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Inter-cohort shifts in chronic disease, dementia, and mortality

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ABSTRACT

Previous work using U.S. data has identified generational shifts, reflected in inter-cohort changes, in the incidence and prevalence of diseases in older ages. This study extends previous findings to England by examining similar results in memory complaints, heart conditions, stroke, diabetes, lung disease, and cancer using data from the English Longitudinal Study of Ageing (ELSA). We fit Cox proportional hazard models to the first eight waves (2002–2016) of the ELSA sample ($n = 18{,}528$). In addition to exploring shifts in disease incidence we also examine shifts in disease mortality. Both general and sex-related differences are examined. Disease incidence has increased for later-born cohorts in England, replicating similar trends in the U.S. Not all diseases showed differences between men and women, but when differences were identified, women had lower risks for disease. In comparison to the U.S. sample, disease trends in England are more negative (i.e. accelerated failure times) for more recently born cohorts. These results showing increasing incidence of disease among the later-born cohorts suggest the possibility of increased disease burden in coming years.

Introduction

Older adults report increasing disease diagnoses over time (Hung et al. 2011). In a U.S. sample, disease burden increased across cohorts (Crimmins et al. 2019). Here, we perform a cross-national replication and extension of those findings from Crimmins et al. (a related all-disease investigation across five countries in Europe is presented in Dolejs 2015). Using survival analyses, we examine cohort trends in disease incidence and mortality among participants in the English Longitudinal Study of Ageing (ELSA; Clemens et al. 2019). We examine the incidence and associated mortality in relation to specific diseases, including dementia, stroke, heart conditions, lung disease, diabetes, and cancer. These

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diseases represent the most common causes of mortality; even small changes in their incidence could spark large changes in future disease burden (Brookmeyer et al. 2007).

Some of these diseases have shown increased incidence, prevalence, or both, over time, while others have not. For example, diabetes has increased in both incidence and prevalence, in parallel with obesity rates (Menke et al. 2015; Passa 2002; Skyler and Oddo 2002). Conversely, prior results point to decreased incidence in stroke, dementia and Alzheimer's disease, although changes in diagnostic criteria may mask some of the actual changes (Cognitive Function and Ageing Studies (CFAS) Collaboration et al. 2016; Grasset et al. 2016; Koton et al. 2014). Despite (potentially) decreasing incidence, the burden of these diseases is forecast to increase (Brookmeyer et al. 2007). Furthermore, research has linked dementia and Alzheimer's disease incidence to cardiovascular diseases (Newman et al. 2005) and excess weight (Ma et al. 2020). As a result, given the large shifts in obesity over time, Alzheimer's disease and dementia incidence may increase. Research has shown increasing trends in disease related to obesity (Atlantis, Lange, and Wittert 2009; 2017), and as the current population ages we may expect worsening general health.

This study aims to replicate and extend the findings of Crimmins et al. (2019), providing a cross-national comparison of results from the USA and England. We are particularly interested in cohort differences in disease incidence and mortality, and whether or not more recent cohorts exhibit changes in disease burden. Justification for a focus on cohort comparisons can be found across the literature (e.g., see Clouston et al. 2021; Soneji 2006; Zheng and Cheng 2018). Recent and similar cohort-based work was reported for grip strength at older ages in O'Keefe et al in the ELSA sample (O'Keefe et al. 2022), and for cognitive aging using an array of cognitive variables in the ELSA sample in O'Keefe et al. (2023) and in the National Health and Aging Trends Study in Zhang et al. (2024).

Methods

Participants

Data came from the English Longitudinal Study of Aging (ELSA). The data consist of an approximately representative sample of people living in England aged 50+ (Mean = 67.08, SD = 9.82). Data collection began in 2002 and continues on a biennial basis. Refreshment samples, to account for attrition and allow the continuation of the study, were added in the third, fourth, sixth, seventh and ninth waves (see the supplemental material for flowchart). We limited analyses to the 18,528 core subjects from waves 1–8 (including core members from refreshment samples), with data in any wave. ELSA collected data not only on the primary "core" sample, but on additional subjects as well, such as spouses of the core members. It was these additional subjects that we excluded in order to maintain approximate population representativeness. Note that we did not apply sampling weights in our analyses, as they were designed for cross-sectional rather than longitudinal analyses. There are, of course, threats to the external validity of the ELSA sample, caused by attrition and left censoring/truncation (earlier death/disability) of respondents, which can introduce selection bias. Although this study capitalizes on external validity emerging from a national probability sample, the primary focus

Table 1. Number of observed disease diagnoses by disease.

	Dementia	Heart Conditions	Stroke	Diabetes	Lung Disease	Cancer
total cases	491	4233	1183	2070	1429	2025
percent of participants	0.03 %	0.26 %	0.07 %	0.13 %	0.09 %	0.13 %
total observations	16057	16058	16058	16058	16057	16057

^^ This table shows total observed cases, the percent of participants who reported a case, and the number of participants with available data, for each condition studied.

is on internal validity. After restricting our analyses to the core members from these waves, 54% of the sample were female and 96% were white.

Measures

Demographics

We included three demographic variables: age, sex, and birth date. Age and birth date essentially measure the same demographic variable and were measured using the year of birth, and the whole number age the respondent reported during their interview. We used birth year as the relevant cohort measure. For cohorts from 1912–1963 there were over 120 observations for each cohort. Data were also included for cohorts from 1908–1912 and for 1964, but there were substantially fewer observations (<40). Sex was measured as a binary variable, with Male = 0 and Female = 1.

Chronic Diseases

Participants reported their disease status (whether they had ever been told by a physician that they had a given disease) at every wave. We selected a subset of diseases for analysis: Alzheimer's disease, dementia, memory complaints, heart conditions, stroke, diabetes, lung disease, and cancer. According to the documentation associated with ELSA, diabetes includes both Type I and Type II diabetes. All disease states were self-reported. Self-reports of dementia, Alzheimer's disease, and memory problems diagnoses were collapsed into a single variable, because of their overlapping symptomatology and to increase statistical power; this variable is referred to here at "dementia." Table 1 provides the overall prevalence of these diseases in our sample (i.e., the number of unique cases reported). As we are using only the core participants, who were randomly selected from the population, the analytic sample is approximately population representative, up to attrition and left censoring/truncation (earlier death/disability that interferes with later reports).

Data Analysis

Our approach differed from prior work in order to better account for interval and left censoring. Interval censoring occurs when the exact date of disease onset is unknown. In our case, the survey design only allows us to determine disease onset within a two-year window (i.e., between survey waves). Left censoring occurs when disease onset occurs prior to the first interview date (but the participant still enters the study). Left censoring stands in contrast to left-truncation, where disease onset prevents participation in the study (e.g., because participants die from disease prior to study entry). We used a semi-parametric Cox model (available in the icenReg package in R), to manage these censoring issues in our

disease incidence models. Birth year was scaled so that a one-unit increase represented a one-decade change in time, and was centered at 1930. In addition, we used the natural scale of age measured in years, as the time-to-event. The model can be seen in equation (1). The model presents the linear predictor aspect of the model, the baseline hazard of the model is omitted. All models were fit using the "survival" and "icenReg" packages (Anderson-Bergman 2017; R Core Team 2021; Therneau 2021). There are two primary types of models used: those that predict a condition (dementia, cancer, diabetes, etc.) and those that predict mortality. In the mortality models, condition becomes a predictor because we want to know if having a condition increases the likelihood of death, and how that varies across cohorts. Cohort represents birth year (as scaled above), and is treated as a continuous quantitative variable in these models.

$$(1)\widehat{condition} = \beta_1 \times Cohort + \beta_2 \times Sex$$

For mortality we used a model predicting death from sex (Male = 0, Female = 1), birth cohort, disease status (condition), and the interaction of cohort and disease status (equation (2)). These models allow us to evaluate if disease increased the likelihood of death and if the effect of disease varied across cohort. For all mortality models, disease status was evaluated at study entry.

$$(2)\widehat{mortality} = \beta_1 \times Cohort + \beta_2 \times Sex + \beta_3 \times Condition + \beta_4 \times Cohort \times Condition$$

Results

To assess the proportionality assumption of our incidence models, Turnbull estimates (similar to Kaplan–Meier curves) were created for each outcome variable for sex and a discretized cohort variable. The resulting hazards were plotted and visually inspected, and results suggested that hazards were approximately proportional. The only notable exceptions were heart disease between cohorts, and cancer between men and women. These exceptions were not thought to be substantial enough to warrant a different modeling approach. Table 2 provides the hazard ratios and associated confidence intervals for the disease incidence models. Table 3 displays coefficients and associated confidence intervals for the mortality models. The general finding is that later-born generations tend to have increased disease risk. When sex differences were observed, they favored women (i.e., women always had a higher survival time if there were statistically significant sex

Table 2. Results from interval-censored cox models for disease incidence.

	Cohort	sex (Male as reference)
Dementia	2.23 (1.95, 2.56)	0.94 (0.78, 1.13)
Stroke	1.23 (1.14, 1.31)	0.74 (0.66, 0.83)
Heart conditions	1.32 (1.27, 1.37)	0.78 (0.73, 0.82)
Diabetes	1.55 (1.47, 1.62)	0.72 (0.66, 0.78)
Lung Disease	1.34 (1.27, 1.42)	0.79 (0.72, 0.88)
Cancer	1.76 (1.67, 1.85)	1.06 (0.97, 1.16)

Note that the table presents the exponentiated model coefficients (and associated confidence intervals in parenthesis), which can be directly interpreted as hazard ratios. Models were fit separately for each condition listed. The Dependent Variable is the disease on the row, within Equation 1.

Table 3. Results from censored Cox models for mortality accounting for disease diagnosis.

	Cohort	Sex	Condition	Cohort X Condition
Dementia	5.75 (5.21, 6.36; p-value <0.001)	0.68 (0.63, 0.74; p-value <0.001)	3.71 (2.66, 5.10; p-value <0.001)	1.42 (1.09, 1.84; p-value = 0.007)
Stroke	5.70 (5.16, 6.30; p-value <0.001)	0.68 (0.63, 0.74; p-value <0.001)	2.16 (1.88, 2.51; p-value <0.001)	1.51 (1.32, 1.73; p-value <0.001)
Heart Conditions	5.64 (5.10, 6.30; p-value <0.001)	0.70 (0.64, 0.76; p-value <0.001)	2.01 (1.84, 2.18; p-value <0.001)	1.35 (1.25, 1.48; p-value <0.001)
Diabetes	5.70 (5.16, 6.30; p-value <0.001)	0.69 (0.64, 0.75; p-value <0.001)	1.63 (1.45, 1.86; p-value <0.001)	1.09 (0.96, 1.25; p-value = 0.197)
Lung Disease	5.58 (5.05, 6.17; p-value <0.001)	0.68 (0.63, 0.74; p-value <0.001)	2.41 (2.12, 2.72; p-value <0.001)	1.57 (1.39, 1.79; p-value <0.001)
Cancer	5.58 (5.05, 6.17; p-value <0.001)	0.67 (0.62, 0.72; p-value <0.001)	2.03 (1.80, 2.32; p-value <0.001)	1.39 (1.23, 1.58; p-value <0.001)

Note that the table presents the exponentiated model coefficients (and associated confidence intervals in parenthesis), which can be directly interpreted as hazard ratios. Models were fit separately for each condition listed. The Dependent Variable is Mortality, with Condition referencing the disease on the row, within Equation 2.

Table 4. Results from censored cox models for mortality accounting for disease diagnosis omitting sex from the model.

	Cohort	Condition	Cohort X Condition
Dementia	5.81 (5.26, 6.42; p-value <0.001)	3.86 (2.77, 5.31; p-value <0.001)	1.49 (1.15, 1.92; p-value = 0.002)
Stroke	5.75 (5.21, 6.36; p-value <0.001)	2.23 (1.92, 2.56; p-value <0.001)	1.51 (1.31, 1.75; p-value <0.001)
Heart Conditions	5.70 (5.16, 6.30; p-value <0.001)	2.05 (1.88, 2.23; p-value <0.001)	1.35 (1.25, 1.48; p-value <0.001)
Diabetes	5.75 (5.21, 6.36; p-value <0.001)	1.70 (1.49, 1.92; p-value <0.001)	1.09 (0.96, 1.25; p-value = 0.18)
Lung Disease	5.64 (5.10, 6.23; p-value <0.001)	2.44 (2.16, 2.77; p-value <0.001)	1.54 (1.35, 1.73; p-value <0.001)
Cancer	5.58 (5.05, 6.17; p-value <0.001)	1.95 (1.72, 2.20; p-value <0.001)	1.40 (1.25, 1.58; p-value <0.001)

Note that the table presents the exponentiated model coefficients (and associated confidence intervals in parenthesis), which can be directly interpreted as hazard ratios. Models were fit separately for each condition listed.

differences). Figure 1 shows the expected prevalence of diseases (the plots presume that there are no deaths) for men and women across three generations, each born 10 years apart. In the figure "early" represents the cohort born in 1920, "middle" is the cohort born in 1930, and "late" is the cohort born in 1940. Across the mortality models, the main effects of cohort and sex were relatively constant (approximately 5.70 for cohort and .79 for sex), because the mortality models used approximately the same data in calculating such effects. Of particular interest is the interaction between cohort and condition in each mortality model. A mortality model was fit in which Sex was omitted from the model, see Table 4; results were almost identical to results when Sex was in the model.

Dementia

Alzheimer's disease, dementia, and memory problems, combined into "Dementia" for these analyses, had a relatively low prevalence in our sample, with only 491 cases out 16,057 observations. The inclusion of memory problems in this combined variable did not affect the prevalence in our sample, but it was retained so that all memory-related conditions were included in this single combination variable. In the interval-censored Cox model, the (exponentiated) linear effect of cohort was 5.75 (95% CI: 5.21, 6.36). The effect of sex was 0.68 (95% CI: 0.63, 0.74). More recently- born cohorts showed an increase in dementia diagnoses, but men and women were not substantially different in their rates of diagnosis. Mortality due to dementia is linked to increases in later-born cohorts with an interaction between condition and cohort such that later-born cohorts with dementia were at greater risk of mortality (interaction effect = 1.42; 95% CI: 1.09,1.84). In a sensitivity analysis these findings were not substantially different when examining dementia alone or Alzheimer's Disease alone, these analyses are presented in the supplemental material.

Stroke

Strokes were somewhat common in our sample, with 1183 reported out of 16,058 observations, or approximately 1 in 16 people experienced a stroke at some point

either during or prior to their participation in the study. The (exponentiated) effect for cohort was 5.70 (95% CI: 5.16, 6.30); later born cohorts were at higher risk. The effect of sex was .68 (95% CI: 0.63, 0.74). In this case, sex was a particularly strong indicator of risk, with women having a risk approximately equivalent to men born 15 years earlier (recall that earlier-born cohorts had lower risks). Mortality due to stroke appears to be linked to increases among later-born cohorts (interaction effect = 1.51: 95% CI: 1.32, 1.73).

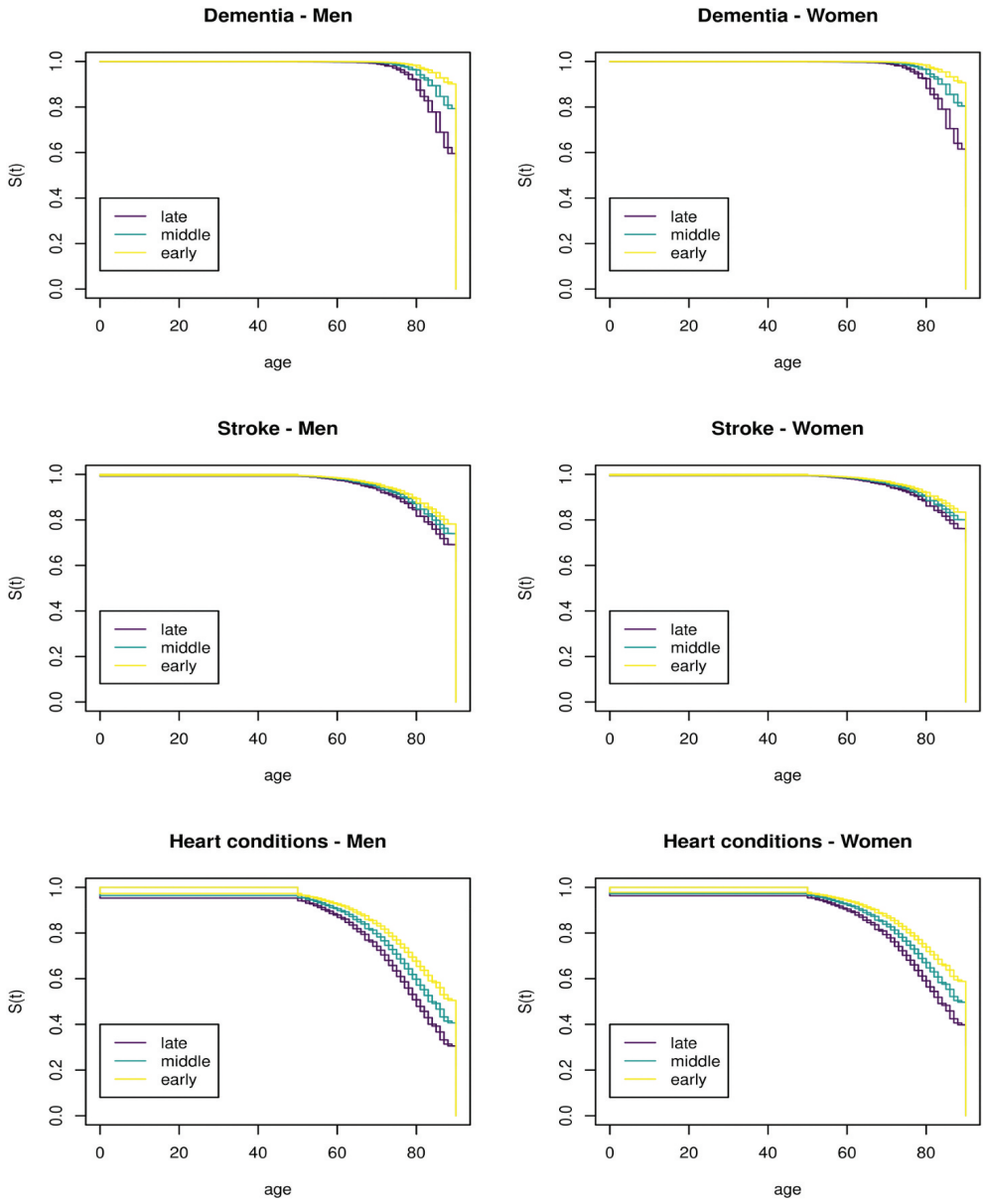

Figure 1a. Projected disease prevalence by sex and Cohort. Note: "Early" represents model implied trajectories for individuals born in 1920, "middle" the same for individuals born in 1930, and "late" is for individuals born in 1940. The x-axis is age, while the y-axis represents the proportion of individuals expected to still be disease free at a given age.

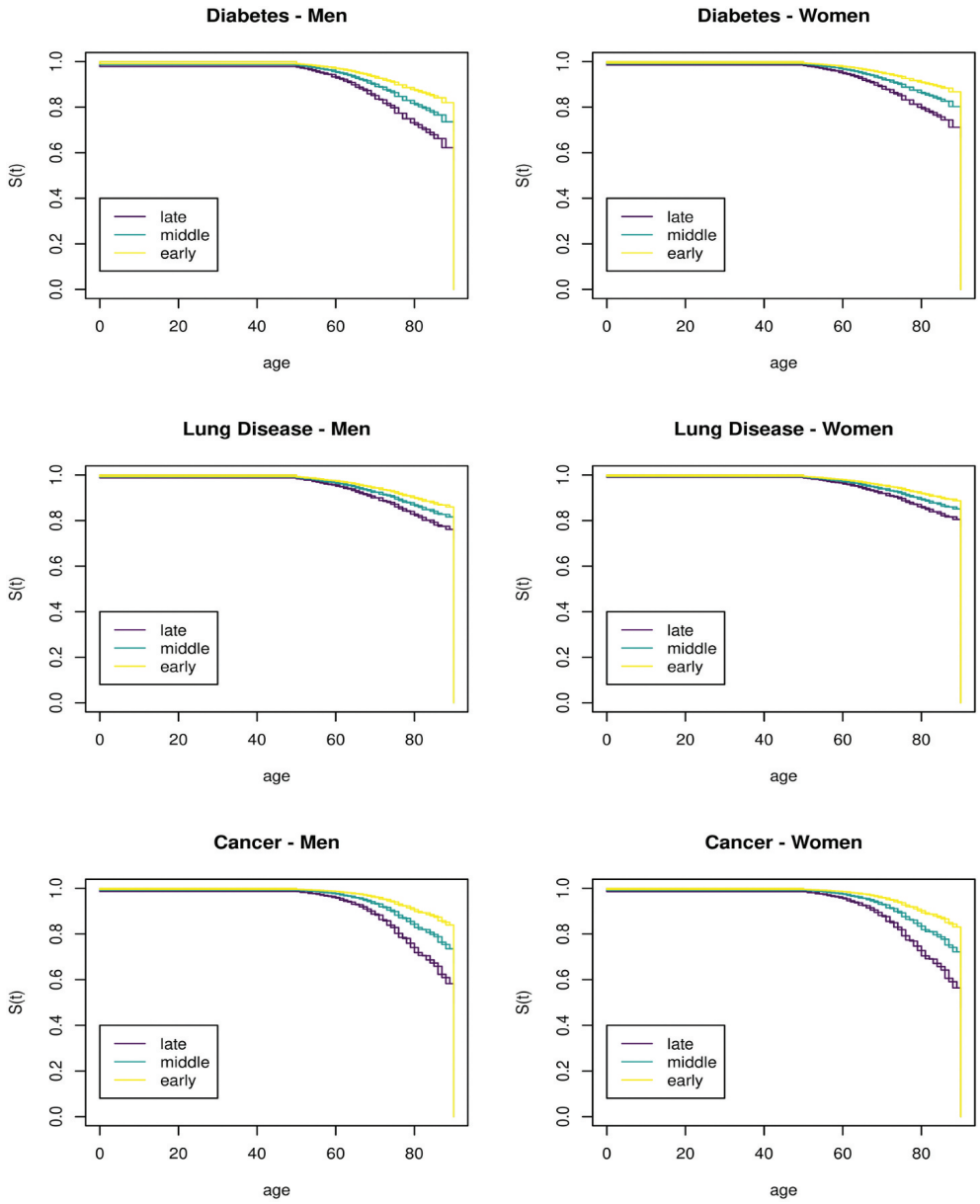

Figure 1b.

Heart Conditions

Heart conditions had a high prevalence in our sample relative to dementia and Alzheimer's disease. There were 4233 reported cases of heart conditions in the ELSA sample (26.40% of the sample). The (exponentiated) effect of cohort was 5.64 (95% CI: 5.10, 6.30). Sex had an effect of .70 (95% CI: 0.64, 0.76). For mortality, as with the other conditions, there were higher rates of mortality due to stroke for the later-born cohorts (interaction effect = 1.35; 95% CI: 1.23, 1.58).

Diabetes

Diabetes had a prevalence of 12.90% ($n = 2070$) in the sample. The (exponentiated) effect of cohort was 5.70 (95% CI: 5.16, 6.30). The effect of sex was 0.69 (95% CI: 0.64, 0.74). Being in a later born cohort was linked to increased mortality, the effect of mortality due to diabetes was not different across cohorts (unlike for previous diseases) with the interaction effect being 1.09 (95% CI: 0.96, 1.25).

Lung Disease

Lung disease was relatively common, with 8.90% ($n = 1429$) reporting lung disease. The (exponentiated) effect of cohort was 5.58 (95% CI: 5.05, 6.17). The effect of sex was 0.68, (95% CI: 0.62, 0.72). In the mortality model, the interaction effect between cohort and condition was 1.57 (95% CI: 1.39, 1.79).

Cancer

Cancer was relatively common, with 12.60% ($n = 2025$) of the sample having cancer at some point during or prior to the study. The (exponentiated) effect of cohort on incidence was 5.58 (95% CI: 5.05, 6.17). The effect of sex was 0.67 (95% CI: 0.62, 0.72). The interaction effect of cancer and cohort on mortality was 1.39 (95% CI: 1.23, 1.58).

Discussion

We examined birth-cohort shifts in the U.K. in the incidence of chronic disease and a dementia cluster, and also mortalities associated with each disease (i.e., models for incidence, and models for mortality). Our findings suggest that, across a number of diseases, the incidence of disease has increased for later-born cohorts. Furthermore, mortality was found to be higher among later-born cohorts. As part of a broader tapestry of related findings, our results partially match the findings of Crimmins et al. (2019). Compared to the Crimmins et al. U.S. sample, the results of the present study are even more negative for more recently born cohorts in England, as we found higher incidence of diseases in later-born cohorts for more diseases. Our results concerning disease mortality were partially in line with previous work, although we did not find reduced mortality for cancer. It is worth noting that Crimmins et al. 2019 used a different analytical framework, discretizing the age and cohort variables. We provide analyses and results comparable to those of Crimmins et al. (2019) in our appendix, and these results more closely replicate the results of Crimmins et al. (2019). We also fit models dropping out Age, Sex, and both Age and Sex, and found relatively stable results. We note that their analytic method is subject to certain limitations, such as a high number of tied survival times, which are problematic in Cox proportional hazards model, and substantial information loss due to discretizing continuous variables.

Extending our comparisons beyond the context of the U.S. we find that results are inconsistent across nations, even within nations at times. Our results broadly replicate the findings of Wennberg et al. (2023), who found that frailty had increased for men and women in Sweden. Although we did not examine frailty directly, findings of

increased disease prevalence would be expected with increased frailty. Examining heart disease, stroke, and dementia, Morovatdar et al. (2022) found age-related increases in prevalence, but generally no increase in incidence (once aging of the population was accounted for) in the Global Burden of Disease Study, which allowed examination of many countries simultaneously. Stephan et al. (2018) found that dementia incidence had declined somewhat, but this decline in incidence is not universal. Their findings were sometimes contradictory within country, and in one case results were contradictory within the same study. Finally, in the U.S. based Framingham Heart Study, dementia incidence was found to decline across a 30-year period (Satizabal et al. 2016). The Framingham Heart Study had the advantage of being able to ensure diagnostic consistency across years, which is not controlled in ELSA.

Considering non-cognitive diseases, stroke, diabetes, cancer, heart conditions, and lung disease, a number of these diseases and conditions showed cohort shifts. Several of these diseases, such as heart conditions, stroke, and diabetes, have well known associations with obesity, which has shown a dramatic increase over time (2017). Hernández et al. (2021), found that obesity increased the odds of later-life disease clusters substantially, moreso, in fact, than nearly any other risk factor examined in their study. Atlantis, Lange, and Wittert (2009), stated that many chronic conditions could be avoided by avoiding obesity, in particular, diabetes and high-cholesterol. They found that cardiovascular disease and high blood pressure have not seen similar increases, attributed to increased and focused management of those specific diseases. Cancer and lung disease have been associated with increases in obesity (Avgerinos et al. 2019; Colditz and Peterson 2018; Dixon and Peters 2018; Peters et al. 2018). Furthermore, some diseases related to obesity (e.g., diabetes and heart conditions) are associated with increased risk of other disease diagnoses (e.g., dementia) (Armstrong et al. 2019; Zilkens et al. 2013), which we found to have also increased over time. Because of the design of our study, if rising rates of obesity were causing increased disease burden, it would appear as a cohort effect in our models (later born cohorts would become obese younger and would be obese for longer). Unfortunately, due to the design of the ELSA, it is not possible to directly test this hypothesis with these data. ELSA was inconsistent in their measurement of BMI-associated variables (weight and height), and we lack a measure of life-long exposure to high BMI (data are only available, at the earliest, at age 50 for core participants). Overall, however, our findings regarding diabetes are similar to findings in India (Nanditha et al. 2019) and Italy (Gnavi et al. 2018). In the Italian context, it was found that aging was partly responsible for increases in overall incidence (as would be expected in an aging population), but that changes in BMI were also partly, but not wholly, responsible for shifts in diabetes diagnoses. Although not a primary goal of the Framingham Heart Study, they also found increases in diabetes (Satizabal et al. 2016).

In the broader context of dementia research, our findings are somewhat unexpected. For example, Derby et al. (2017), found that dementia incidence was declining among successive birth cohorts in the Einstein Aging Study. Matthews et al. (2013) found decreasing rates of dementia across cohorts in England. Stephan et al. (2018) conducted a systematic review of secular trends in dementia, finding that the extant evidence is inconclusive. There is evidence for declines, particularly in some high-income nations, however this evidence is contradicted by other findings within the same countries. Morovatdar et al. (2022) found an increasing burden of disease, including dementia, due to population aging, but found that the age-standardized incidence was declining, which was true for their global sample, but

especially so for OECD countries. Finally, Grasset et al. (2018), found decreasing dementia incidence among women (although not men) during the late 20th century.

An important distinction between our study and some of the aforementioned studies is the use of self-report versus algorithmic diagnostic criteria. Our study used self-report disease diagnosis, which may be susceptible to changes in diagnostic criteria, and awareness among the medical community, among other things, a limitation in any study utilizing self-reports. As partial explanation, we note studies (Clouston et al. 2021; Grasset et al. 2018) showing that the lack of algorithmic criterion can mask declines in dementia incidence. However, even if the sample itself is not becoming sicker, the influx of new diagnoses may still represent increased costs as previously undiagnosed cases of dementia become diagnosed (and presumably treated) cases of dementia. While this process is likely to improve individual well-being, it could represent increased costs that individuals, and society, will bear. Of particular importance for our sample, starting in 2009 the British National Health Service introduced the "Health Check" for everyone aged 40+ . This program, and others like it, may increase detection of disease in more recent years (which would favor detection among later-born cohorts). Earlier detection of disease is consistent with our findings.

Although our results, with regard to dementia, may represent some level of increased awareness at the individual level, an increase in dementia diagnoses is also consistent with the increased prevalence of obesity, which, as previously noted, may be driving the other patterns observed. Razay, Vreugdenhil, and Wilcock (2006), found that obesity significantly influenced the odds of receiving an Alzheimer diagnosis, as did Ma et al. (2020). In a study on the effects of bariatric surgery on mild cognitive impairment, such impairment was prevalent among adults with severe obesity, even among younger adults (Rochette et al. 2016). Similarly, adolescents who had insulin resistance and were obese showed significantly elevated levels of amyloid β-protein and presenilin 1, both biomarkers of Alzheimer's disease (Luciano et al. 2015). Moreover, in a correlational study, deliberate weight loss among older adults was associated with cognitive improvements (Horie et al. 2016). In summary, studies suggest that obesity is related to cognitive impairments that are similar to Alzheimer's disease and related dementias. Given the obesity epidemic, a rising rate of diagnosis of dementia and Alzheimer's disease may be indirectly related to an obesity-related increase. Obesity is a tempting candidate explanation, as other obvious potential causes have seen marked declines over the same time period (e.g., smoking), and plausible protective factors have seen an increase over time (e.g., education).

We have included analyses of cohort differences in mortality in an effort to provide a cross-national comparison to previous results (Crimmins et al. 2019), but these results, and others like them, should be interpreted cautiously. The results could represent real shifts in mortality, but it is possible that survivorship bias could be affecting our results. Survival models generally have difficulty with left truncation (i.e., because those who died before the study were never included in the study). Unlike disease incidence, in which we had left censoring (the exact timing of first occurrence of the disease was not observed, but people with the disease could still enter the study), left truncation will have systematic effects across cohorts such that the older cohorts will have seen greater levels of left truncation. This would bias the results of survival models (only the healthiest members of the oldest cohorts make it into the data). For the present analyses, it is possible that mortality has increased for later-born cohorts, but it is plausible that some of the patterns could be due to sampling bias. Virtually any panel study like the ELSA will have precisely these difficulties. Using other

datasets that have different features – for example, one that would provide satisfactory measurement approaches to distinguish severity of different diseases – would be valuable in future research. Our study does improve upon previous work by accounting for the interval censored nature of our data, as well as allowing for left censoring. These features give us increased confidence in our results, particularly those for disease incidence.

The present study does call into question whether or not later born cohorts are leading healthier lives, at least in England. In research on international populations it remains an open question whether trends are positive or negative once aging patterns are accounted for. Our results for England suggest the possibility of more negative patterns. We are forced to confront the possibility that people may be becoming sicker earlier. If that is true it would require us to evaluate what has changed in people's lives among more recently born cohorts to cause worse health.

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No potential conflict of interest was reported by the author(s).

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Data Availability Statement

Data are available from the UK Data Service

Ethics Approval

The ELSA data received ethical approval from the appropriate institution at each wave. Approval information for each wave can be found at: https://www.elsa-project.ac.uk/ethical-approval.

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