and related disorders
71	Other psychoses
72	Anxiety; somatoform; dissociative; and personality disorders
73	Preadult disorders
74	Other mental conditions
75	Personal history of mental disorder; mental and behavioral problems; observation and screening for mental condition
76	Meningitis (except that caused by tuberculosis or sexually transmitted disease)
77	Encephalitis (except that caused by tuberculosis or sexually transmitted disease)
78	Other CNS infection and poliomyelitis
79	Parkinson‘s disease
80	Multiple sclerosis
81	Other hereditary and degenerative nervous system conditions
82	Paralysis
83	Epilepsy; convulsions
84	Headache; including migraine
85	Coma; stupor; and brain damage
86	Cataract
87	Retinal detachments; defects; vascular occlusion; and retinopathy
88	Glaucoma
89	Blindness and vision defects
90	Inflammation; infection of eye (except that caused by tuberculosis or sexually transmitteddisease)
91	Other eye disorders
92	Otitis media and related conditions
93	Conditions associated with dizziness or vertigo
94	Other ear and sense organ disorders
95	Other nervous system disorders
96	Heart valve disorders
97	Peri-; endo-; and myocarditis; cardiomyopathy (except that caused by tuberculosis or sexually transmitted disease)

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98	Essential hypertension
99	Hypertension with complications and secondary hypertension
100	Acute myocardial infarction
101	Coronary atherosclerosis and other heart disease
102	Nonspecific chest pain
103	Pulmonary heart disease
104	Other and ill-defined heart disease
105	Conduction disorders
106	Cardiac dysrhythmias
107	Cardiac arrest and ventricular fibrillation
108	Congestive heart failure; nonhypertensive
109	Acute cerebrovascular disease
110	Occlusion or stenosis of precerebral arteries
111	Other and ill-defined cerebrovascular disease
112	Transient cerebral ischemia
113	Late effects of cerebrovascular disease
114	Peripheral and visceral atherosclerosis
115	Aortic; peripheral; and visceral artery aneurysms
116	Aortic and peripheral arterial embolism or thrombosis
117	Other circulatory disease
118	Phlebitis; thrombophlebitis and thromboembolism
119	Varicose veins of lower extremity
120	Hemorrhoids
121	ther diseases of veins and lymphatics
122	Pneumonia (except that caused by tuberculosis or sexually transmitted disease)
123	Influenza
124	Acute and chronic tonsillitis
125	Acute bronchitis
126	Other upper respiratory infections
127	Chronic obstructive pulmonary disease and bronchiectasis
128	Asthma
129	Aspiration pneumonitis; food/vomitus
130	Pleurisy; pneumothorax; pulmonary collapse
131	Respiratory failure; insufficiency; arrest (adult)
132	Lung disease due to external agents
133	Other lower respiratory disease
134	Other upper respiratory disease
135	Intestinal infection
136	Disorders of teeth and jaw
137	Diseases of mouth; excluding dental
138	Esophageal disorders
139	Gastroduodenal ulcer (except hemorrhage)
140	Gastritis and duodenitis
141	Other disorders of stomach and duodenum
142	Appendicitis and other appendiceal conditions
143	Abdominal hernia
144	Regional enteritis and ulcerative colitis
145	Intestinal obstruction without hernia
146	Diverticulosis and diverticulitis
147	Anal and rectal conditions
148	Peritonitis and intestinal abscess
149	Biliary tract disease
150	Liver disease; alcohol-related

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151	Other liver diseases
152	Pancreatic disorders (not diabetes)
153	Gastrointestinal hemorrhage
154	Noninfectious gastroenteritis
155	Other gastrointestinal disorders
156	Nephritis; nephrosis; renal sclerosis
157	Acute and unspecified renal failure
158	Chronic renal failure
159	Urinary tract infections
160	Calculus of urinary tract
161	Other diseases of kidney and ureters
162	Other diseases of bladder and urethra
163	Genitourinary symptoms and ill-defined conditions
164	Hyperplasia of prostate
165	Inflammatory conditions of male genital organs
166	Other male genital disorders
167	Nonmalignant breast conditions
168	Inflammatory diseases of female pelvic organs
169	Endometriosis
170	Prolapse of female genital organs
171	Menstrual disorders
172	Ovarian cyst
173	Menopausal disorders
174	Female infertility
175	Other female genital disorders
176	Contraceptive and procreative management
177	Spontaneous abortion
178	Induced abortion
179	Postabortion complications
180	Ectopic pregnancy
181	Other complications of pregnancy
182	Hemorrhage during pregnancy; abruptio placenta; placenta previa
183	Hypertension complicating pregnancy; childbirth and the puerperium
184	Early or threatened labor
185	Prolonged pregnancy
186	Diabetes or abnormal glucose tolerance complicating pregnancy; childbirth; or the puerperium
187	Malposition; malpresentation
188	Fetopelvic disproportion; obstruction
189	Previous C-section
190	Fetal distress and abnormal forces of labor
191	Polyhydramnios and other problems of amniotic cavity
192	Umbilical cord complication
193	OB-related trauma to perineum and vulva
194	Forceps delivery
195	Other complications of birth; puerperium affecting management of mother
196	Normal pregnancy and/or delivery
197	Skin and subcutaneous tissue infections
198	Other inflammatory condition of skin
199	Chronic ulcer of skin
200	Other skin disorders

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201	Infective arthritis and osteomyelitis (except that caused by tuberculosis or sexually transmitted disease)
202	Rheumatoid arthritis and related disease
203	Osteoarthritis
204	Other non-traumatic joint disorders
205	Spondylosis; intervertebral disc disorders; other back problems
206	Osteoporosis
207	Pathological fracture
208	Acquired foot deformities
209	Other acquired deformities
210	Systemic lupus erythematosus and connective tissue disorders
211	Other connective tissue disease
212	Other bone disease and musculoskeletal deformities
213	Cardiac and circulatory congenital anomalies
214	Digestive congenital anomalies
215	Genitourinary congenital anomalies
216	Nervous system congenital anomalies
217	Other congenital anomalies
218	Liveborn
219	Short gestation; low birth weight; and fetal growth retardation
220	Intrauterine hypoxia and birth asphyxia
221	Respiratory distress syndrome
222	Hemolytic jaundice and perinatal jaundice
223	Birth trauma
224	Other perinatal conditions
225	Joint disorders and dislocations; trauma-related
226	Fracture of neck of femur (hip)
227	Spinal cord injury
228	Skull and face fractures
229	Fracture of upper limb
230	Fracture of lower limb
231	Other fractures
232	Sprains and strains
233	Intracranial injury
234	Crushing injury or internal injury
235	Open wounds of head; neck; and trunk
236	Open wounds of extremities
237	Complication of device; implant or graft
238	Complications of surgical procedures or medical care
239	Superficial injury; contusion
240	Burns
241	Poisoning by psychotropic agents
242	Poisoning by other medications and drugs
243	Poisoning by nonmedicinal substances
244	Other injuries and conditions due to external causes
245	Syncope
246	Fever of unknown origin
247	Lymphadenitis
248	Gangrene
249	Shock
250	Nausea and vomiting
251	Abdominal pain
252	Malaise and fatigue
253	Allergic reactions

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254	Rehabilitation care; fitting of prostheses; and adjustment of devices
255	Administrative/social admission
256	Medical examination/evaluation
257	Other aftercare
258	Other screening for suspected conditions (not mental disorders or infectious disease)
259	Residual codes; unclassified
260	E Codes: All (external causes of injury and poisoning)

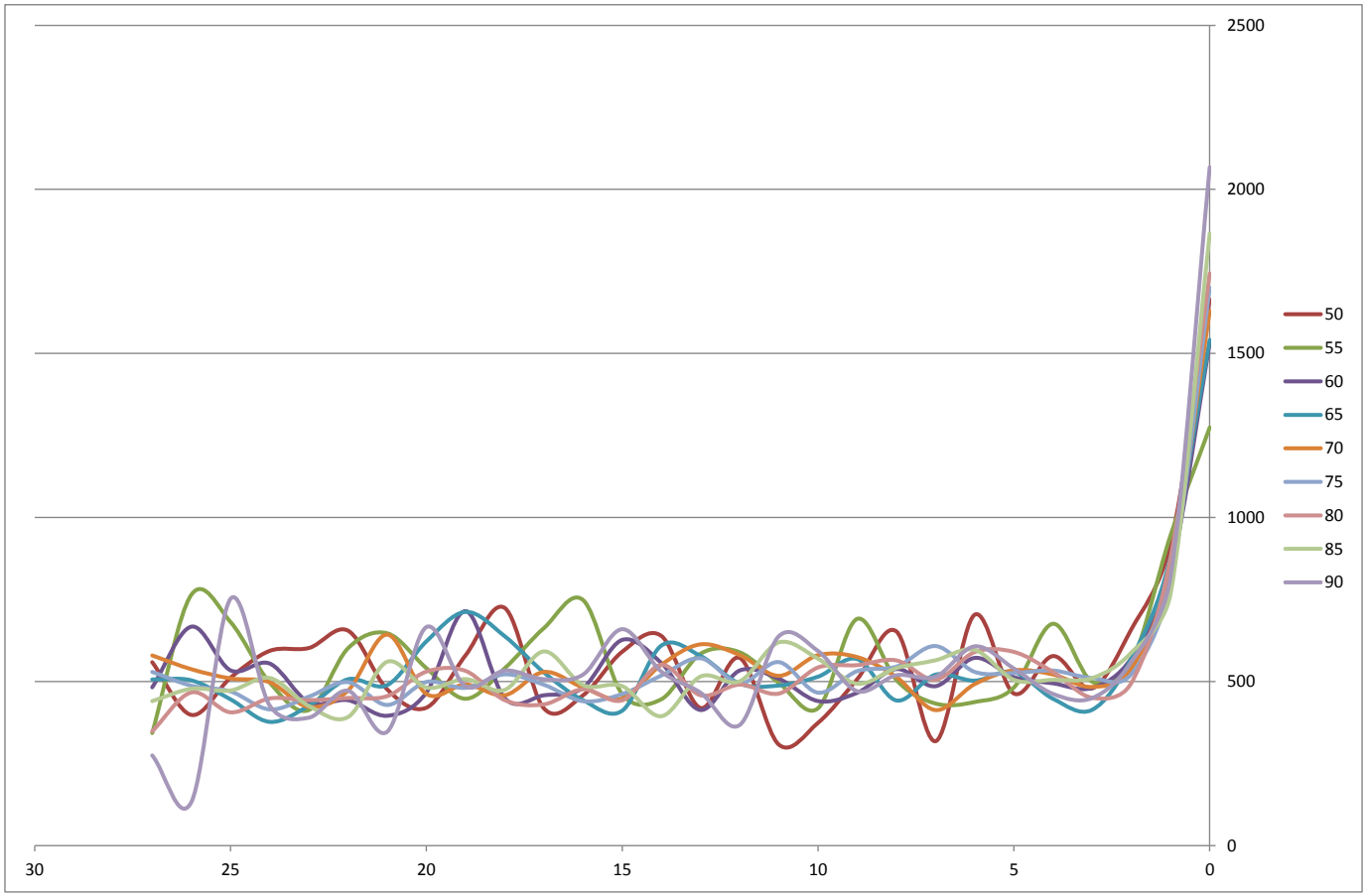

Figure 7: Healthcare expenditures by proximity to death, males by age

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