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A unified framework to account for selective mortality in lifecycle analyses of the social gradient in health

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Abstract

This paper establishes a unified framework to fully account for the changing social gradient over the lifecycle in terms of a sufficient set of mobility indices characterizing the coevolution of socioeconomic status and health within each of a series of overlapping cohorts. We proceed to demonstrate the impact of selective mortality on health inequality changes, making use of a counterfactual health distribution for the start of the study period that leaves out those who are known to die before the end. Specifically, initial differences between the average health and educational attainment rank of survivors and nonsurvivors are found in our empirical study to be an increasingly important factor explaining changes in the education-health gradient in older cohorts in Great Britain. Our identification strategy has the advantage that it does not require the imputation of the “would be” health of nonsurvivors, which if carried out using inverse probability weighting procedures—as in several previous studies—is shown to bias estimates of this direct effect of selective mortality toward zero. Parallel results for the income-health gradient exhibit stronger confounding influences due to a number of other factors given that income is a less stable indicator of socioeconomic status in adulthood.

KEYWORDS

Great Britain, lifecycle, mobility analysis, selective mortality, social gradient

1 | INTRODUCTION

The lifecycle literature on the social gradient in health has been dominated by the cumulative (dis)advantage and age-as-leveler hypotheses (Siegel & Allanson, 2016). The former posits that social gradients develop in early life and become stronger as socioeconomic and health disadvantages accumulate over the lifecycle, while the latter maintains that health inequalities narrow in older age groups as changes in health become more closely associated with age than socioeconomic status (SES). In combination, these two mechanisms may serve to explain the stylized fact that health inequalities in adulthood widen through middle and early old age and then diminish in later old age (Deaton & Paxson, 1998; Siegel & Mosler, 2014; Smith, 2007), with selective mortality also recognized as a potentially important confounder in older cohorts if only the more robust in lower SES groups survive (Dupre, 2007). van Kippersluis et al. (2010, p. 436)

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conclude that there is unlikely to be a “single explanation for the observed lifecycle pattern of the social gradient” but rather “many mechanisms interacting.”

However, if this is the case then a notable weakness of the literature is the lack of a unified framework to identify all the channels through which these mechanisms may influence the coevolution of SES and health over the lifecycle and quantify their contribution to changes in (SES-related) health inequality. We would argue that this shortcoming has resulted in a series of assessments of the relative importance of specific mechanisms, resulting in evidence that is often implicit in nature and missing key aspects. In particular, neither Beckett (2000) nor Herd (2006) nor Baeten et al. (2013) explicitly evaluate the impact of selective mortality on health inequality, relying on indirect evidence instead to suggest that it can only play a minor role. The main objective of this paper is to fill this gap by fully accounting for the changing social gradient at different ages in terms of a sufficient set of mobility indices characterizing the coevolution of SES and health.

For this purpose, we propose a procedure to identify the impact of individual SES changes, morbidity changes, and deaths on health inequality within each of a series of overlapping cohorts, building on the set of decomposition methods developed in Allanson and Petrie (2013a) to analyze such changes at the population level using longitudinal data. A particular attraction of our approach is the clear demonstration of how the changing association between SES and health on the one hand and death on the other affects the impact of selective mortality on the evolution of health inequality over the lifecycle. The decomposition procedure further allows for the potentially bidirectional relationship between SES and health, conditional upon survival, facilitating the interpretation of the results from a Granger causality perspective.

To help fix ideas about the possible impact of selective mortality, consider a simple example based on a population of whom half are high SES and relatively healthy and the other half low SES and relatively unhealthy. We consider two scenarios of possible health changes over some time interval. In Scenario 1, all the high SES individuals retain their SES status but half of them experience a positive health change and the other half an equal but negative health change. Similarly, the low SES individuals keep their SES status but half experience a positive health change and the other half an equal but negative health change. Health inequality will be the same at the end of the interval since the average health change is zero for both high and low SES individuals. In Scenario 2, the only difference is that the low SES individuals who experience the negative health shock are all now assumed to die as a result. The observed health inequality in the extant population will clearly be lower at the end of the interval due to the “direct” effect of the loss of half the unhealthy low-SES individuals from the initial population. Moreover, as an “indirect” effect of these deaths, health changes among the surviving population will now appear to be progressive or “pro-poor” since the average health change of the low SES individuals conditional on survival will be positive. We consider below how these “direct” and “indirect” effects of selective mortality relate to established explanations of changes in cross-sectional health inequality within older cohorts.

The most straightforward interpretation of the selective mortality hypothesis rests on the proposition that the people who die over some time interval will have been on average both of lower SES and less healthy at the start of the interval than those who survive, all else equal, where this is the case in Scenario 2 of our illustrative example and more generally holds true (see, e.g., Herd, 2006). However, this between-group effect—between nonsurvivors and survivors—is not the only one that must be taken into account when considering the direct impact of selective mortality on inequality. In particular, the change in cross-sectional health inequality will also be affected by any difference in the levels of health inequality within the nonsurvivor and survivor groups at the start of the interval, where in Scenario 2, this within-group difference effect will have partially offset the fall in health inequality due to the between-group effect because the nonsurvivors were all equally low-SES and unhealthy at the start of the interval unlike the heterogeneous survivor group. Moreover, rank-dependent health inequality measures will be sensitive to the difference between the relative positions of survivors at the start of the interval in the distributions of SES defined over the full and survivor populations. It follows that the direct effect may not be negative even if non-survivors are on average of lower SES and less healthy than survivors.

According to the age-as-leveler hypothesis, the attenuation of health inequality in older cohorts occurs because socioeconomic advantages can only delay not prevent morbidity (House et al., 2005), with age increasingly dominant as a determinant of health in later life (Lynch, 2003). In Scenario 2 of our example, we observe a convergence in health trajectories between the rich and poor conditional upon survival, but as previously noted this is only because of the indirect effect of selective mortality. Mirowsky and Ross (2008) report findings consistent with the existence of such convergence due to selective mortality, dismissing its importance “when combining the full range of adult ages” (p. 112) but noting “the possibility of significant compression or selection effects in [...] much older samples” (p. 114). Importantly, the existence of such indirect selection effects will be a source of bias in methods, such as those used by Beckett (2000) and Baeten et al. (2013), that seek to estimate the direct effect of selective mortality on health inequality by imputing the “would be” health (cf. Lynch, 2003) of nonsurvivors at the end of the interval based on the observed health outcomes of

survivors who had similar characteristics to them at the start. In Scenario 2, this approach would imply assigning nonsurvivors the end of interval health of low-SES survivors, but this counterfactual prediction is higher even than the average final health of the low-SES group in Scenario 1. More generally, it seems likely to generate upwardly biased estimates of health under most reasonable assumptions (Noymer, 2001). For example, we present results in this paper that imply that average health changes among survivors of similar age, SES, and health as nonsurvivors at the start of the study period are consistently more positive/less negative than among all survivors. Thus, imputation methods based on the observed outcomes of survivors are likely to underestimate the size of the direct effect of selective mortality, where it may be noted that this argument does not rest on the proposition that the actual health outcome of non-survivors, that is death, is manifestly worse than any health state experienced by those whom survive (cf. Petrie et al., 2011).

The next section of the paper sets out our decomposition methodology, which serves to identify both the direct effects of selective mortality and the contributions of health and SES changes to health inequality changes among survivors. Our approach has the advantage of not requiring the imputation of the “would be” health of nonsurvivors, although this is necessary if one wishes to put bounds on the likely size of the indirect effect of selective mortality. Section 3 introduces the empirical study with the results presented in the following section (Section 4). We use longitudinal data for Great Britain (GB), as an empirical example, to explore the dynamics of SES and health and, more specifically, to demonstrate that selective mortality can be an important driver of changes in cross-sectional health inequality over the lifecycle, contrary to the findings of many previous studies. Finally, Section 5 concludes this paper.

2 | ACCOUNTING FOR CHANGES IN HEALTH INEQUALITY BY AGE GROUP USING LONGITUDINAL DATA

This section proposes a procedure to decompose the change in health inequality within an age/birth cohort over some time interval marked by a start and an end date. The first stage of the decomposition serves to separate out the direct effect of selective mortality from the net effect of morbidity and SES changes, where the order in which these two effects are taken into account in the decomposition may be expected to affect the results given the familiar path dependency problem (see, e.g., Lerman & Yitzhaki, 1985).¹ Figure 1 illustrates this choice. The conventional approach (illustrated by the shorter dashes) has been to consider what the joint distribution of SES and health might have been at the end date if there had been no deaths during the time interval (e.g., Baeten et al., 2013; Beckett, 2000; Lynch, 2003; Herd, 2006; Mirowsky & Ross, 2008). However, the construction of this end date counterfactual requires the imputation of the “would be” health (and SES) of nonsurvivors which either is likely to lead to underestimates of the direct effect of selective mortality if based on the health outcomes of survivors, as argued above, or require data on the health of nonsurvivors prior to the start date if based on the extrapolation of individual health trajectories from some preceding time interval. The equally valid alternative (illustrated by the longer dashes in Figure 1) is to first consider what the joint distribution might have been at the start date in the absence of those who are known to die during the time interval. This approach is straightforward to implement if the counterfactual is simply taken to be the joint distribution of the survivor group at the start date on the assumption that this is independent of the existence of the non-survivor group.² We focus on this alternative approach but present decompositions based on both start and end date counterfactuals so as to permit an empirical assessment of the implications of the choice.

We illustrate our proposed decomposition procedures using the Erreygers index (Erreygers, 2009) to measure health inequality and with health standardized (in the sense of Erreygers & Van Ourti, 2011) to lie between zero and one. The Erreygers index assumes an equal absolute loss (or gain) of health for everyone will leave inequality unchanged and is equal to one when the richest 50% of the population are in the best possible health and the poorest 50% of the population in the worst possible health. Similar methods could also be used to examine health inequality changes from a relative perspective in either attainments or shortfalls (see Allanson & Petrie, 2013b and Kjellsson et al., 2015), but the resultant decompositions would be more unwieldy as changes in relative health inequality will depend on changes in both the absolute dispersion and mean level of health.

2.1 | Decomposition procedure based on start date counterfactual

We consider the change in cross-sectional health inequality between some start date s and an end date f in an age cohort that is only subject to change due to deaths.³ The initial population of the cohort is $\Omega_s = \Omega_{CO}$ of whom a subgroup

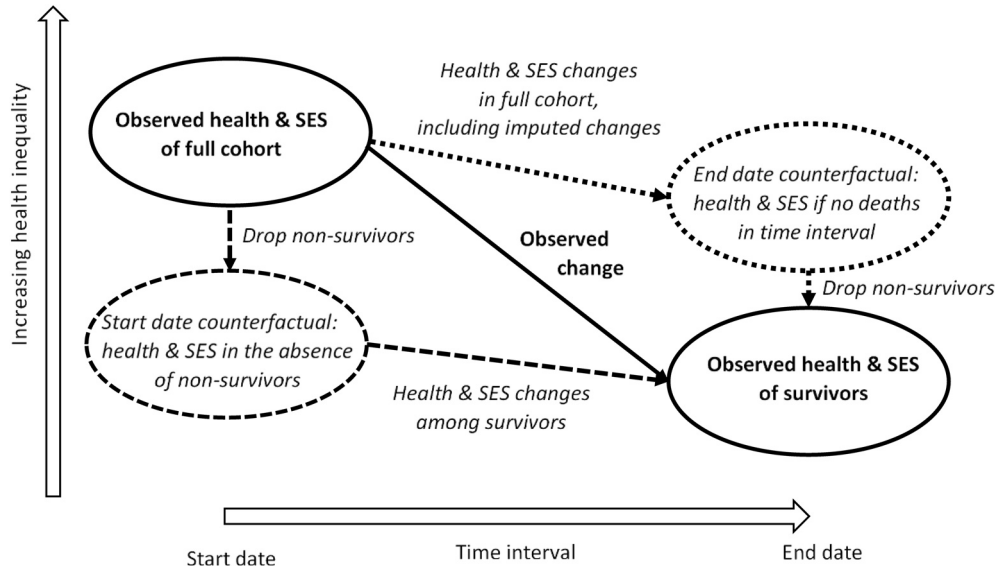


FIGURE 1 Illustration of choice of decomposition pathway

of nonsurvivors Ω_{NS} die during the time interval to leave the population of survivors $\Omega_f = \Omega_{SU}$ at the end date. Let $EI_t = 8\text{cov}(\Omega_t, h_t, F_{\Omega_t}(y_t))$ denote the Erreygers index of the cohort at date t ($t = s, f$), which is given for the population of interest Ω_t as eight times the covariance between health h_t and the cumulative distribution function or fractional rank of SES $F_{\Omega_t}(y_t) = P(y_t < y)$ within Ω_t . Finally, let $EI_s^g = 8\text{cov}(\Omega_g, h_s, F_{CO}(y_s))$ be the within-group Erreygers index of group g ($g = SU, NS$) at the start date, which is defined over the relevant subgroup Ω_g but is based on fractional SES ranks $F_{\Omega_{CO}}(y_s)$ within the whole cohort not just the group itself.

To proceed we assume that $EI_s^{CFL} = 8\text{cov}(\Omega_{SU}, h_s, F_{\Omega_{SU}}(y_s))$ is the counterfactual level of health inequality that would have been observed at the start date in the absence of those who die by the end date, where this is simply taken to be the intragroup level of health inequality among survivors based on fractional SES ranks $F_{\Omega_{SU}}(y_s)$ within the survivor group—not the cohort as a whole. Hence, the change in health inequality may be written as

$$EI_f - EI_s = \{EI_f - EI_s^{CFL}\} + \{EI_s^{CFL} - EI_s\} = M^{SU} + M^{NS}, \quad (1)$$

where M^{NS} fully captures the direct effect of selective mortality on health inequality changes while M^{SU} captures the net effect of health and SES rank changes among the surviving members of the cohort.

M^{NS} can in turn be broken down into a set of component mobility indices that result from the sequential elimination of the three direct effects of selective mortality identified in the introduction. Following the approach taken in Allanson and Petrie (2013a), EI_s may be exactly decomposed in the manner of Yitzhaki (1994) into a between-group component EI_s^{BTW} and a weighted sum of within-group components, EI_s^{SU} , and EI_s^{NS} :

$$\begin{aligned} EI_s &= EI_s^{BTW} + p^{SU} EI_s^{SU} + p^{NS} EI_s^{NS} \\ &= 8\text{cov}(\Omega_{CO}, \bar{h}_s^g, \bar{F}_{\Omega_{CO}}^g(y_s)) + p^{SU} 8\text{cov}(\Omega_{SU}, h_s, F_{\Omega_{CO}}(y_s)) + p^{NS} 8\text{cov}(\Omega_{NS}, h_s, F_{\Omega_{CO}}(y_s)), \end{aligned} \quad (2)$$

where the weights p^{SU} and p^{NS} are the population proportions of the two groups in the cohort, \bar{h}_s^g is the mean health of group g at the start date, and $\bar{F}_{\Omega_{CO}}^g(y_s)$ is the corresponding mean fractional rank in the full cohort population. Hence, M^{NS} is equal to

$$M^{NS} = -EI_s^{BTW} + p^{NS} [EI_s^{SU} - EI_s^{NS}] + [EI_s^{CFL} - EI_s^{SU}] \equiv M^{NSB} + M^{NSW} + M^{NSR}, \quad (3)$$

where the derivation relies on the identity $p^{SU} = (1 - p^{NS})$.

First, the between-group mobility index $M^{NSB} = -EI_s^{BTW}$ captures the effect of average differences between survivors and nonsurvivors at the start of the interval. Specifically, $M^{NSB} = 8\text{cov}(\Omega_{CO}, h'_s - h_s, F_{CO}(y_s))$, where the health

counterfactual $h'_s = \left(h_s^{SU}, h_s^{NS} + \left[\bar{h}_s^{SU} - \bar{h}_s^{NS} \right] \right)$ is obtained from $h_s = \left(h_s^{SU}, h_s^{NS} \right)$ by an equal absolute change in the health of nonsurvivors that eliminates the mean health gap between the two groups. M^{NSB} is expected to be negative, as has previously been discussed, implying that health inequality at the end date is lower than otherwise would have been the case had the average health and/or SES rank of the two groups been the same initially. This equalizing effect is likely to be increasingly large in older cohorts since $EI_s^{BTW} = p^{NS} \left(1 - p^{NS} \right) \left(\bar{h}_s^{SU} - \bar{h}_s^{NS} \right) \left(\bar{F}_{\Omega_{CO}}^{SU} \left(y_s \right) - \bar{F}_{\Omega_{CO}}^{NS} \left(y_s \right) \right)$ is increasing in the mortality rate p^{NS} if $p^{NS} < 0.5$.

Second, the within-group difference mobility index $M^{NSW} = p^{NS} \left[EI_s^{SU} - EI_s^{NS} \right]$ captures the effect of any initial difference in the levels of health inequality within the survivor and nonsurvivor groups. $M^{NSW} = 8\text{cov} \left(\Omega_{CO}, h_s'' - h'_s, F_{CO} \left(y_s \right) \right)$, where $h_s'' = \left(h_s^{SU}, \bar{h}_s^{SU} + EI_s^{SU} \left(h_s^{NS} - \bar{h}_s^{NS} \right) / EI_s^{NS} \right)$ is obtained from h'_s by a mean-preserving spread/contraction of the health of nonsurvivors about the translated mean that further eliminates the difference in within-group inequality between the two groups. M^{NSW} will equal zero if the social gradients of the two groups within the overall cohort population were initially the same. In general, whether M_c^{NSW} is positive or negative is an empirical question, which will depend on both the variation in health (and SES ranks) and the strength of the correlation between health and SES ranks in each of the two groups (cf. Milanovic, 1997). However, all other things equal, the size of the effect will be larger in older cohorts with higher mortality rates.

Third, the reranking index $M^{NSR} = \left[EI_s^{CFL} - EI_s^{SU} \right]$ captures the effect of any initial differences between the ranking of survivors within the survivor and full cohort populations, where $EI_s^{SU} = 8\text{cov} \left(\Omega_{CO}, h_s'', F_{CO} \left(y_s \right) \right)$ by construction. $M^{NSR} = 8\text{cov} \left(\Omega_{SU}, h_s, \left(F_{\Omega_{SU}} \left(y_s \right) - F_{\Omega_{CO}} \left(y_s \right) \right) \right) = p^{NS} 8\text{cov} \left(\Omega_s^{SU}, h_s, \left(F_{\Omega_{SU}} \left(y_s \right) - F_{\Omega_{NS}} \left(y_s \right) \right) \right)$, which will equal zero if the risk of mortality is independent of SES and generally seems to be negligible even in older cohorts with higher mortality rates.⁴

M^{SU} may also be further decomposed to offer a more detailed analysis of the effects of health and SES rank changes among the surviving population. For this purpose, we refine the decomposition procedure in Allanson et al. (2010) to provide a symmetric treatment of SES and health changes based on the “threefold” decomposition (cf. Jann, 2008)

$$\begin{aligned} M^{SU} &= M^{SUS} + M^{SUS} + M^{SUC} \\ &= 8\text{cov} \left(\Omega_{SU}, \left(h_f - h_s \right), F_{\Omega_{SU}} \left(y_s \right) \right) + 8\text{cov} \left(\Omega_{SU}, h_s, \left(F_{\Omega_{SU}} \left(y_f \right) - F_{\Omega_{SU}} \left(y_s \right) \right) \right) \\ &+ 8\text{cov} \left(\Omega_{SU}, \left(h_f - h_s \right), \left(F_{\Omega_{SU}} \left(y_f \right) - F_{\Omega_{SU}} \left(y_s \right) \right) \right), \end{aligned} \quad (4)$$

where M^{SUH} is defined as the negative of the Allanson and Petrie (2013b) income-related health mobility index for the Erreygers index, while M^{SUS} and M^{SUC} sum to the corresponding health-related income mobility index.

M^{SUH} , M^{SUS} , and M^{SUC} capture the effects on health inequality among survivors of health changes associated with initial socioeconomic status, SES changes related to initial health, and contemporaneous health and SES changes, respectively. For example, M^{SUH} will be positive if contemporaneous changes in health and SES among survivors are disqualifying in absolute terms, which will be the case if changes in health and SES rank over the interval are positively correlated with each other. The decomposition is potentially informative about the dynamic interdependence between health and SES to the extent that health and SES rank at the start date are predictive of health and SES rank changes over the interval. In particular, nonzero values of M^{SUH} and M^{SUS} may be indicative respectively of causal pathways from SES to health and vice versa, while nonzero values of M^{SUC} could be reflective of a bidirectional relationship and/or the existence of other factors that simultaneously influence both SES and health (cf. Cutler et al., 2008). But it must be borne in mind that all three mobility indices will be affected by the indirect effects of selective mortality. Petrie et al. (2011) have previously shown that explicitly taking death into account can change the direction of the association between relative health changes and initial SES rank from negative to positive.

2.2 | Decomposition procedure based on end of interval counterfactual

For comparative purposes, we also provide a decomposition based on the imputation of the “would be” health of nonsurvivors at the end date. Let $h_f^* = \left(h_f^{SU}, \hat{h}_f^{NS} \right)$ and $y_f^* = \left(y_f^{SU}, \hat{y}_f^{NS} \right)$, where h_f^{SU} and y_f^{SU} are, respectively, the observed levels of health and SES of survivors at the end date, and \hat{h}_f^{NS} and \hat{y}_f^{NS} are the corresponding imputed levels for nonsurvivors.

Hence, $EI_f^{CFL} = 8\text{cov}(\Omega_{CO}, h_f^*, F_{\Omega_{CO}}(y_f^*))$ is the counterfactual level of health inequality that would obtain at the end date if there had been no deaths during the time interval. The change in health inequality between the two dates may then be written as

$$EI_f - EI_s = \{EI_f - EI_f^{CFL}\} + \{EI_f^{CFL} - EI_s\} = \tilde{M}^{NS} + \tilde{M}^{CO}, \quad (5)$$

where \tilde{M}^{NS} provides an alternative measure of the direct effect of selective mortality on health inequality and \tilde{M}^{CO} captures the net effect of the partially imputed morbidity and SES rank changes in the full cohort population.

\tilde{M}^{NS} may be decomposed in the same way as M^{NS} to give

$$\begin{aligned} \tilde{M}^{NS} &= \tilde{M}^{NSB} + \tilde{M}^{NSW} + \tilde{M}^{NSR} \\ &= -8\text{cov}(\Omega_{CO}, \bar{h}_f^{*g}, \bar{F}_{\Omega_{CO}}^g(y_f^*)) + p^{NS} \left(8\text{cov}(\Omega_{SU}, h_f^{SU}, F_{\Omega_{CO}}(y_f^*)) - 8\text{cov}(\Omega_{NS}, \hat{h}_f^{NS}, F_{\Omega_{CO}}(y_f^*)) \right) \\ &\quad + 8\text{cov}(\Omega_{SU}, h_f^{SU}, F_{\Omega_{SU}}(y_f^*)) - 8\text{cov}(\Omega_{SU}, h_f^{SU}, F_{\Omega_{CO}}(y_f^*)), \end{aligned} \quad (6)$$

where \tilde{M}^{NSB} , \tilde{M}^{NSW} , and \tilde{M}^{NSR} may be interpreted respectively as between-group, within-group difference, and reranking mobility indices, as before, but will in general differ from M^{NSB} , M^{NSW} , and M^{NSR} due to the path dependency of the decomposition procedure. Moreover, the value of all three indices will depend upon the method chosen to impute the “would be” health and SES of nonsurvivors.

Finally, \tilde{M}^{CO} may also be decomposed to yield

$$\begin{aligned} \tilde{M}^{CO} &= \tilde{M}^{COH} + \tilde{M}^{COS} + \tilde{M}^{COC} \\ &= 8\text{cov}(\Omega_{CO}, (h_f^* - h_s), F_{\Omega_{CO}}(y_s)) + 8\text{cov}(\Omega_{CO}, h_s, (F_{\Omega_{CO}}(y_f^*) - F_{\Omega_{CO}}(y_s))) \\ &\quad + 8\text{cov}(\Omega_{CO}, (h_f^* - h_s), (F_{\Omega_{CO}}(y_f^*) - F_{\Omega_{CO}}(y_s))), \end{aligned} \quad (7)$$

where the three indices respectively capture the effects on health inequality among the full cohort in partially imputed health changes associated with initial SES rank, partially imputed SES rank changes related to initial health, and partially imputed contemporaneous health and SES rank changes.

3 | EMPIRICAL ANALYSIS

We employ the decomposition procedures to uncover the changing nature of the interdependence between health and SES throughout adulthood using individual panel data for GB from the United Kingdom Household Longitudinal Study (UKHLS, also known as “Understanding Society”; University of Essex and Institute for Social and Economic Research, 2020) and British Household Panel Survey (BHPS; University of Essex & Institute for Social & Economic Research, 2010).⁵ Specifically, the main analysis uses UKHLS data to examine changes in health inequality in GB for rolling age groups between 2013 and 2014 and 2018 and 2019, which is treated as a single time interval in order to ensure the occurrence of at least some deaths in all study cohorts. UKHLS is a nationally representative household panel survey that began collecting information in 2009–2010 on over 60,000 enumerated persons in 30,000 responding private households, with the sample boosted from Wave 2 by the inclusion of participants from the predecessor BHPS, and Wave 10 the latest data release at the time of writing. Annual personal interviews are conducted with all adult household members and provide rich information on a wide range of topics including sociodemographic characteristics and self-assessed health (see Knies, 2017, for further details). We also undertake a number of supplementary analyses to explore the extent to which our main findings may be affected by key aspects of the study design, most notably the choice of SES variable, and may differ across the economic cycle.

3.1 | Main analysis design

UKHLS records the cause of sample attrition between waves, including death, where this is known. For the main analysis, we use data from Waves 5 (2013–2014) and 10 (2018–2019) to construct an unbalanced panel consisting of observations

on the subset of individuals in the survey for whom full information on health, age, gender, educational attainment, and income was available for both the start and end date of the study period or for whom full information was available for the start date and the individual was known to have died by the end date. Sample weights were used throughout the analysis with these being given by adjusted cross-sectional survey weights for the start date, where the adjustments were made using inverse probability weights (see Wooldridge, 2001) to allow both for missing data for either the start or end date and for nonmortality-related sample attrition over the study period (see Petrie et al., 2011, for further discussion). Bootstrap standard errors were obtained for all mean, inequality, and mobility measures by the resampling of primary sampling units within each stratification class to reflect the survey sample design.

The cohort analysis is based on 15 year overlapping age groups (25–40, ..., 65–80, 70+) based on age in 2013–2014, where the choice of age span was guided by the familiar trade-off between bias and variance. Longer age spans resulted in excessive smoothing of the age-stratified estimates as age groups start to encompass individuals facing very different mortality risks, particularly in later life. But age spans shorter than 15 years led to excessive variability in the estimates of the component mobility indices by age group, with the reduction in sample size becoming increasingly problematic in younger age groups in which the mortality rate is lower. Adults younger than 25 years old are excluded from the analysis since deaths in this age group were very uncommon.

3.2 | Main analysis variable definitions

The health measure used in the study is Quality Adjusted Life Years (QALYs) derived from the responses to the SF-12v2 questionnaire in UKHLS using the SF-6D preference-based algorithm (Brazier et al., 2002). QALYs allow both the quality and quantity of health individuals experience to be combined into a single meaningful measure (see Drummond et al. (2015) for further discussion). The measure is bounded in the unit interval with full health corresponding to a value of 1, the lowest possible health utility of anyone alive being equal to 0.345, and with death assigned a QALY of 0.

The most common indicators of SES in health inequality studies are education, income, and social class (Darin-Mattsson et al., 2017). The current study is limited to the use of education and income indicators because social class in the UKHLS is based on current occupation and therefore unavailable for those not or no longer in work, with the former preferred to the latter as the main measure of SES for a number of reasons. First educational attainment, defined as highest educational or vocational qualification, provides a more stable indicator of SES in adulthood. In particular, the transition from working life into retirement is associated with a significant fall in income (see, e.g., Bardasi et al., 2002), with this leading to considerable movement within the income distributions of older age groups not least because of the range of ages over which people retire. Income is also more likely subject to reverse causation: poor health has been associated with both below-average income growth among working age adults (Deaton & Paxson, 1998) and above-average income growth among retirement age adults eligible for the receipt of additional disability and social care benefits (Zaidi, 2008). Second, income may be more prone to measurement error, with this a particular concern in older age groups (Zaidi, 2008). Finally, education has been found to provide a stronger predictor of both the onset of chronic health conditions and mortality in adulthood than income or other financial measures (see, e.g. Smith, 2007; Torssander & Erikson, 2010). Educational attainment in the UKHLS is measured by a derived variable that distinguishes between those with a degree, other higher degree, A level or equivalent, GCSE or equivalent, other qualifications, and none.

3.3 | Supplementary analyses

A number of supplementary analyses were undertaken to explore the robustness of the main findings to key aspects of the study design. First, we repeated the main analysis using income rather than educational attainment as the SES variable, where this might be expected to have at least some effect on the results for the reasons discussed above. Income is defined as net monthly household equivalent income and is equal to the total monthly income of all household members net of taxes and national insurance contributions and adjusted for household size and composition using the OECD-modified equivalence scale. Separate subgroup analyses were also conducted for men and women. Second, we used UKHLS Wave 2 and 7 data to repeat the main analysis for the period 2010–2011 to 2015–2016. Finally, we used BHPS data to generate results for the 5-year time span 1999–2004, which was a time of strong economic growth and investment in public services in contrast to the austerity conditions that have prevailed throughout the period covered by the UKHLS. Further details of the supplementary analyses are provided in Appendix 2.

4 | RESULTS

Table 1 presents descriptive statistics by rolling age group for health and educational attainment rank for the main analysis. Average cohort health in 2013–2014 was relatively stable into early old age and then dropped markedly in the two oldest cohorts. However, the average health of survivors in all cohorts declined over the 5-year time interval to 2018–2019, with the largest falls in early adulthood and old age. Moreover, the mortality rate was strictly increasing with age, with 27.9% of the over-70 age group recorded as dead by 2018–2019. Nonsurvivors in all age groups had on average both worse health and lower SES rank than survivors in 2013–2014, providing prima facie evidence of selective mortality.

Contrary to the stylized fact that health inequalities diminish in old age but consistent with a number of other studies (e.g., Mirowsky & Ross, 2005; Ross & Wu, 1995), Figure 2 shows no narrowing of the health gap across levels of education in older cohorts in 2013–2014. This might appear to cast doubt on the possibility of significant selective mortality effects on health inequalities, but such a conclusion would be unwarranted as the shape of each of the curves will in general reflect a combination of age, period and cohort effects. More revealing is a comparison of the location of the two curves, with health inequalities lower in 2018–2019 than in 2013–2014 in all cohorts aged 45–60 and above. Thus, the pattern of individual health changes, educational attainment changes, and deaths within each of these cohorts led to a fall in the social gradient among the surviving members of the cohort, although Table 2 shows that these changes were only statistically significant in the oldest cohorts.

To further explore how the coevolution of health and SES gave rise to the observed change in health inequality over the lifecycle, Table 2 presents results from our preferred decomposition procedure based on the start date counterfactual which leaves out those who will die during the study period. The change in health inequality within each cohort ($EI_f - EI_s$) is accounted for by the direct effect of selective mortality M^{NS} and the net effect of changes in morbidity and educational attainment among survivors M^{SU} , where both these terms are further broken down into three component mobility indices. In all cases, positive values indicate a contribution that results in greater health inequality among those still alive in 2018–2019.

Looking first at the direct effects of selective mortality M^{NS} , then the contribution to the overall change in health inequality is relatively small in early adulthood, when mortality rates are very low, but becomes significantly negative and increasingly large in older cohorts. The detailed decomposition show that the main driver of this effect was initial differences between the average health and SES rank of survivors and non-survivors M^{NSB} , with the disproportionate number of deaths among less educated and unhealthier individuals contributing to significantly lower levels of health inequality among the surviving members of all but the youngest age groups. Estimates of the within-group difference and reranking mobility indices, M^{NSW} and M^{NSR} , are generally insignificant with the latter also being negligible in size in all but the oldest cohort which has the highest mortality rate.

The patterns in the other half of the decomposition are less clear with only two of the estimates of M^{SU} and very few of its component mobility indices significantly different from zero at the 5% level. The dominant component of M^{SU} in virtually all age groups is SES-related health mobility M^{SUH} , which is to be expected given the relative stability of educational attainment in adulthood. The positive values of M^{SUH} in early adulthood imply that health losses among survivors were concentrated among the less educated and are therefore consistent with the cumulative advantage hypothesis although not statistically significant. That this does not continue to be the case in older age groups may be due in part to the indirect effects of selective mortality given that it was the less educated and less healthy in each cohort who were more likely to die. Indeed, M^{SUH} is both negative and significant in the two oldest age groups. The other two components of M^{SU} , M^{SUS} , and M^{SUC} , are both consistently smaller in magnitude, with contemporaneous health and SES changes equalizing in most cohorts.

4.1 | Alternative results based on end date counterfactuals

The alternative decomposition procedure is based on the imputation of the health and educational attainment of non-survivors to construct end date counterfactuals that include those individuals who are known to die during the study period. Table 3 presents selected results obtained using three different imputation procedures, with full results provided in Appendix 1.

The first procedure uses inverse probability weights to correct for selective attrition with the weights derived from a logit model in which the probability of survival to 2018–2019 is specified as a function of age, sex, health, highest

TABLE 1 Descriptive statistics by rolling age groups: GB with SES = educational attainment

	Age in 2013–2014									
	25–40	30–45	35–50	40–55	45–60	50–65	55–70	60–75	65–80	70+
Sample size 2013–2014	4589	5534	6165	6643	6560	6254	6123	5403	4298	3015
Mean health 2013–2014	0.772	0.773	0.771	0.768	0.763	0.761	0.765	0.767	0.759	0.728
Mortality rate	0.003	0.006	0.008	0.013	0.020	0.033	0.050	0.074	0.113	0.279
Mean SES rank 2013–2014 (nonsurvivors)	0.363	0.323	0.351	0.355	0.352	0.365	0.374	0.396	0.423	0.431
Mean SES rank 2013–2014 (survivors)	0.500	0.501	0.501	0.502	0.503	0.505	0.507	0.508	0.510	0.527
Mean health 2013–2014 (nonsurvivors)	0.599	0.643	0.643	0.641	0.647	0.674	0.697	0.694	0.690	0.662
Mean health 2013–2014 (survivors)	0.772	0.774	0.772	0.770	0.765	0.764	0.768	0.773	0.768	0.754
Mean health 2018–2019 (survivors only)	0.754	0.757	0.758	0.757	0.756	0.760	0.761	0.761	0.748	0.727

Source: Own calculation from UKHLS data.

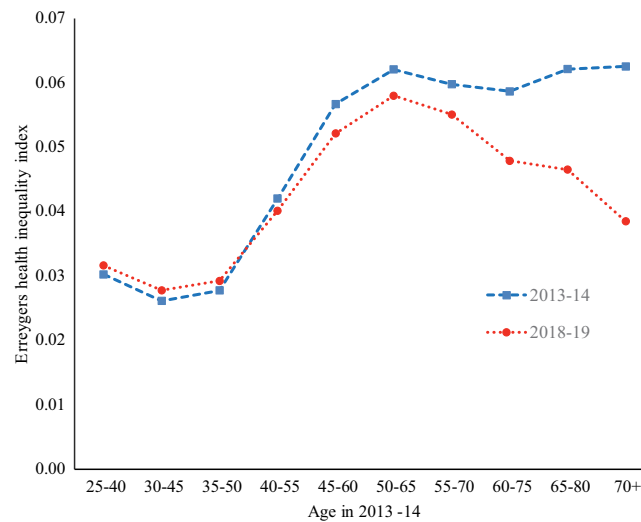


FIGURE 2 Intracohort educational qualification gradient in health by cohort and year

qualification, and the interaction of health and highest qualification in 2013–2014. Thus, in the manner of Baeten et al. (2013), the imputed health and SES of non-survivors at the end of the study period is based on the observed health and SES outcomes of survivors who possessed similar characteristics to them at the start. As expected this procedure leads to smaller estimates of the direct effect of selective mortality than our preferred decomposition procedure, particularly in older age groups with lower survival rates (with exactly offsetting changes in the other half of the decomposition given that the components sum to the observed change in health inequality in both cases). This difference in the results between the two procedures is almost entirely due to the smaller size of the between-group mobility terms, which in turn is largely attributable to the imputed health gaps between survivors and non-survivors in 2018–2019 being smaller than the corresponding actual health gaps in 2013–2014. That the IPW-imputed health gaps at the end date are likely biased downward due to the indirect effect of selective mortality may be inferred from Figure 3. This shows that in every age group, the average imputed health of nonsurvivors in 2018–2019 is higher than their average actual health in 2013–2014, whereas the average health of survivors in all cohorts declined over the study period as reported in Table 1.

We further note that the IPW estimates of the between-group effect for older cohorts are generally smaller than in the second set of end date counterfactual results, which are based on the assumption that the health and highest qualification of nonsurvivors would have been the exactly the same in 2018–2019 as in 2013–2014 had they not died in the intervening period. This naïve “no change” imputation procedure may plausibly be seen to provide an upper bound on the “would-be” health of nonsurvivors with the resultant estimates of the direct effect of select mortality for the oldest cohorts still appreciably smaller than in our preferred approach. Finally, we present an estimate of SES-related health mobility based on the assignment of a QALY score of zero to nonsurvivors in 2018–2019 but with no change in their

TABLE 2 Start date counterfactual health inequality change decomposition: GB with SES = educational attainment, 2013–2014 to 2018–2019

	Age in 2013–2014									
	25–40	30–45	35–50	40–55	45–60	50–65	55–70	60–75	65–80	70+
Initial inequality EI_s	0.0303***	0.0261***	0.0278***	0.0420***	0.0567***	0.0621***	0.0598***	0.0587***	0.0621***	0.0626***
	0.0052	0.0047	0.0050	0.0049	0.0048	0.0048	0.0047	0.0047	0.0053	0.0064
Final inequality EI_f	0.0317***	0.0278***	0.0293***	0.0402***	0.0522***	0.0580***	0.0551***	0.0479***	0.0465***	0.0385***
	0.0057	0.0049	0.0050	0.0047	0.0049	0.0049	0.0048	0.0049	0.0056	0.0070
Change in inequality $EI_f - EI_s$	0.0014	0.0017	0.0015	-0.0019	-0.0046	-0.0041	-0.0047	-0.0108**	-0.0156***	-0.0241***
	0.0061	0.0051	0.0051	0.0043	0.0043	0.0045	0.0044	0.0046	0.0053	0.0073
Nonsurvivors mobility M^{NS}	-0.0007	-0.0012*	-0.0012	-0.0020**	-0.0031***	-0.0034***	-0.0049***	-0.0059***	-0.0046**	-0.0088*
of which due to:	0.0005	0.0006	0.0006	0.0008	0.0010	0.0012	0.0013	0.0016	0.0022	0.0048
–Between-groups M^{NSB}	-0.0006	-0.0010*	-0.0013**	-0.0020***	-0.0028***	-0.0032***	-0.0036***	-0.0049***	-0.0054***	-0.0141***
	0.0005	0.0006	0.0005	0.0007	0.0008	0.0008	0.0009	0.0010	0.0012	0.0024
–Within-group difference M^{NSW}	-0.0001	-0.0002	0.0001	0.0001	-0.0002	-0.0002	-0.0012	-0.0010	0.0009	0.0041
	0.0003	0.0003	0.0004	0.0005	0.0007	0.0009	0.0010	0.0013	0.0019	0.0042
–Reranking M^{NSR}	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	-0.0001	0.0000	0.0000	0.0012***
	0.0000	0.0000	0.0000	0.0000	0.0001	0.0001	0.0001	0.0001	0.0001	0.0004
Survivors mobility M^{SU}	0.0020	0.0029	0.0027	0.0001	-0.0015	-0.0007	0.0002	-0.0049	-0.0111**	-0.0152**
of which due to:	0.0060	0.0051	0.0051	0.0043	0.0043	0.0044	0.0043	0.0046	0.0050	0.0066
–SES-related health changes M^{SUH}	0.0056	0.0054	0.0047	0.0016	-0.0002	0.0000	0.0004	-0.0051	-0.0111**	-0.0154**
	0.0060	0.0051	0.0050	0.0043	0.0042	0.0043	0.0043	0.0046	0.0050	0.0065
–Health-related SES changes M^{SUS}	-0.0008	-0.0005	-0.0007	-0.0002	0.0003	0.0005	0.0005	0.0001	0.0003	0.0007
	0.0015	0.0012	0.0010	0.0007	0.0006	0.0005	0.0004	0.0004	0.0004	0.0007
–Correlated health & SES changes M^{SUC}	-0.0027	-0.0020	-0.0012	-0.0012	-0.0016**	-0.0012**	-0.0007	0.0001	-0.0003	-0.0005*
	0.0018	0.0015	0.0009	0.0007	0.0007	0.0006	0.0005	0.0002	0.0002	0.0003

Note: Cluster-robust standard errors in italics. *** significant at the 1% level, ** significant at the 5% level, * significant at the 10% level.

TABLE 3 Selected decomposition results from alternative end date counterfactuals: GB with SES = educational attainment, 2013–14 to 2018–19

		Age in 2013–2014										
		25–40	30–45	35–50	40–55	45–60	50–65	55–70	60–75	65–80	70+	
Change in inequality $E_f - EI_s$		0.0014	0.0017	0.0015	-0.0019	-0.0046	-0.0041	-0.0047	-0.0108**	-0.0156***	-0.0241***	
IPW imputation		0.0061	0.0051	0.0051	0.0043	0.0043	0.0045	0.0044	0.0046	0.0053	0.0073	
Full cohort mobility \tilde{M}^{CO}		0.0017	0.0024	0.0022	-0.0007	-0.0029	-0.0016	-0.0013	-0.0064	-0.0113**	-0.0178**	
of which due to:		0.0061	0.0051	0.0051	0.0043	0.0042	0.0044	0.0044	0.0046	0.0052	0.0076	
– SES-related health changes \tilde{M}^{COH}		0.0053	0.0049	0.0042	0.0007	-0.0016	-0.0009	-0.0012	-0.0065	-0.0113**	-0.0180**	
Nonsurvivors mobility \tilde{M}^{NS}		0.0061	0.0051	0.0050	0.0043	0.0042	0.0044	0.0044	0.0046	0.0052	0.0076	
of which due to:		-0.0003	-0.0007**	-0.0007**	-0.0012**	-0.0017***	-0.0025***	-0.0034***	-0.0044***	-0.0043**	-0.0063	
– Between-groups \tilde{M}^{NSB}		0.0003	0.0003	0.0003	0.0005	0.0006	0.0009	0.0009	0.0011	0.0018	0.0036	
No change imputation		-0.0002	-0.0005*	-0.0005**	-0.0008**	-0.0013***	-0.0020***	-0.0026***	-0.0035***	-0.0041***	-0.0090***	
Full cohort mobility \tilde{M}^{CO}		0.0002	0.0003	0.0002	0.0003	0.0004	0.0005	0.0006	0.0007	0.0009	0.0017	
of which due to:		0.0020	0.0028	0.0027	0.0000	-0.0016	-0.0007	0.0000	-0.0052	-0.0111**	-0.0147***	
– SES-related health changes \tilde{M}^{COH}		0.0060	0.0051	0.0051	0.0042	0.0042	0.0042	0.0041	0.0042	0.0044	0.0047	
Nonsurvivors mobility \tilde{M}^{NS}		0.0055	0.0052	0.0045	0.0013	-0.0004	-0.0001	0.0001	-0.0054	-0.0112**	-0.0150***	
of which due to:		0.0060	0.0051	0.0050	0.0042	0.0042	0.0042	0.0041	0.0042	0.0044	0.0046	
– Between-groups \tilde{M}^{NSB}		-0.0006	-0.0011*	-0.0012*	-0.0019**	-0.0030***	-0.0033***	-0.0047***	-0.0056***	-0.0045**	-0.0093**	
Imputation with $\hat{h}_f^{NS} = 0$ & $\hat{y}_f^{NS} = y_s^{NS}$		0.0005	0.0006	0.0006	0.0008	0.0010	0.0012	0.0013	0.0015	0.0021	0.0047	
– SES-related health changes \tilde{M}^{COH}		-0.0005	-0.0010	-0.0012**	-0.0019***	-0.0027***	-0.0031***	-0.0033***	-0.0042***	-0.0040***	-0.0101***	
of which due to:		0.0004	0.0005	0.0005	0.0007	0.0008	0.0008	0.0008	0.0009	0.0010	0.0019	
– Between-groups \tilde{M}^{NSB}		0.0072	0.0100	0.0107*	0.0108**	0.0135**	0.0213***	0.0314***	0.0325***	0.0312***	0.0770***	
Nonsurvivors mobility \tilde{M}^{NS}		0.0061	0.0055	0.0055	0.0050	0.0053	0.0059	0.0066	0.0075	0.0091	0.0155	

Note: Cluster-robust standard errors in italics. *** significant at the 1% level, ** significant at the 5% level, * significant at the 10% level.

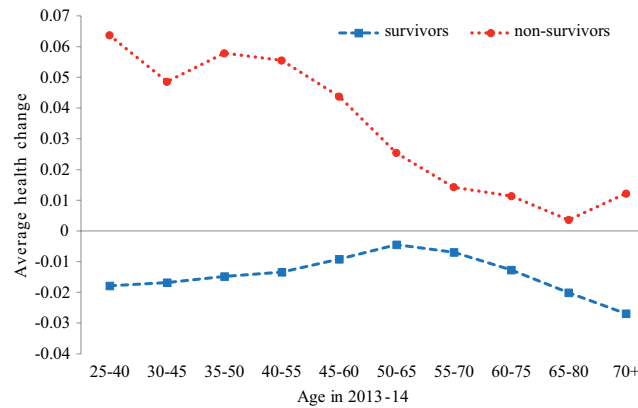


FIGURE 3 Average health changes 2013–2014 to 2018–2019 by cohort and survival status: actual changes for survivors and IPW-imputed changes for nonsurvivors

educational attainment, which shows that health losses would indeed be concentrated among the less educated members of each cohort if deaths were explicitly taken into account in this way.

4.2 | Results from supplementary analyses

The supplementary analyses are designed to explore the robustness of the main findings to key aspects of the study design, with results presented only for our preferred start date counterfactual. Table 4 presents results with income rather than educational attainment as the SES variable, where this change might be expected to result in clearer evidence of direct selective mortality effects given that Figure 4 shows that health inequalities in both 2013–2014 and 2018–2019 were lower in each successively older cohort beyond the 50–65 age group. In practice, the findings are only partly supportive of the importance of direct selective mortality effects as a consequence of the previously identified weaknesses of income as a useful indicator of SES across the adult lifespan. It remains the case that the between groups effect M^{NSB} becomes significantly negative and increasingly large in older age groups. But this is more than offset by the within-group difference effect M^{NSW} in the two oldest age groups—such that the total direct effect of selective mortality M^{NS} turns insignificantly positive—with the positive values of M^{NSW} in older cohorts explained not by the health in 2013/2014 of those would die by 2018/2019 being more uniform than that of survivors but rather by a lack of association between their health and income rank in comparison to survivors. Moreover, survivor mobility effects are relatively more important drivers of the overall change in health inequality due to the extent of income mobility in older age groups, with evidence of reverse causality provided by the health-related income mobility index M^{SUS} estimates.

Educational attainment is taken as the SES variable in the other supplementary analyses, which provide findings for women and men separately and for the 5-year time intervals 2010–2011 to 2015–2016, and 1994–1999. The decomposition results from these analyses are presented in Appendix 2 and closely conform with those from the main analysis presented in Table 2. In particular, the between groups effects M^{NSB} become significantly negative and increasingly large into late old age and thereby becomes the most important factor in explaining the observed change in the social gradient in health in later life. Moreover, SES-related health mobility among survivors tends to become less positive/more negative in older cohorts subject to higher mortality rates.

5 | CONCLUSION

This paper does not aim to present evidence on the causal nexus between SES and health over the lifecycle, but rather to inform such analysis by fully accounting for the changing strength of the association between SES and health at different ages in terms of the contributions of selective mortality, morbidity changes, and SES changes. In particular, the mobility indices presented in the paper should be interpreted as a sufficient set of summary statistics characterizing the coevolution of health and SES over time rather than as structural parameters of some underlying dynamic

TABLE 4 Start date counterfactual health inequality change decomposition: GB with SES = income, 2013–2014 to 2018–2019

	Age in 2013–2014									
	25–40	30–45	35–50	40–55	45–60	50–65	55–70	60–75	65–80	70+
Initial inequality EI_s	0.0538***	0.0603***	0.0656***	0.0769***	0.0865***	0.0885***	0.0794***	0.0675***	0.0611***	0.0404***
	0.0055	0.0050	0.0053	0.0046	0.0047	0.0044	0.0046	0.0047	0.0052	0.0071
Final inequality EI_f	0.0514***	0.0567***	0.0650***	0.0745***	0.0801***	0.0743***	0.0641***	0.0458***	0.0313***	0.0024
	0.0059	0.0052	0.0050	0.0049	0.0050	0.0046	0.0047	0.0050	0.0061	0.0079
Change in inequality $EI_f - EI_s$	-0.0024	-0.0036	-0.0006	-0.0024	-0.0065	-0.0142**	-0.0153***	-0.0217***	-0.0298***	-0.0379***
	0.0067	0.0061	0.0056	0.0050	0.0053	0.0053	0.0053	0.0055	0.0065	0.0089
Nonsurvivors mobility M^{NS}	-0.0007	-0.0015*	-0.0017*	-0.0034***	-0.0041***	-0.0040***	-0.0027**	-0.0013	0.0003	0.0050
of which due to:										
– Between-groups M^{NSB}	0.0004	0.0008	0.0009	0.0012	0.0012	0.0012	0.0013	0.0016	0.0023	0.0049
	-0.0007*	-0.0009	-0.0012*	-0.0028***	-0.0038***	-0.0042***	-0.0041***	-0.0054***	-0.0054***	-0.0075***
– Within-group difference M^{NSW}	0.0004	0.0006	0.0007	0.0010	0.0011	0.0010	0.0009	0.0010	0.0012	0.0024
	0.0001	-0.0006	-0.0005	-0.0008	-0.0005	-0.0001	0.0015	0.0041***	0.0057***	0.0121***
	0.0002	0.0004	0.0006	0.0007	0.0008	0.0009	0.0011	0.0014	0.0021	0.0043
– Reranking M^{NSR}	0.0000	0.0000	0.0000	0.0002**	0.0002**	0.0003**	0.0000	0.0000	0.0000	0.0004
	0.0000	0.0000	0.0000	0.0001	0.0001	0.0001	0.0001	0.0002	0.0002	0.0004
Survivors mobility M^{SU}	-0.0018	-0.0022	0.0010	0.0010	-0.0024	-0.0102*	-0.0126**	-0.0204***	-0.0301***	-0.0429***
of which due to:										
– SES-related health changes M^{SUH}	0.0066	0.0061	0.0055	0.0050	0.0051	0.0052	0.0052	0.0056	0.0065	0.0088
	0.0046	-0.0052	-0.0064	-0.0070*	-0.0045	-0.0073	-0.0064	-0.0095*	-0.0162***	-0.0254***
	0.0058	0.0055	0.0050	0.0043	0.0044	0.0042	0.0045	0.0049	0.0052	0.0075
– Health-related SES changes M^{SUS}	-0.0028	-0.0010	0.0018	0.0016	-0.0029	-0.0092**	-0.0147***	-0.0153***	-0.0148***	-0.0158***
	0.0044	0.0039	0.0038	0.0033	0.0037	0.0040	0.0040	0.0039	0.0047	0.0060
– Correlated health & SES changes M^{SUC}	-0.0036	0.0041	0.0056	0.0064*	0.0051	0.0063	0.0085**	0.0044	0.0010	-0.0017
	0.0049	0.0045	0.0041	0.0037	0.0037	0.0039	0.0038	0.0041	0.0044	0.0061

Note: Cluster-robust standard errors in italics. *** significant at the 1% level, ** significant at the 5% level, * significant at the 10% level.

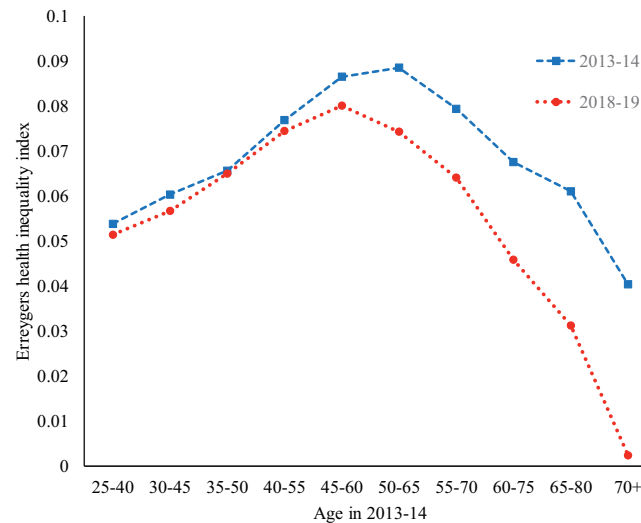


FIGURE 4 Intracohort income gradient in health by cohort and year

process, providing “catch-all” measures that encompass the variety of possible mechanisms as discussed, for instance, in Mirowsky & Ross (2008).

A particular attraction of our longitudinal decomposition methodology is the detailed account it provides of how selective mortality impacts on the evolution of health inequality over the lifecycle. The results from our main empirical analysis reveal that selective mortality is an important driver of changes in cross-sectional health inequality over the lifecycle, especially in older age groups in which survival rates are lower, contrary to the findings in studies such as Beckett (2000) and Baeten et al. (2013). Specifically, initial differences between the average health and educational attainment rank of survivors and nonsurvivors are shown to be an increasingly important factor in explaining the observed change in the social gradient in health in later life. This contrast in findings is at least in part attributable to the utilization of a counterfactual for the start rather than the end of the study period to identify the direct effects of selective mortality. Our approach, as a result, does not rely on the imputation of the “would be” health and SES of nonsurvivors, which it is shown yields estimates of the between-group selective mortality effect that are biased toward zero if based on the health outcomes of survivors. We also demonstrate that estimates of the direct effects of selective mortality are sensitive to the choice of SES measure, with the negative between-group effect more than offset in older cohorts by a positive within-group difference effect if income is chosen instead of educational attainment.

Our preferred decomposition procedure further allows for the detailed analysis of the effects of health and SES changes conditional upon survival, with the symmetric treatment of health and SES rank changes having the potential to identify the nature of the interdependence of health and SES over the lifecycle. The results of this further analysis are relatively uninformative, perhaps because the study lacked sufficient statistical power to detect all empirically relevant aspects of the potentially bidirectional relationship between SES and health, given the limited number of observations on each age cohort in the UKHLS. Nevertheless, the findings do point to the likely importance of taking indirect selective mortality effects into consideration in balanced panel studies, with income-related health mobility among survivors consistently found to be less positive/more negative in older cohorts subject to higher mortality rates.

In conclusion, the proposed approach provides a coherent analytical framework to fully identify the drivers of the changing social gradient in health over the lifecycle. Further studies are required to explore whether our empirical findings are more generally characteristic of lifecycle dynamics in other countries, where it may be noted that estimation of the direct effects of selective mortality only requires follow-up data on deaths even though full longitudinal data is needed for the complete decomposition. It would also be of interest to examine both the short-run impact and longer term effects of the COVID-19 epidemic on the pattern of age-specific health inequalities. Finally, the methodology might be used to investigate the dynamics of the social gradient in specific health conditions and risk factors such as obesity and smoking over the lifecycle.

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CONFLICT OF INTERESTS

The authors declare that there are no conflict of interests.

DATA AVAILABILITY STATEMENT

The data that support the findings of this study are openly available from the UK Data Service.

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ENDNOTE

- ¹ In principle this indeterminacy can be resolved through the use of a 'Shapley value' average of the two possible decomposition pathways (see Shorrocks, 2013), but the resultant measures lack ready interpretation while still requiring imputation of the health of non-survivors.
- ² The counterpart assumption in end date counterfactual analyses is that the joint distribution of the survivor group at the end date is independent of the survival of the non-survivor group.
- ³ Consideration of sources of sample attrition other than mortality is addressed in the empirical section.
- ⁴ See Allanson and Petrie (2013a) for further discussion of the properties of the re-ranking index, which it may be noted is not dependent on the health of non-survivors at the start date.
- ⁵ The analysis is limited to GB for comparative purposes because the BHPS data for Northern Ireland do not constitute a panel over the supplementary analysis period 1999 to 2004, which are the only two years in which the BHPS carried the SF questionnaire that provides the basis for the health variable.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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