

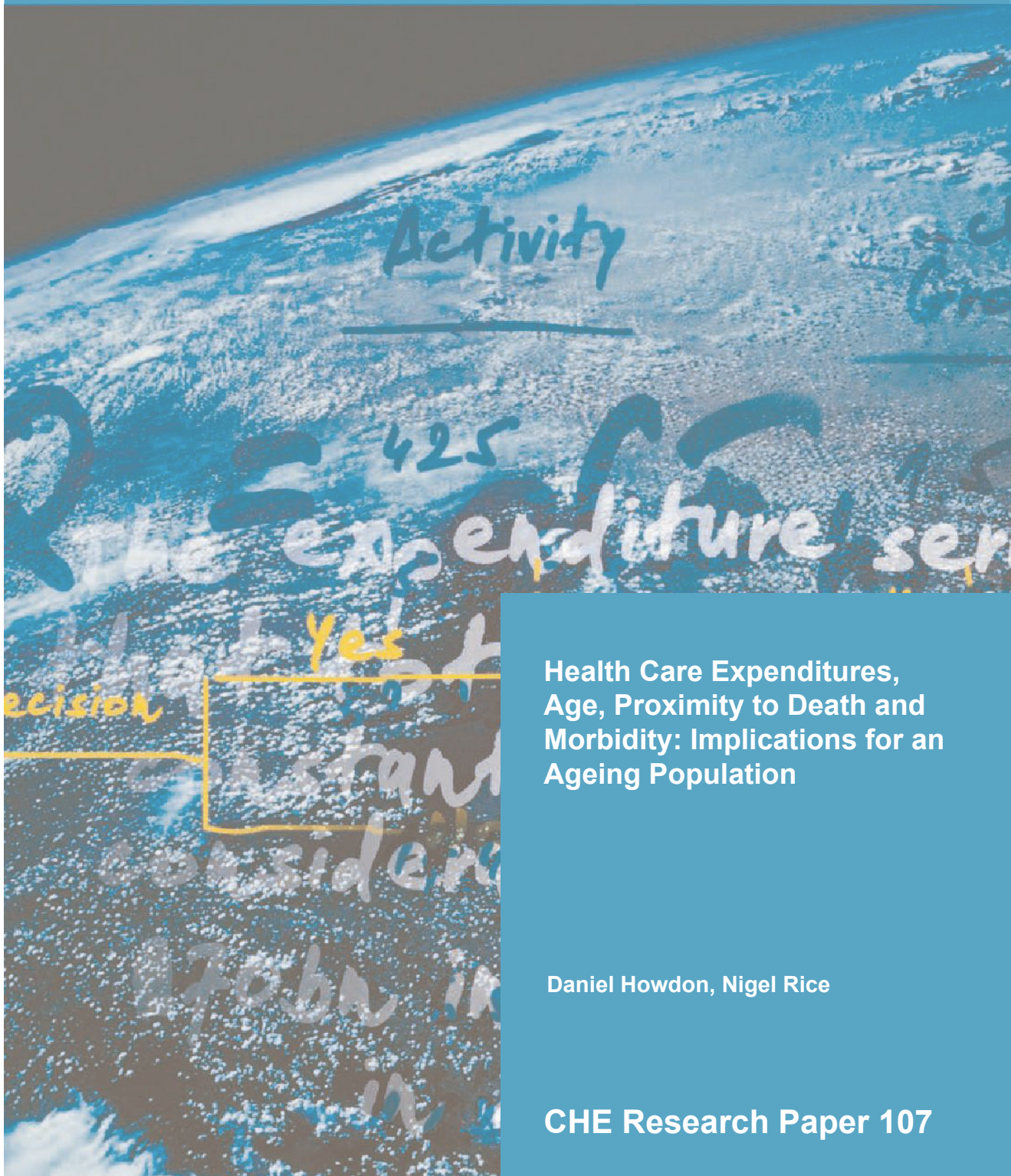
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**Health Care Expenditures,
Age, Proximity to Death and
Morbidity: Implications for an
Ageing Population**

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CHE Research Paper 107

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

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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.

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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 health 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 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 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 Dixon et al. (2011)).

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

¹ Emphasis ours.

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 healthily 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.

Evidence on compression of morbidity

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) observe 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 provided 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.

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 2²), 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 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.

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

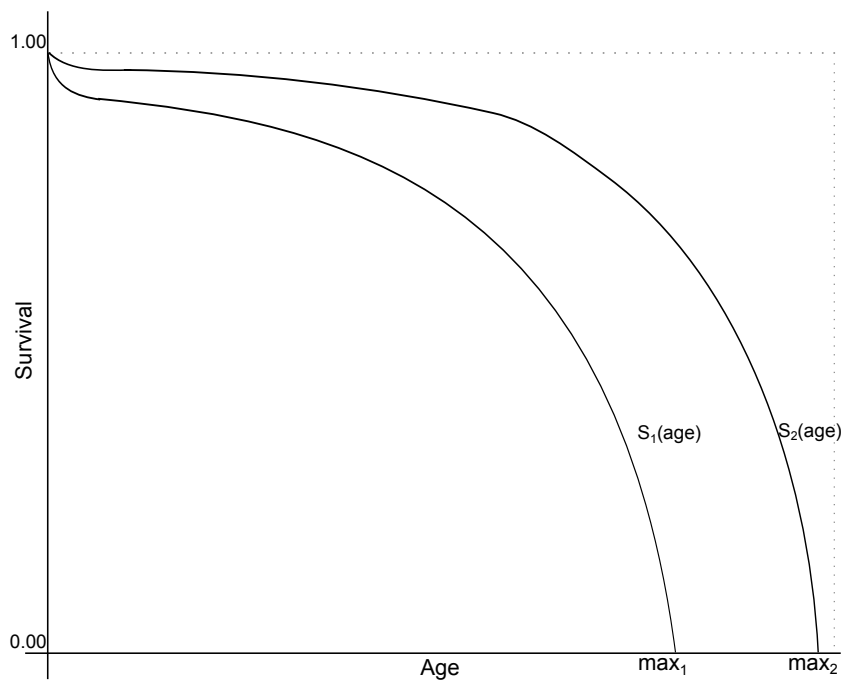


Figure 1: Stylised change in survival curves

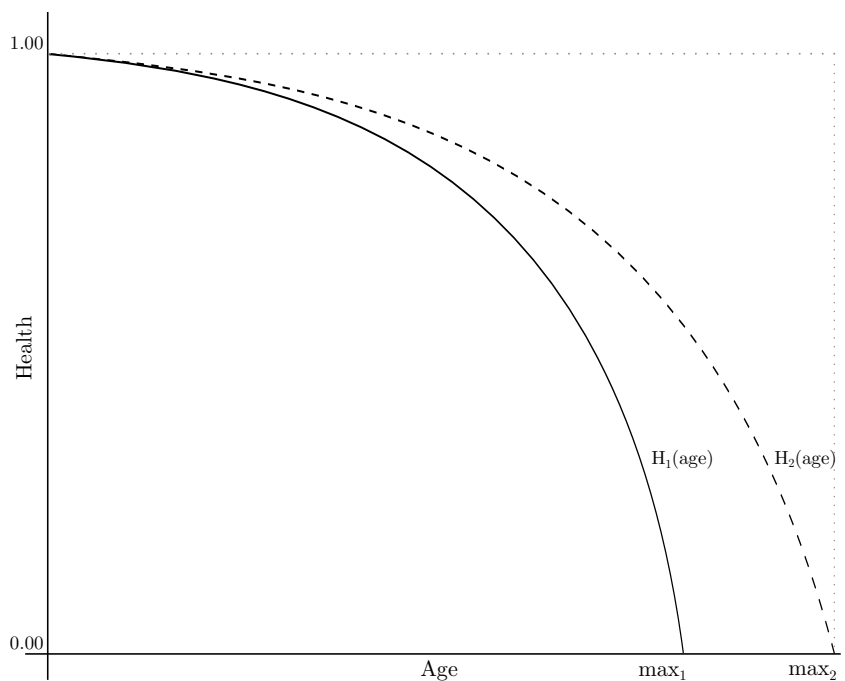


Figure 2: Stylised change in health profiles

Age and time-to-death

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, finds that no effect of age on health care expenditures existed after controlling for 'time-to-death' (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 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.

While use has been made of morbidity markers in models of long-term care expenditures (LTCE) (see 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. 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 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

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.

herring literature. Indeed, 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 and/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 allocation (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. 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)).

Data

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 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.⁴

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 HRG. 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

⁴ 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.

spell is obtained by matching data on costs for that provider in the Reference Costs database to HRG 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, the `hesid` variable allows individuals to be tracked across episodes, to create spells – multiple episodes unseparated by a temporal break outside of hospital. 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.⁵

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 2011/12⁶. 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 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 to a more manageable 260 mutually-exclusive, and clinically meaningful, categories⁷. Where individuals do not have any episodes in a 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

⁵ Coding TTD in this way is akin to assuming all deaths occur at the end of a quarter.

⁶ This falls to 11,809 men and 16,343 women after the exclusion of the first four quarters, inclusion upon which sampling is conditional.

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

the gravity of any residual health problem, this assume 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 (YPLL). This consists of a standardised measure of premature mortality calculated using information for all individuals 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; Department for Communities and Local Government 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,

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.

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 |

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-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, treating waves with no observations as missing, 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. The assumption being that as the population ages, ignoring the drop in expenditures at very high ages as this is likely due to low sample sizes, the curve continues to rise as an extrapolation of the observed trend. The observation that expenditures rise with age, however, is an artifact 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 who have, on average, lower expenditures for this period (bottom-left panel). 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 naïve observed relationship between age and expenditure displays an increasing trend. Note, however, that average expenditures for both decedents

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.

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.

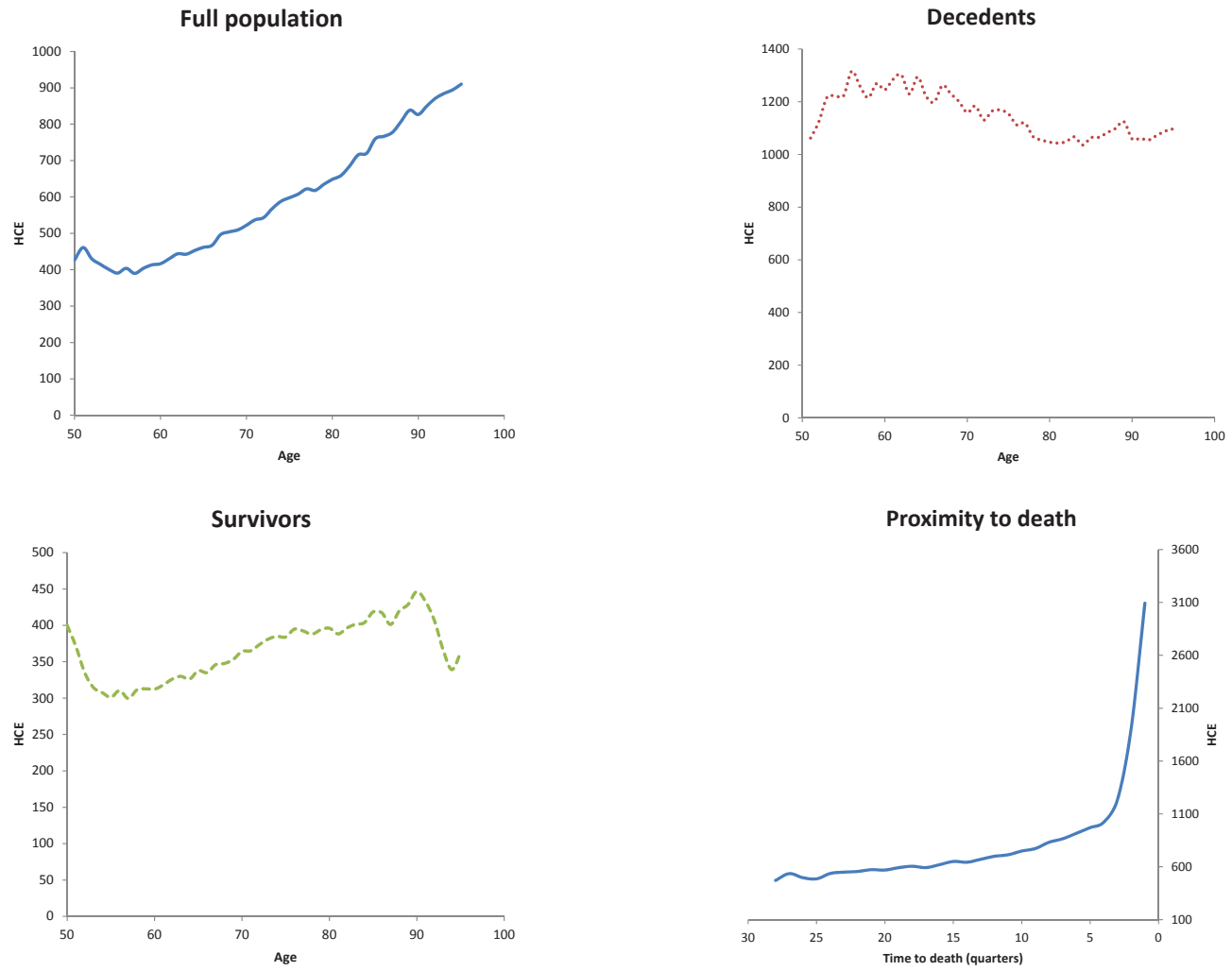


Figure 3: Healthcare expenditures by age and proximity to death, males

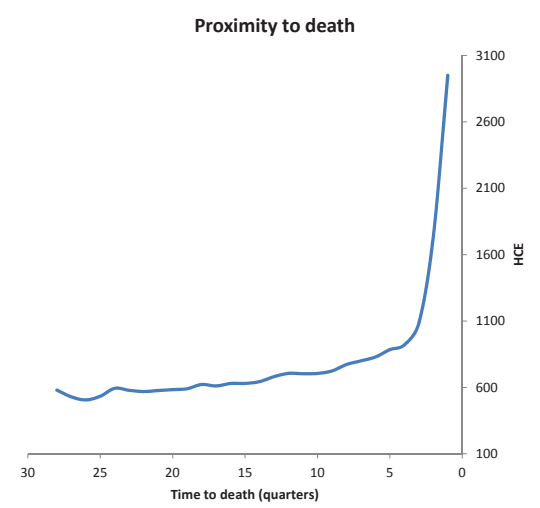
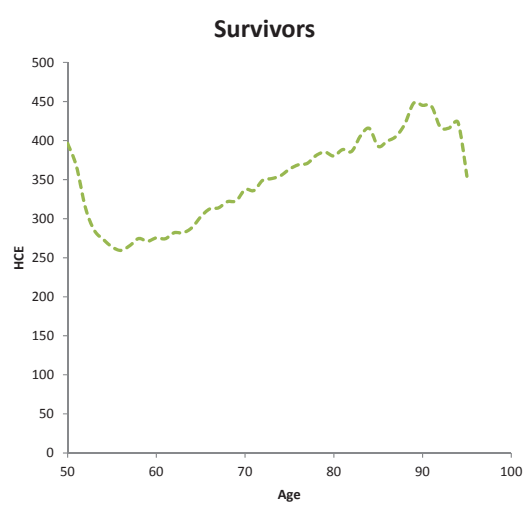
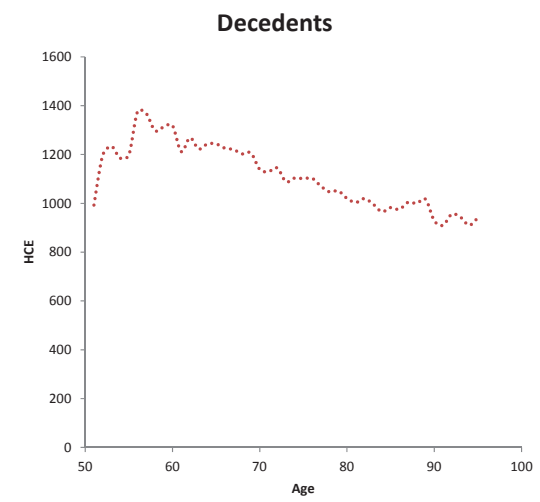
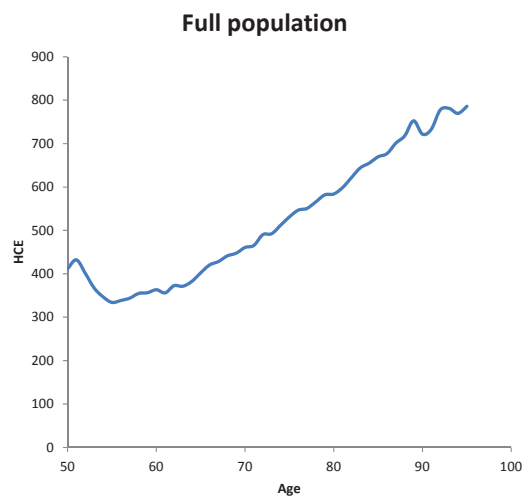


Figure 4: Healthcare expenditures by age and proximity to death, females

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.

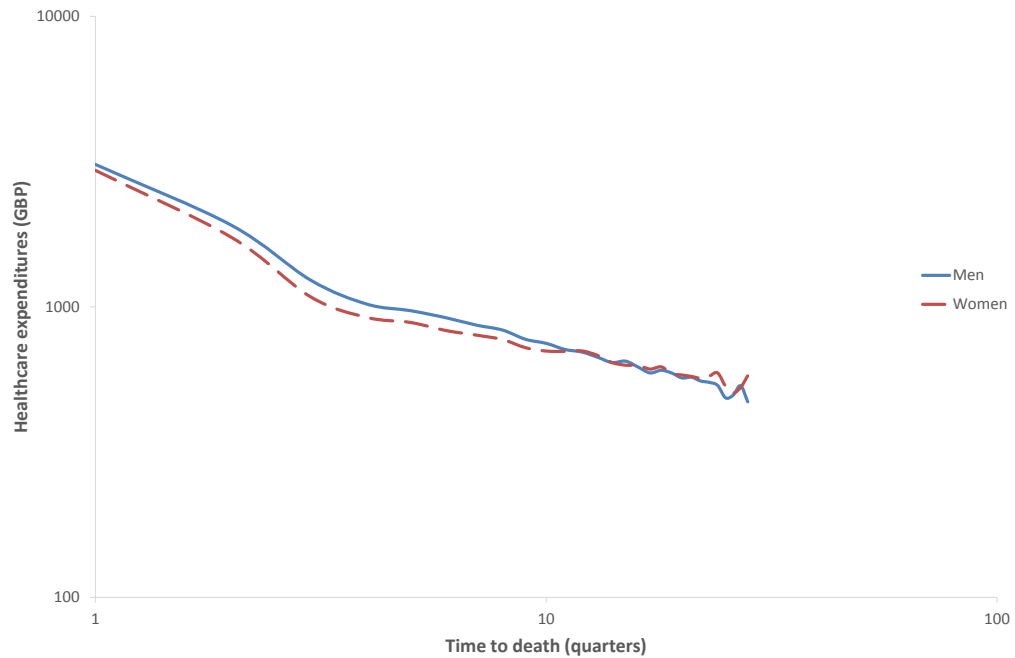


Figure 5: Average health care expenditures according to quarters to death (log scale for x- and y-axes)

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.

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

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:

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

We argue that individual morbidity is an omitted variable in this type of model. Accordingly, we augment the model as follows:

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

where CCS_n represents a recorded morbidity of CCS type n ($n = 1 \dots 260$). 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 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. 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.

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 coefficient.¹¹

Table 5 presents the results of various specification of a random effects panel data model of $\log(\text{HCE})$ on age, $\log(\text{TTD})$ 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 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 adopted¹².

¹¹ All results presented here employ one wave of the YPLL instrument. Where both instruments appear as relevant at the first stage, we estimate 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, 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.

¹² 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) |
| Men | | | |
| 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*** |
| | (.00001) | (.00001) | (.00000) |
| log(TTD) | | -.27927*** | -.09789*** |
| | | (.00604) | (.00520) |
| Morbidities | | | included |

* $p < 0.05$ ** $p < 0.01$ *** $p < 0.01$

Table 6: Results, first wave sampling.

| Model | Missing observations treated as missing | | | | |
|--|---|------------|---------------|-----------|-----------------|
| | (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) |
| <i>Joint F-test of relevance (p-value)</i> | | | | 0.0000 | 0.0003 |
| 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) |
| <i>Joint F-test of relevance (p-value)</i> | | | | 0.0000 | 0.0006 |
| Morbidities | | | included | | included |

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| Model | Missing observations treated as zeros | | | | |
|--|---------------------------------------|-------------------------|------------------------|-------------------------|-----------------------|
| | (1) | (2) | (3) | (4) | (5) |
| Men | | | | | |
| Age | -.06500*** (.02465) | .10191*** (.02265) | -.00691 (.01462) | .28057*** (.09001) | .03547 (.06062) |
| Age ² | .00049*** (.00016) | -.00088*** (.00015) | .00001 (.00010) | -.00363*** (.00113) | -.00036 (.00058) |
| log(TTD) | | -1.19842*** (.01205) | -.18669*** (.00745) | -2.05176*** (.38267) | -.31020 (.23856) |
| <i>Joint F-test of relevance (p-value)</i> | | | | 0.0000 | 0.0000 |
| Morbidities | | | included | | included |
| Women | | | | | |
| Age | .06863*** (.00928) | .08155*** (.00934) | .02635*** (.00806) | .61590 (.40329) | .05830 (.05937) |
| Age ² | -.00039*** (.00928) | -.00048*** (.00006) | -.00016*** (.00005) | -.00393 (.00261) | -.00038 (.00040) |
| log(TTD) | | -.15705*** (.00801) | -.03723*** (.00716) | -6.43066 (4.95179) | -.64920 (.8581702) |
| <i>Joint F-test of relevance (p-value)</i> | | | | 0.0000 | 0.0000 |
| Morbidities | | | included | | included |
| * $p < 0.05$ ** $p < 0.01$ *** $p < 0.01$ | | | | | |

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. 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. 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. First-stage regressions show, as expected, a negative and significant relationship between YPLL and TTD and an F-test of these instruments strongly suggests their relevance as a predictor of TTD (with p-values of between 0.0000 and 0.0006 obtained).

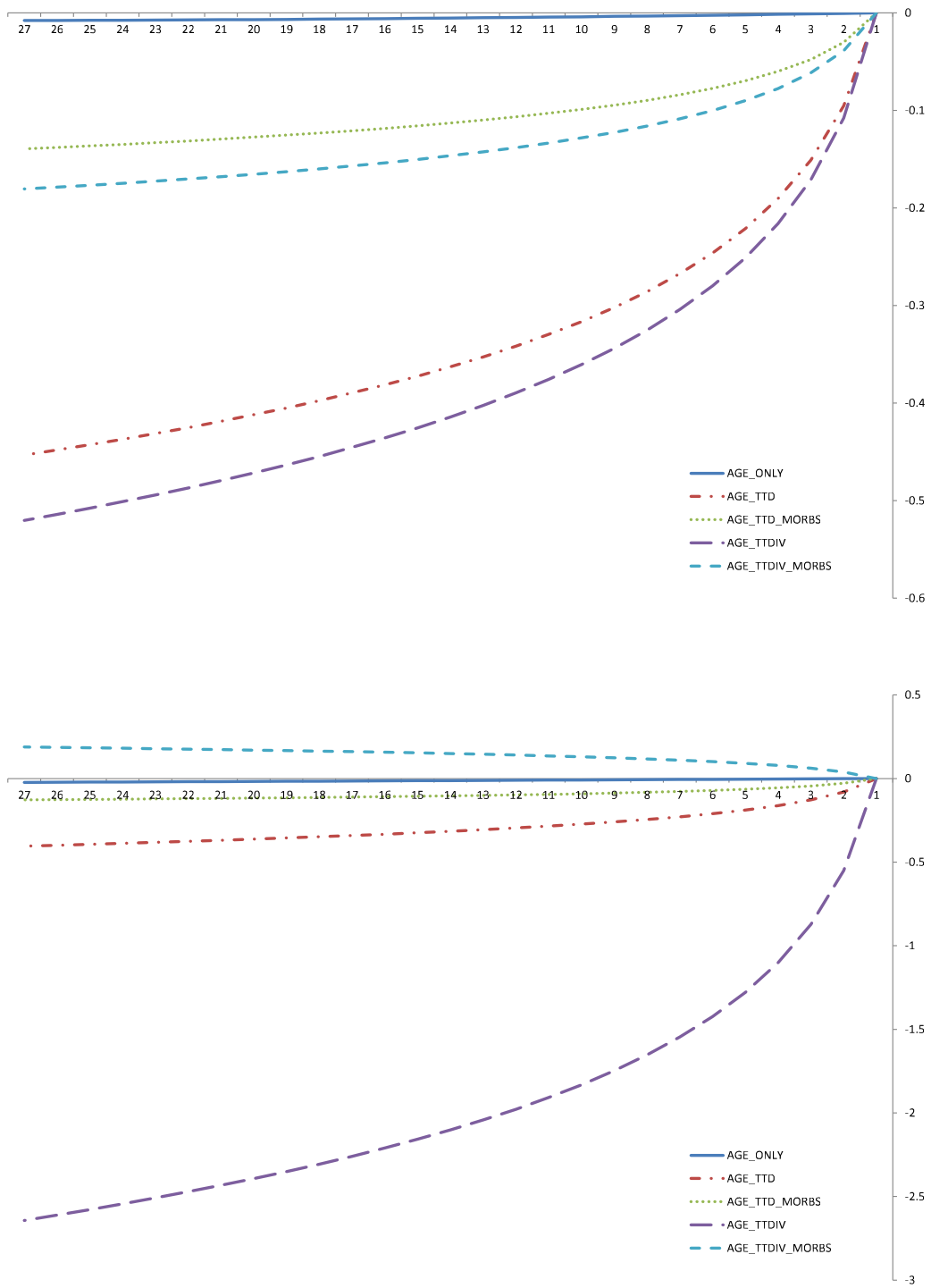


Figure 6: Change in HCE according to time-to-death and age, hypothetical individual dying at 75 (top – men, bottom – women)

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 morbidity 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. We extend this literature by showing that TTD in large part proxies for morbidity. 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.

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 |
| 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 |

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| | |
|-----|---|
| 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 transmitted disease) |
| 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) |
| 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 |

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| | |
|-----|--|
| 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 | Other 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 |
| 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 |

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| 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 |
| 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 |

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| 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 |
| 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) |

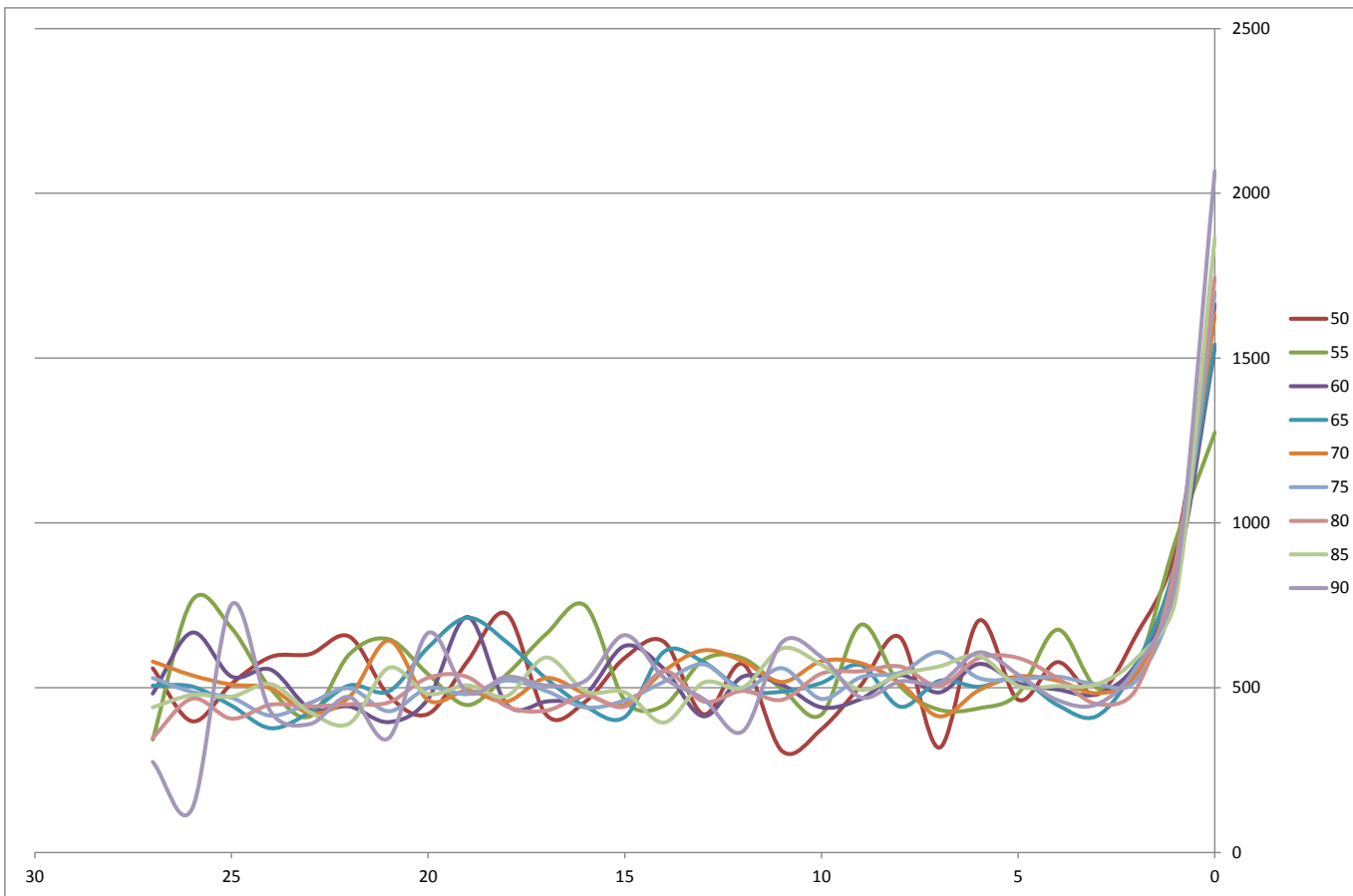


Figure 7: Healthcare expenditures by proximity to death, males by age

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