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Full length article

## Healthy ageing trends in England between 2002 to 2018: Improving but slowing and unequal<sup>☆</sup>

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### ABSTRACT

Growing life expectancy and a rising proportion of older people make the issue of whether cohorts are ageing better a key individual, social and economic issue. Using data from the English Longitudinal Study of Ageing we characterise how frailty develops with age, how this differs across demographic groups, whether more recent cohorts are ageing better and what the key areas of focus for health policy should be. We find cohort effects such that frailty at each age has been decreasing over time but that this trend shows modest signs of slowing and is less pronounced for those with lower wealth. Improvements across cohorts reflect improvements in ADLs, cognitive function, and mobility but limited progress in reducing the incidence of diseases such as cancer, cardiovascular disease, etc. We find mobility and ADLs the main driver of average differences across regions but cross-regional differences are driven more by within than between group inequality.

### Introduction

Like most countries the UK has experienced a long-term trend of rising life expectancy and a growing proportion of older people. Life expectancy at birth has risen from 78.2 years in 2002 to 81.1 in 2018 (ONS, 2019b). The number of people aged 50 years and over has risen from 17 million (31% of the population) in 1970 to 25 million (37%) in 2018 and is expected to reach 31 million (42%) by 2043 (ONS, 2019a).

These trends make achieving healthy longevity a key individual, social, and policy priority (Scott, 2023). Aside from the potential benefits to individuals (Scott et al., 2021), if longer lives are also healthier then employment at older ages can rise, thus boosting GDP (Banks et al., 2011; Berkman and Truesdale, 2023). Similarly, improvements in health at older ages can reduce the costs associated with age-related diseases and care (Kingston et al., 2018). Given the UK Office for Budget Responsibility predicts an increase in age-related spending by nearly 4% of GDP in the years ahead (Office for Budget Responsibility, 2018), the potential savings are substantial. Combined, these effects point to the multi-billion-dollar benefits of achieving healthy longevity.

Unfortunately, there are growing concerns that UK health outcomes are deteriorating (Marshall et al., 2015), that healthy life expectancy

is not keeping pace with increases in the State Pension age (Lynch et al., 2022) and that withdrawals of older workers from the labour market are being driven by long term illness (Haskel and Martin, 2022). These concerns are particularly acute around widening health inequality (Marmot, 2020), as life expectancy improvements stall and even reverse for some groups (Rashid et al., 2021), while the number of years spent in poor health increases (Welsh et al., 2021). These inequalities are increasingly a policy concern, and the UK government has committed to reduce regional health inequalities as part of the “Levelling Up” programme (UK Government, 2022).

To examine whether cohorts are ageing better, this paper utilises nine waves of the English Longitudinal Study of Ageing (ELSA) dataset, covering the period 2002 to 2018, to answer the following questions: i) how does health vary with age for those aged over 50 years and how does this compare with other countries? ii) what are the trends over time in how people are ageing and has there been any improvement? iii) what are the differences in these trends due to sex, regions, income, and education? iv) which aspects of health have driven any improvements or deteriorations? v) which areas of health improvements should be the focus of policy? vi) what factors lie behind regional inequalities?

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As an empirical proxy for healthy ageing we follow the Frailty Index approach advocated by [Mitnitski et al. \(2001\)](#) and [Searle et al. \(2008\)](#) which has proved popular in the economics literature. This offers a convenient way of summarising overall health and functionality across a broad range of indicators. We compare not just how ageing is varying across cohorts in England but also draw comparisons with the results of [Abeliansky and Strulik \(2018, 2019\)](#) for Europe and [Abeliansky et al. \(2020\)](#) for the United States. Compared to previous studies on English data ([Marshall et al., 2015](#); [Niederstrasser et al., 2019](#)), we make use of longer time spans and control for unobserved but systematic individual level selection effects rather than relying only on fixed effects or control variables. This leads to important differences in results. We also decompose the trends in the aggregate frailty index by studying the contribution made by different sub-components to understand the main drivers of changes in frailty at each age and illuminate the areas which require greater policy focus.

**Data**

We use data from waves 1 through 9 of the English Longitudinal Study of Ageing (ELSA) covering the period 2002 to 2018. Both the original sample and all replacement samples are part of the analysis. Survey responses on disease conditions, ADLs/IADLs, depression, and cognitive function are used to construct a measure of individual frailty ([Mitnitski et al., 2001](#); [Searle et al., 2008](#)). This frailty index measures the proportion of health conditions or limitations an individual is experiencing at a specific age. In constructing our index we use the same items as [Rogers et al. \(2017\)](#), subject to data availability constraints.

Our frailty index contains potentially 50 items covering mobility difficulties, functional disabilities (ADLs/IADLs), general health, depressive symptoms, the prevalence of health conditions, and cognitive function. For a comprehensive description of the construction of our frailty index, see Appendix A.1.

Following the literature ([Searle et al., 2008](#); [Abeliansky et al., 2020](#)) we impose several restrictions on our sample. We include only individuals i) aged between 50 and 90 years (the number of observations and so the precision of statistical estimation decreases at higher ages) ii) born in the UK (to ensure results are not driven by immigration and the influence of childhood years) iii) for whom we have available data on at least 30 out of the 50 items so as to ensure reliability of the frailty index. Thus, from a total of 90,068 observations on 19,801 individuals, we use 78,858 observations on 17,269 individuals. The process of the sample selection is summarised in a flow chart in Appendix Figure A.1.

We are not only interested in the relationship between frailty and age and how this may have changed over time, but also the role of socio-economic determinants, which we include as covariates in the analysis. To identify regional trends, we obtained confidential ELSA data on individuals’ region of residence, which we aggregate up to nine NUTS-1 level regions. For education, we use the internationally standardised classification of completed education up to less than secondary level (38% in our sample), upper secondary level and vocational training (47%) and tertiary education (15%). For wealth, we follow [Marshall et al. \(2015\)](#) and use the natural logarithm of the sum of financial and housing wealth for a given household.

Before proceeding to estimation results it is helpful to look at the raw data. [Table 1](#) shows the mean value of our frailty index for different age bands from Waves 1 (2002), 5 (2010) and 9 (2018). Our study is focused on two issues: the relationship between frailty and age and whether this has changed over time. On the former, as expected, [Table 1](#) shows frailty increases with age within every wave. On the latter, the mean values broadly point to reductions in frailty for each age band across cohorts.

[Table 2](#) shows further evidence on changing health dynamics for three different waves of ELSA by detailing the cumulative distribution

**Table 1**  
Mean frailty index values by age group, waves 1-9.

↓ Wave	Age→	50–54	55–59	60–64	65–69	70–74	75–79	80+
1		0.091	0.106	0.118	0.126	0.149	0.171	0.223
5		0.094	0.097	0.108	0.122	0.142	0.170	0.241
9		0.083	0.096	0.115	0.110	0.131	0.160	0.216

**Notes:** The table shows the mean of the frailty index (measured as the proportion of health deficits reported by an individual, and ranging from 0 to 1) by age groups and ELSA waves.

of frailty levels for different age bands. It shows two somewhat discordant trends: on the one hand, across a variety of age bands, the number of people experiencing lower levels of frailty has increased, often substantially. Focusing on frailty levels below 0.3 for every age group there has been an increase between Waves 1 and 9 in the proportion of people at these lower levels of frailty, with only one exception. On the other hand, among older sections of the population, the share of individuals with very high levels of frailty has also increased, albeit at a smaller rate. Focusing on higher frailty levels above 0.5 there has been an increase in every age group between Waves 1 and 9 in the mass at these high levels. The shift in mass to lower levels of frailty is much larger than the shift to the higher levels but the increase in highest levels of frailty is especially marked for those aged over 80. This latter result is consistent with suggestions that improvements in medical and care services may serve to increase the survival of individuals with greater levels of frailty ([Marshall et al., 2015](#)). In other words, there has been an increase in the tails of the frailty distribution, with a larger increase at lower levels of frailty than in the upper tails.

**Results**

To better understand how frailty varies with age and other characteristics, we estimate the following relationship:

$$\ln F_{iw} = \alpha + \beta \cdot \text{age}_{iw} + \gamma_1 X_{1iw} + \gamma_2 X_{2i} + \varepsilon_{iw} \tag{1}$$

where  $F_{iw}$  denotes the frailty index for individual  $i$  in wave  $w$ ,  $\text{age}_{iw}$  denotes the age of the individual,  $X_{1iw}$  is a set of individual time-varying controls (region and wealth),  $X_{2i}$  is a set of individual controls that is time-invariant in our data (sex and education), and  $\varepsilon_{iw}$  represents an error term reflecting other factors which may be individual-specific and time-varying. We follow [Searle et al. \(2008\)](#) in using a logarithmic model for frailty rather than the linear framework of [Marshall et al. \(2015\)](#). A logarithmic formulation follows naturally from the Gompertz–Makeham law, facilitates easier interpretation of coefficients (the coefficient on age is the percentage increase in frailty each year), and is also preferred by a Box–Cox test for functional form.

The results are pictured in [Fig. 1\(a\)](#).<sup>1</sup> and show that age is a statistically significant predictor for individual frailty. The results reveal a wide range of variation in frailty at each age depending on demographic characteristics. Females have higher levels of frailty than males, frailty is decreasing in household wealth and higher education has a marked negative impact on frailty. There are also marked regional differences: conditional on age, gender, education and wealth, living in the North East, North West, or East Midlands of England, among others, is associated with significantly more frailty (although these effects are not found for individuals who migrate between regions).

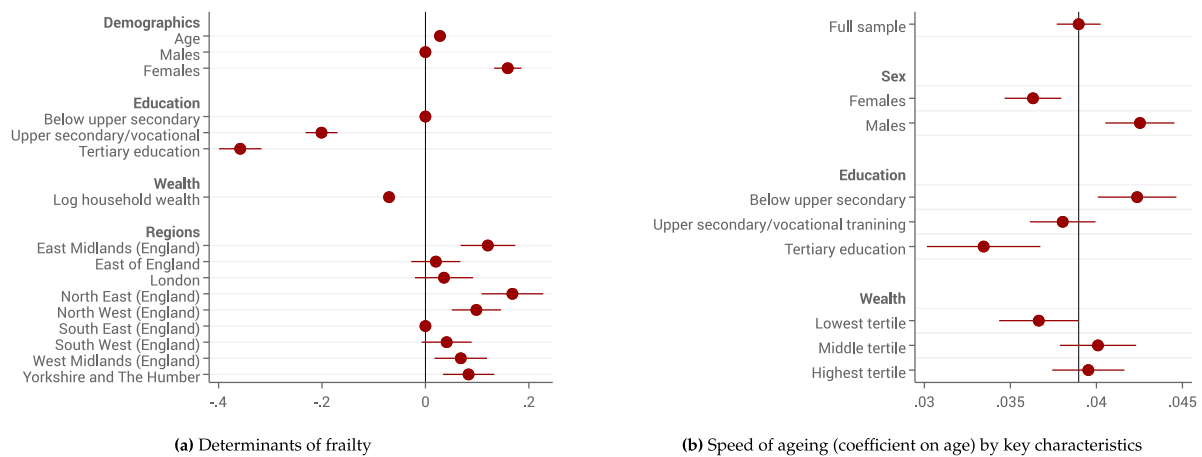
While [Fig. 1\(a\)](#) shows that the *level* of frailty differs across a variety of demographic characteristics, in [Fig. 1\(b\)](#), we investigate whether the *rate* of ageing itself (e.g the slope of the frailty function with respect to age) differs across these characteristics. Since sex, education, and

<sup>1</sup> Full regression results for both [Fig. 1\(a\)](#) and [Fig. 1\(b\)](#) are detailed in Appendix Table B.1.

**Table 2**  
Cumulative distribution of frailty across age bands and waves.

Frailty Index	50 to 64			65 to 79			80+		
	Wave 1	Wave 5	Wave 9	Wave 1	Wave 5	Wave 9	Wave 1	Wave 5	Wave 9
0.00	12.2%	11.7%	13.1%	4.2%	4.6%	5.2%	0.8%	1.1%	0.3%
0.05	42.8%	43.6%	46.2%	23.4%	26.0%	27.6%	6.7%	7.8%	8.3%
0.10	62.3%	63.8%	67.3%	43.8%	46.6%	50.9%	19.5%	18.4%	25.2%
0.15	77.2%	78.1%	80.6%	63.6%	65.9%	70.5%	38.2%	35.9%	44.3%
0.20	82.8%	84.2%	85.1%	72.9%	74.2%	78.3%	48.4%	47.5%	55.7%
0.25	88.6%	89.4%	89.1%	82.1%	82.3%	85.6%	61.9%	60.3%	67.1%
0.30	91.2%	92.0%	91.6%	86.2%	86.6%	88.9%	70.7%	65.9%	74.9%
0.40	95.9%	95.6%	95.3%	93.5%	94.3%	94.7%	85.6%	81.8%	85.5%
0.50	98.9%	98.7%	98.1%	98.2%	98.2%	98.2%	96.2%	92.7%	93.1%
0.60	99.7%	99.7%	99.3%	99.5%	99.1%	99.2%	98.5%	95.9%	96.1%
0.70	100.0%	100.0%	99.9%	100.0%	99.6%	99.7%	100.0%	98.5%	98.2%
0.80	100.0%	100.0%	100.0%	100.0%	99.9%	99.9%	100.0%	99.9%	99.8%

**Notes:** The table shows the cumulative distribution of frailty for three age groups, in waves 1, 5, and 9 of ELSA. The table is read in columns, such that each number represents the share of individuals with a frailty index value up to the level noted in the leftmost column.



**Fig. 1.** Determinants and speed of frailty.

**Notes:** Coefficients shown as dots, 95% confidence intervals shown as lines. The left panel shows coefficients from estimating Eq. (1). The right panel shows the coefficient on age from fixed-effects regressions as in Eq. (2), run for the full sample and separately by sex, education, and wealth.

wealth are likely correlated with unobserved individual characteristics that influence the speed of ageing, we modify Eq. (1) to:

$$\ln F_{iwt} = \alpha_i + \beta \cdot \text{age}_{iwt} + \gamma_1 X_{1iwt} + \epsilon_{iwt} \quad (2)$$

where  $\alpha_i$  is now an individual-specific fixed effect. The time-invariant controls  $X_{2iwt}$  cannot be included in this fixed-effects regression so we estimate this regression for the full sample and then separately by sex, education level, and the wealth tertile of the individual based on their first appearance in the sample.

For the full sample, we estimate an age-related frailty accumulation of 3.9% per year. This rate of ageing is similar to that found for a range of European countries (Abeliansky and Strulik, 2018, 2019), but less than the 5% rate estimated for the U.S (Abeliansky et al., 2020). Comparing coefficients in Fig. 1(b) shows that, in line with previous studies, males accumulate health deficits faster than females (4.3% per year vs. 3.6% per year). Given that females tend to have higher levels of initial frailty, this points towards health converging at later ages.<sup>2</sup> In addition, individuals with higher education see their frailty levels rise more slowly than individuals with low levels of education (3.3% per year vs. 3.8% and 4.2%). We find no statistical evidence of the rate of ageing varying across wealth tertiles although Appendix Table B.1 shows that within each tertile the level of frailty is diminishing with wealth.

<sup>2</sup> Using these fixed effects estimates we estimate male and female frailty to converge at a rather distant 109 years, broadly in line with the age of convergence estimated by Abeliansky et al. (2020) for the US.

Combined these effects shows marked inequalities across England in terms of frailty. An example of these inequalities is visualised in Fig. 2, which plots the fit of a local polynomial regression of frailty on age based on the latest three waves of ELSA, along with 95% confidence bands. The figure compares individuals in the North East in the lowest tertile of wealth who left school at 16, compared with college graduates in the South East in the highest wealth tertile. A wide gap opens up from 50 years onwards, this gap remains large and broadly constant between 60 and 70 years of age before starting to narrow at oldest ages. A similar pattern of health inequities has been found for the US (Case and Deaton, 2005), where health status smoothly deteriorates for the richest income quartile, whereas, for the poorest quartile, it increases rapidly until retirement, after which the deterioration of health flattens off. Recently, Abeliansky and Strulik (2023) used data across Europe and found large positive impacts of retirement on health for workers in low-status occupations, and negligible impacts for high-status occupations. The pattern in Fig. 2 is consistent with these patterns, and suggests a partial “catch-up” in frailty for high-SES individuals at later ages.

*Are cohorts ageing better?*

Table 2 characterises ageing across all nine waves of ELSA, but a key question is whether there have been improvements across cohorts in the rate at which frailty accumulates. There is evidence that this has happened for the US (Levine and Crimmins, 2018; Abeliansky et al., 2020) and Europe (Abeliansky and Strulik, 2018, 2019), and we

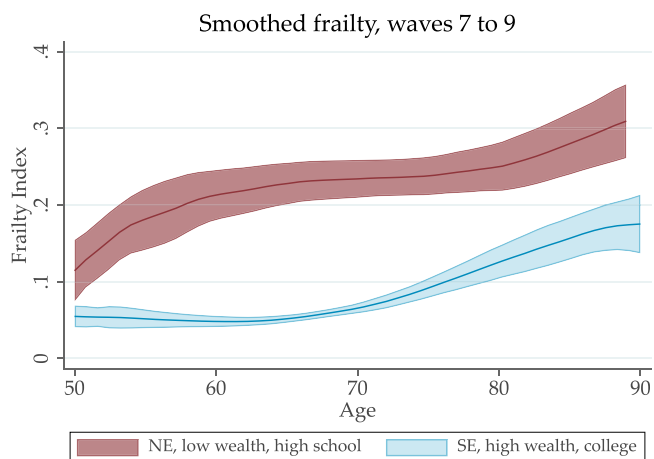


Fig. 2. Frailty accumulation: smoothed local regression fit with 95% confidence bands, using Epanechnikov kernel with bandwidth = 4.

now investigate whether the same occurs for England. Other studies, including on ELSA, have found evidence of frailty deteriorating across cohorts (Marshall et al., 2015; Yang and Lee, 2010; Stephan et al., 2020).

To estimate cohort effects, we modify Eq. (1) by including a linear year of birth trend to capture age-specific improvements in frailty over time. Identifying cohort effects requires allowing for various unobserved characteristics. To do so, we follow Abeliansky et al. (2020) and use three different estimators — pooled OLS, random effects regression, and Mundlak estimation (Mundlak, 1978).

Since the year of birth does not vary over time for individuals, standard fixed-effects models to account for individual heterogeneity cannot be used when estimating cohort effects. Hence, it is sensible to use random effects. However, if there is individual heterogeneity that is systematically related to both frailty and cohort, random effects would be inconsistent. We therefore follow Mundlak (1978) and add the mean of all time-varying covariates as additional regressors (Mundlak, 1978; Wooldridge, 2021). In particular, we include the mean age of individual  $i$  as a control variable. If this variable is significant (as it is in our analysis) then systematic individual heterogeneity is present and these Mundlak estimates are the most reliable.<sup>3</sup> For purposes of robustness we also show results with other estimators. Reassuringly our main results are repeated across all estimation methods but the significance of the mean age variable means the Mundlak ones are the focus of our attention in the discussion below.

Table 3 shows our estimation results, where columns are separate regressions estimated with pooled OLS, random effects, and Mundlak estimation, with and without controls, respectively. There is strong evidence of a cohort effect whereby frailty is improving over time, indicated by the negative and significant coefficients on the year of birth. Focusing on the Mundlak estimates with a full set of controls (column 6), being born one year later for a given age goes along with a roughly six month improvement ( $0.019/0.038 \times 12 = 5.89$  months, 95% CI: [4.65 – 7.13]) in frailty. That is about twice the rate of improvement previously found for the U.S (Abeliansky et al., 2020). To summarise the cohort effects, we calculate the age at which an individual in 2018 is expected to have the same level of frailty as a 70-year-old individual in 2002. This is shown in the last row of Table 3, which indicates that 75 (in 2018) is the new 70 (in 2002) (Levine and Crimmins, 2018).

<sup>3</sup> Following Bell and Jones (2015), we demean age at the individual level in these regressions to facilitate interpretation of the mean age coefficient. Neither our findings of significant Mundlak terms nor the predicted cohort trends are sensitive to this.

In finding evidence of cohort improvements our results differ from Marshall et al. (2015), who use the first five waves of ELSA and a multilevel growth model for frailty. Given we still find evidence of cohort improvements when we restrict our estimation to the first five waves, the variance in results must reflect other specification differences such as their use of a linear model, five-year age intervals and inclusion of a quadratic term in age. Including the quadratic term does not change our results and neither does specifying our model in linear terms (with our logarithmic specification preferred statistically). Attempting to replicate their methods but using logarithms and one year age intervals gives weaker evidence for cohort improvements. Another important difference in our study is the use of the Mundlak terms in estimation. Given the theoretical argument for their inclusion and their significance in our estimation, this points to the importance of allowing for systematic heterogeneity. Reassuringly though the plausibility of cohort improvements does not rest solely on selection of a particular estimator based on the inferred presence of unobserved heterogeneity. As shown in Appendix B.2, raw data from various waves also shows clear and simple support for the notion of improving frailty across cohorts. In addition, Appendix Table B.4 presents a series of robustness checks showing that the results are not driven by details on the construction of the frailty index, sample selection issues, and missingness patterns in the data.

Table 3 assumes a constant rate of cohort improvement. To investigate whether this rate varies over time, we include a quadratic term in year of birth in the analysis, summarised in Table 4 and visualised in Fig. 3. All specifications include the full set of control variables. Both coefficients in the quadratic expression are statistically significant, suggesting that later cohorts are ageing better in terms of frailty than past cohorts and that the rate of improvement is slowing. Using the historical estimates from this quadratic specification, a 75.5-year-old in 2018 (born in 1943) has the frailty of a 70-year-old in 2002 (born in 1932) (shown in column 3). However, we can use our model to project forward frailty for future cohorts. The projections for the next sixteen years, the same duration as in our data, are shown in the bottom row of Table 4 and visualised in Fig. 3. Based on these projections, a 74.5-year-old in 2034 is expected to have the frailty of a 70-year-old in 2018. Thus, frailty improvements are slowing at a relatively modest rate (from a five-and-a-half year improvement over 16 years to a four-and-a-half year improvement). Further evidence supporting this conclusion is shown in Appendix Figure B.1, which shows estimated values for the year of birth effect when a full set of year of birth dummies is included in the estimation rather than simply relying on linear or quadratic trends.

#### What is driving the improvement?

To identify the proximate drivers of these cohort improvements, we follow Rogers et al. (2017) and separate the frailty index into five mutually exclusive domains: Mobility difficulties (e.g., problems walking, or lifting objects), ADL/IADLs (e.g., difficulties getting dressed or making phone calls), depressive symptoms, self-reported health conditions (e.g., whether a respondent had or has had cancer or diabetes), and cognitive function (e.g., word recall)<sup>4</sup>

Table 5 shows the results from Mundlak regressions with controls for these five domains estimated separately. Because we use the log of each component (to echo the specification for the overall frailty index) the number of observations varies across domains as we need to exclude all zero observations. As a consequence, Table 5 only provides evidence on the intensive rather than extensive margin, i.e. variations in the severity of conditions rather than changes in who has the conditions. For robustness purposes we did estimate results using the transform  $\ln(1+frailty)$  as our dependent variable and found essentially unchanged results (see Appendix Table B.5).

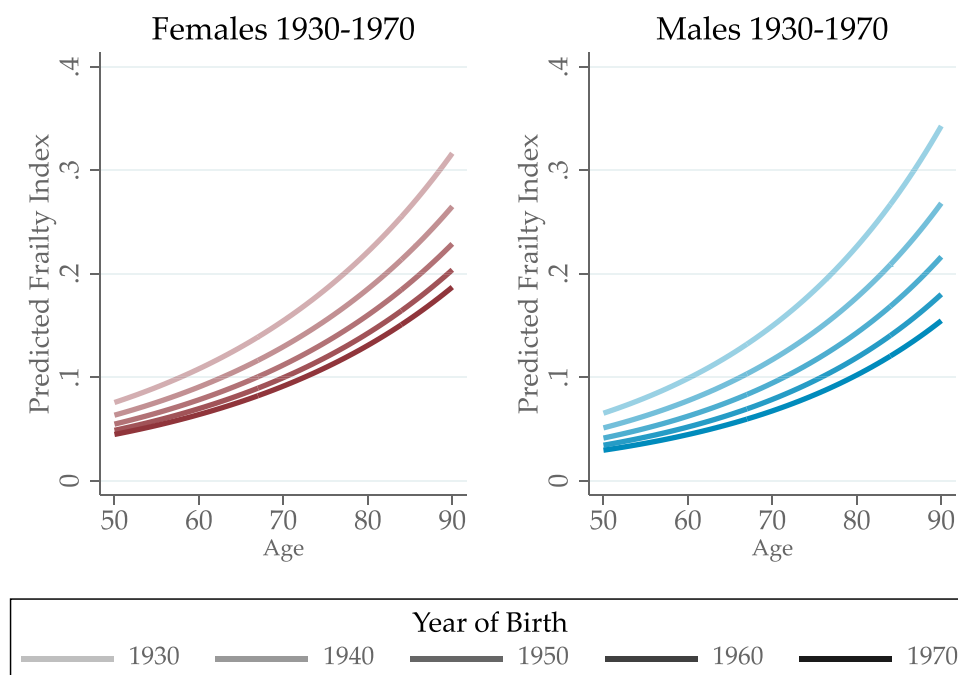
<sup>4</sup> We drop one item from our analysis at this stage: self-reported general health status as it is not included in the sub-indices leaving us with 49 components).



**Table 3**  
Estimating birth cohort trends in frailty.

	(1)	(2)	(3)	(4)	(5)	(6)
Age	0.033*** (0.003)	0.036*** (0.002)	0.038*** (0.003)	0.039*** (0.003)	0.038*** (0.003)	0.038*** (0.003)
Year of birth	-0.026*** (0.001)	-0.023*** (0.001)	-0.028*** (0.001)	-0.025*** (0.001)	-0.029*** (0.003)	-0.019*** (0.002)
Mean Age					-0.002 (0.003)	0.008*** (0.002)
Observations	72,705	65,694	72,705	65,694	72,705	65,694
Sample	All	All	All	All	All	All
Method	P-OLS	P-OLS	RE	RE	Mundlak	Mundlak
Controls	None	All	None	All	None	All
What is the new 70?	77.1	76.3	76.7	76.2	76.9	75.3

**Notes:** Robust standard errors clustered at year of birth level in parentheses. Significance levels: \*10%, \*\*5%, \*\*\*1%. Age is demeaned at individual level. Controls: Sex, NUTS1 region dummies (+ mean for Mundlak specifications), education, log wealth (+ mean for Mundlak specifications). The last row displays the predicted age in 2018 in which frailty equals the frailty level of a 70-year-old in 2002.



**Fig. 3.** Fitted changes in frailty profiles across birth cohorts, specifications with squared year of birth term.

**Table 4**  
Frailty trends: With quadratic in year of birth.

	(1)	(2)	(3)
Age	0.037*** (0.002)	0.039*** (0.003)	0.038*** (0.003)
Year of birth	-0.857*** (0.175)	-1.018*** (0.192)	-0.625*** (0.171)
(Year of Birth <sup>2</sup> )/1000	0.215*** (0.045)	0.256*** (0.049)	0.156*** (0.044)
Observations	65,694	65,694	65,694
Sample	All	All	All
Method	P-OLS	RE	Mundlak
What is the new 70 in 2018?	76.5	76.4	75.5
What is the new 70 in 2034?	75.2	74.9	74.5

**Notes:** Robust standard errors clustered at year of birth level in parentheses. Significance levels: \*10%, \*\*5%, \*\*\*1%. Age is demeaned at individual level. Controls: Sex, NUTS1 region dummies (+ mean for Mundlak specifications), education, log wealth (+ mean for Mundlak specifications).

The last two rows use the regression results to calculate the age in 2018 of a person with the same projected frailty level as a 70 year old in 2002 and the age in 2034 of a person with the same projected level of frailty as a 70 year old in 2018.

With the exception of the depression component, frailty in each domain rises with age in a statistically significant manner, albeit at differing rates. There is also evidence for improvements across cohorts in all domains. For the mobility, ADL/IADL, and cognitive components, the cohort effect is substantial at around eight to nine months per year. However, the evidence for improvements in underlying physical conditions is weak and numerically very small.

Recent research has found that the evidence on improving frailty in Europe may be driven by sample selectivity bias (Börsch-Supan et al., 2021). Reassuringly, the declining frailty trend in our data is not due to any one dimension, and the trend in our data is no different for the dimensions with non-trivial shares of missing data, as visualised in Appendix Figure A.2.

*Which demographic groups have seen the most progress?*

To investigate whether these cohort trends differ across groups we ran separate Mundlak regressions (with control variables) for different demographic characteristics. Numerical estimates suggest that males have experienced more rapid improvements than females although a Wald test finds the difference in trend to be not statistically significant.

**Table 5**  
Cohort trends by sub-components of frailty index.

	(1) Mobility (10)	(2) ADL/IADL (13)	(3) Depression (8)	(4) Conditions (12)	(5) Cognitive (6)
Age	0.026*** (0.002)	0.030*** (0.003)	0.002 (0.001)	0.039*** (0.001)	0.018*** (0.002)
Year of birth	-0.017*** (0.002)	-0.024*** (0.003)	-0.012*** (0.001)	-0.002* (0.001)	-0.014*** (0.002)
Mean Age	-0.007*** (0.002)	-0.020*** (0.003)	-0.011*** (0.001)	0.007*** (0.002)	-0.003* (0.002)
Observations	39,044	18,565	38,285	53,916	24,870
Mean of DV	0.351	0.234	0.321	0.170	0.279

**Notes:** All columns show Mundlak regressions. Robust standard errors clustered at year of birth level in parentheses. Significance levels: \*10%, \*\*5%, \*\*\*1%. Age is demeaned at individual level. Controls: Sex, NUTS1 region dummies (+ mean), education, log wealth (+ mean). Number of items in each sub-component listed in parentheses in the column headers.

**Table 6**  
Longevity trends: Heterogeneous effects.

	Gender		Education			Baseline wealth		
	(1) Males	(2) Females	(3) Low	(4) Middle	(5) High	(6) Low	(7) Middle	(8) High
Age	0.041*** (0.003)	0.036*** (0.003)	0.042*** (0.003)	0.038*** (0.003)	0.033*** (0.003)	0.036*** (0.002)	0.040*** (0.003)	0.038*** (0.003)
Year of birth	-0.022*** (0.003)	-0.016*** (0.002)	-0.016*** (0.003)	-0.022*** (0.003)	-0.012*** (0.005)	-0.010*** (0.004)	-0.020*** (0.003)	-0.025*** (0.003)
Mean Age	0.004 (0.003)	0.012*** (0.003)	0.009*** (0.004)	0.005 (0.003)	0.021*** (0.005)	0.010** (0.005)	0.009*** (0.004)	0.007* (0.004)
Observations	30,046	35,648	23,530	31,953	10,211	20,934	21,754	23,006
Method	Mundlak	Mundlak	Mundlak	Mundlak	Mundlak	Mundlak	Mundlak	Mundlak
What is the new 70?	75.6	74.8	74.5	75.9	74.3	73.6	75.4	76.3
p-value Interaction term	0.083		0.05			<0.001		

**Notes:** All columns show Mundlak regressions. Robust standard errors clustered at year of birth level in parentheses. Significance levels: \*10%, \*\*5%, \*\*\*1%. Age is demeaned at individual level.

Education levels are: Low: below upper secondary, Middle: upper secondary/vocational training, High: tertiary education. Baseline wealth is split in tertiles relative to five-year age group in the first occurrence in the survey. Controls: Sex, NUTS1 region dummies (+ mean), education, log wealth (+ mean). In the specifications for sex and education, the respective variable is not included as a control.

The last row is from a joint specification in which year of birth was interacted with all levels of the respective variable. The p-value is from a Wald-test on the interaction terms of year of birth and the respective variable levels, with the null hypothesis that the interaction terms are jointly zero.

Whilst higher levels of education are associated with slower rates of frailty accumulation, **Table 6** suggests that it is lower levels of education that have seen the fastest rates of improvements although the effect is relatively small and of only borderline statistical significance. Conversely, whilst we found that across wealth tertiles the rate of ageing was broadly similar when we allow for cohort differences we find strong evidence of faster rates of improvements for those in the highest wealth tertile. In Appendix Table B.6, we repeat the same specification and include quadratic terms in year of birth, which point to some degree of convergence, as groups with lower levels of frailty and better trends see a larger slowdown in the year-of-birth effects.

*Individual and regional frailty dynamics*

We can use our frailty index and its sub-components to identify the major contributors to frailty at each age and the most important causes of deterioration between different ages. To do so **Table 7** shows two statistics for each sub-component (defined over different numbers of items, listed in parentheses in the column heading): the median (P50), and the mean of the highest frailty quartile in each sub-component (HFQ). The largest single contributor to median frailty for people in their 50s and 60s (both weighting each sub-component by the number of items it contains and also in unweighted terms) is depression (including restless sleep, feeling depressed/sad, feeling not being able to get going, etc.). Note though that depression does not increase with age but is simply a substantial contributor to frailty at all ages.<sup>5</sup> In moving

from the 50s age group into the 60s, declines in mobility are the most important contributor to increases in overall frailty, and in transiting from the 60s to the 70s, increased incidence of disease conditions is the most important factor in explaining increased median frailty. In the 70s, these disease conditions are the largest single component contributing to the level of median frailty and in the 80s that role is taken by mobility restrictions. Mobility restrictions are also the main cause of frailty deteriorating in the 70s, followed by an increase in cognitive problems.

**Table 2** showed that focusing on median outcomes omits important distributional details, so **Table 7** also shows evidence around the upper tail of frailty by showing the average value amongst the quartile with the highest level of frailty for each domain (columns labelled HFQ). This is a way of capturing which components of frailty are explaining the worst outcomes at each age. For those in their 50s and 60s, depression is highest for the lower frailty quartile. Mobility restrictions is another important factor as well as the dominant driver of frailty in the bottom quartile of the distribution in the 70s and 80s.

To understand the drivers of regional inequalities, **Fig. 4** shows the coefficients on the regional dummies in the Mundlak regressions by subcomponents. The South East of England is the designated reference group (as it has the lowest level of average frailty across all regions). The coefficients point to systematic regional differences across a variety of factors. Cross-regional inequalities in mobility and ADL/IADL are most pronounced, with lesser variation amongst disease

<sup>5</sup> The fact that in this case we find no strong evidence that depression increases with age raises the interesting issue of whether it should be included

in a frailty index given the methodology of [Searle et al. \(2008\)](#) and the emphasis on selecting components that increase with age

**Table 7**  
Median (P50) and average of highest frailty quartile (HFQ) of frailty sub-components and overall frailty index, by age group.

Age Group	Mobility (10)		ADL(13)		Depression (8)		Conditions (12)		Cognitive (6)		Overall Index	
	P50	HFQ	P50	HFQ	P50	HFQ	P50	HFQ	P50	HFQ	P50	HFQ
50–59	0	0.435	0	0.038	0.125	0.492	0.083	0.21	0	0.054	0.06	0.242
60–69	0.1	0.436	0	0.045	0.125	0.464	0.083	0.224	0	0.243	0.08	0.283
70–79	0.1	0.605	0	0.216	0.125	0.447	0.167	0.301	0	0.285	0.111	0.338
80–89	0.3	0.744	0.077	0.437	0.125	0.564	0.167	0.304	0.167	0.465	0.18	0.447

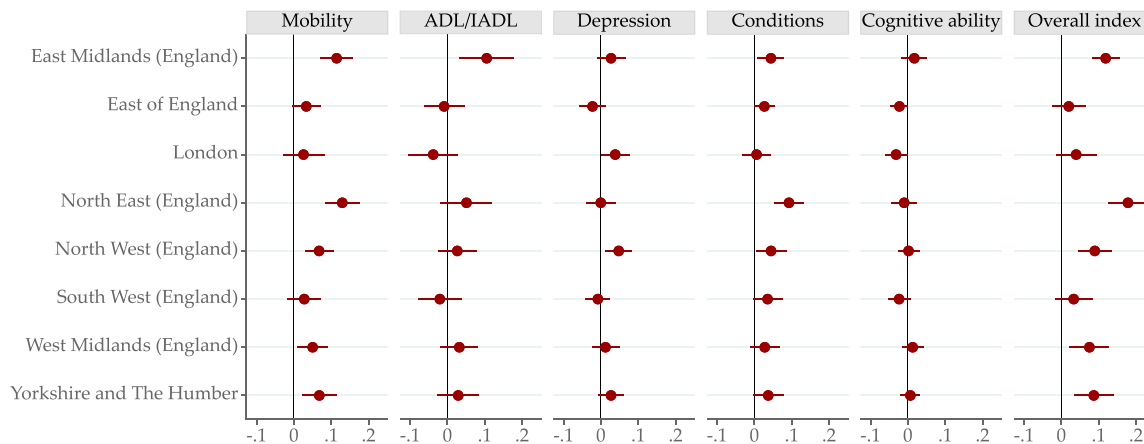


Fig. 4. Coefficients on regional dummies from Mundlak regressions.

conditions (aside from the North East) and relatively small differences in depression and cognitive abilities.

Whilst Fig. 4 points to differences in regional averages across various frailty components, closer inspection reveals that the major drivers of these regional differences are within-region effects. In support of this argument, Fig. 5 presents for different age groups the distribution of frailty for the top three regions compared with the bottom three regions. These show relatively small differences in modes and medians, but what leads to better average outcomes in the best regions are much larger tails at low levels of frailty and lower tails at high levels of frailty. In other words, the better regions are substantially better in terms of the best and worst outcomes but only moderately more successful in terms of median outcomes.

**Conclusion**

Using nine waves of ELSA from 2002 to 2018 we find : i) frailty rises at an approximate rate of 3% to 4% per annum, similar to many EU countries but at a slower rate than the U.S ii) there are stark differences in frailty based on sex, region, education and wealth such that those with better education and wealth have substantially lower frailty at older ages, iii) frailty at each age has been improving over time but there is evidence the rate of improvement may be slowing albeit modestly iv) the rate of improvement varies significantly across different groups and has been highest for those with the highest levels of wealth v) improvements in frailty have been driven mostly by improvements in mobility and ADLs but very little by reductions in underlying disease conditions vi) depression is the largest component of frailty in the 50s and 60s but mobility is most important in explaining deteriorating frailty in the 50s, disease conditions in the 60s and mobility again in the 70s, and (vii) differences in mobility and ADLs are most important in explaining differences in regional averages but regional variations are mainly due to greater dispersion in health within rather than across regions.

Our results provide some good news in terms of health trends in England, especially around the substantial cohort improvement trends we estimate. However, our results also explain why a number of studies express concern over recent health trends in the U.K. Whilst there

have been large improvements in the lower tail of frailty, there has also been an increase in the upper tail. The former dominates the latter, leading to average improvements but both in terms of levels and trend improvements there are significant differences in frailty based on education and wealth.

Our results cover frailty trends before Covid-19 and so shed no light on the impact of the pandemic on frailty amongst the English population aged over 50 years. Both the immediate and long term impact of Covid-19 on older people’s health is likely to be substantial and may significantly affect the trends documented here. Our study also only documents trends and so provides no insight as to the measures that have led to reductions in frailty and neither does it identify steps to achieve further reductions or tackle the profound differences in frailty by region and education. Further, whilst frailty measures are a convenient way to capture health and functionality of older adults they fail to capture the intrinsic capacity that individuals can draw on or how they are affected by these health deficits. If different frailty components have differential impacts on individual’s depending on their education or wealth then our results based on equal weighted frailty components will tell an incomplete story.

Our results have a number of implications. Firstly they emphasise the potential malleability of how health deficits accumulate over time. That malleability manifests itself in two ways. The first is the clear impact of socio-economic variables such as education and wealth in influencing frailty. The second is in the trend improvements across cohorts we find consistently and robustly across a wide range of specifications. Given the increasing importance of ageing well as people live longer (Goldman et al., 2013; Scott et al., 2021), this finding suggests that much is at stake and outcomes can be affected. The need to exploit this malleability is made all the greater by the evidence this paper reveals regarding the slowdown in these trends and substantial inequalities. Finally our breakdown of regional differences suggests policies to narrow these should focus on achieving greater improvements in mobility and ADLs amongst poorer performing regions and tackling within region inequality. Finally the fact that our results suggest there have been only limited improvements in the incidence of age-related diseases and conditions points to the importance of better understanding the underlying biology of these diseases and the development of potential therapeutics (Campisi et al., 2019).



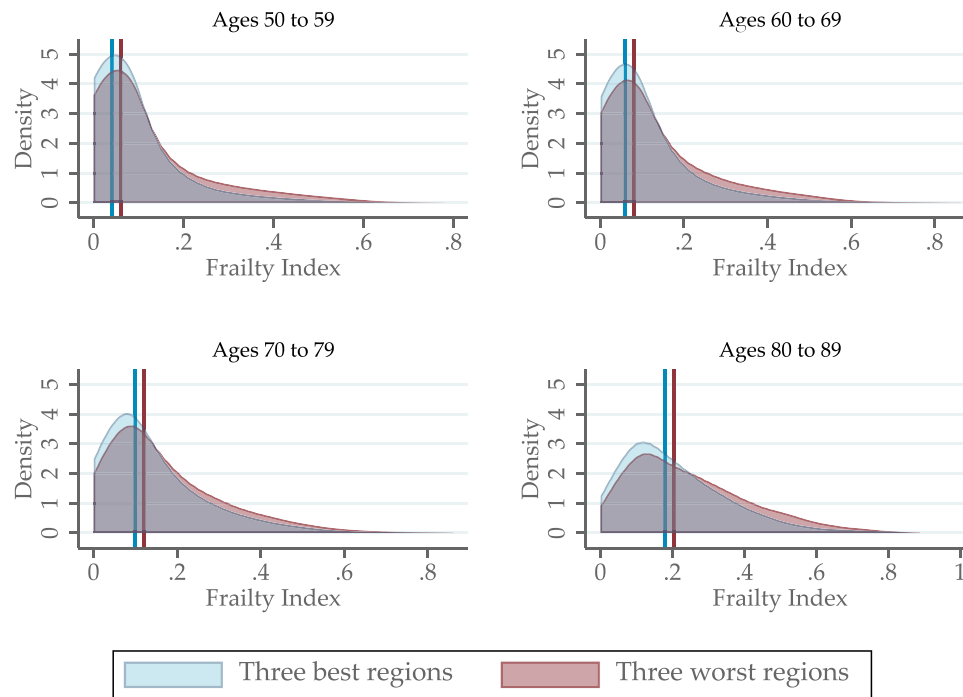


Fig. 5. Distribution of frailty: Comparing high and low frailty regions. Epanechnikov kernel density estimates, vertical lines show medians.

**CRedit authorship contribution statement**

**Jonathan Old:** Methodology, Data curation, Software, Formal analysis, Writing – original draft, Writing – review & editing. **Andrew Scott:** Conceptualization, Writing – original draft, Writing – review & editing, Supervision, Project administration, Funding acquisition.

**Declaration of competing interest**

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Andrew Scott is a co-founder of the not-for-profit "The Longevity Forum". He is an Associate Editor of the journal and received a financial contribution from Elsevier for a conference he arranged titled "The Economics of Longevity", which was published as a special issue in this journal. Jonathan Old has no conflicts of interest.

**Appendix A. Supplementary data**

Supplementary material related to this article can be found online at <https://doi.org/10.1016/j.jeoa.2023.100470>.

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