



Widening disparities in all-cause and despair-related mortality among Swedish youths: Disentangling selection and causation

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ABSTRACT

Rising rates of "deaths of despair" – mortality from suicide, drug overdose, and alcohol-related causes – have contributed to widening educational disparities in mortality. It is not known to what extent the trends are due to selection effects (health causing education, or a third factor causing both) or social causation (education affecting health). This study investigated the relative contribution of selection and causation for these trends, focusing on the recently documented widening achievement-based disparities in mortality among Swedish youths.

To this end, two cohorts of Swedish compulsory school graduates (graduating in 1992–1993 and 2009–2010, respectively) were followed for eight years after graduation using comprehensive administrative data ($n = 424,715$). Logistic regression models were used to assess the role of pre-graduation selection, while inverse odds ratio-weighting was used to assess mediation by post-graduation socioeconomic disadvantages.

Roughly half of the association between low achievement and all-cause and despair-related mortality within cohorts was due to selection. However, selection effects did not explain the widening disparities over time. Socioeconomic mediators accounted for most of the remaining disparities within cohorts as well as of for most of the increase in these disparities over time. Overall, social causation was more important than selection in explaining the widening educational disparities in all-cause and despair-related mortality.

We conclude that low academic achievement increasingly constrains life-course prospects of Swedish youths, amplifying its adverse health consequences. These findings highlight the need for lower barriers in the education system and for viable educational and employment pathways for low-achieving students in an increasingly knowledge-intensive labor market.

1. Introduction

Rising rates of "deaths of despair" – mortality from suicide, drug overdose, and alcohol-related causes – have emerged as a critical public health concern (King et al., 2022). Popularized in the wake of seminal work by economists Case and Deaton (2015), the term "despair" seeks to capture the shared sense of hopelessness, distress, and loss of purpose that, it is argued, underlie these seemingly disparate causes of death (Case and Deaton, 2020). While first observed in the United States, similar increases have been documented in other English-speaking countries (Augarde et al., 2022; Benny et al., 2023; Camacho et al., 2024; Loverock et al., 2024) as well as in Sweden (Högberg et al., 2025), suggesting a broader international trend. Most initial attention was directed toward mid-life mortality among whites (Case and Deaton, 2015), but evidence suggests a notable impact at younger ages as well

(Gutin and Hummer, 2021). Moreover, the increase has largely been driven by the less educated, resulting in marked and growing disparities in despair-related mortality (Case and Deaton, 2022; Högberg et al., 2025).

Subsequent explanatory research has identified multiple drivers of these trends, including broad, structural transformations such as deindustrialization, declining employment prospects, and growing inequality (Autor et al., 2019; Benny et al., 2023; Camacho et al., 2024; Case and Deaton, 2022; Kuo and Kawachi, 2023; Loverock et al., 2024) as well as changes in drug supply, most notably the greater availability of prescribed and illicit opioids (Alpert et al., 2021; Barnett et al., 2017; Finkelstein et al., 2022). While providing valuable insights, research on the widening disparities in despair has largely assumed a social causation perspective – that is, that education, or other indicators of socioeconomic status, causes despair. This assumption aligns with

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paradigmatic social and public health theories on health inequalities (McLeod and Pavalko, 2008), such as fundamental cause theory (Phelan et al., 2010) and the social determinants of health framework (Marmot and Wilkinson, 2005). However, it is well-established that health inequalities, in addition to social causation, can also reflect direct health selection (where health influences education) or indirect selection (where a third factor influences both education and poor health) (Grossman, 2008; Mackenbach, 2012; Montez and Friedman, 2015). Disregarding selection processes risks overstating the role of social causation, thereby skewing our understanding of the underlying drivers of the trends and resulting in misguided policy responses.

The relative importance of social causation and selection processes varies depending on contextual factors (Montez and Friedman, 2015). Social causation theories posit that inequalities are particularly pronounced for preventable causes of death (Phelan et al., 2010) – such as those related to despair – and that, moreover, social and mortality inequalities rise in tandem, such that the widening income gaps observed across the Western world should translate into widening mortality gaps as well (Gutin and Hummer, 2021). Likewise, selection processes are thought to be particularly important for health conditions that strongly inhibit socially or economically valued abilities (McLeod and Pavalko, 2008). It is well-established that the behavioral and psychological underpinnings of despair – e.g., substance use, depression and self-harm – impede educational and labor market success (Dalsgaard et al., 2020; Esch et al., 2014). Furthermore, as disorders of cognitive and behavioral functioning, these “diseases of despair” (Shanahan et al., 2019) may be particularly debilitating in societies characterized by skill-biased technological change, educational expansion, and increasingly knowledge-intensive labor markets.

In other words, both social causation and selection theories offer compelling explanations for the large and widening educational disparities in deaths of despair. However, they make competing predictions concerning the temporal, and ultimately causal, order. If the social causation model is correct, we would expect low educational achievement and attainment to increasingly constrain subsequent socioeconomic prospects, in turn resulting in despair. In contrast, if the selection model is correct, we would expect that health conditions or other disadvantages early in life increasingly undermine educational and socioeconomic prospects, and that the stronger association between education and despair, in a sense, is a statistical artifact.

The present study sought to disentangle these competing explanations, using Sweden as a case. The Swedish context is particularly instructive as it may be seen as a “least likely case” from the perspective of social causation theories. Sweden has long been hailed as perhaps the archetypical social democratic, cradle-to-grave welfare state, characterized by a more egalitarian income distribution, lower poverty rates, and a stronger social safety net compared to other countries with documented increases in deaths of despair, namely the United States, Canada and the United Kingdom. Nonetheless, cracks in the Swedish social model have become increasingly visible – cracks that have disproportionately affected vulnerable segments of the youth population, with rising rates of school failure and persistent youth unemployment (SOU, 2019). Recent studies also report increased despair-related mortality among Swedish youths, particularly youths with low educational achievements (Högberg et al., 2025; Ågren and Bremberg, 2022). Furthermore, Swedish administrative register data provide unique opportunities to track the health and socioeconomic status of multiple cohorts across key life course transitions and thereby sort out the relative contributions of social causation and selection for the widening educational disparities among youths. In contrast, much existing explanatory research on trends in despair has been restricted to area-level data (Bjorklund, 2023; Knapp et al., 2019; Siddiqi et al., 2019), increasing the risk of ecological bias (Camacho et al., 2024). In view of this, the present paper investigated the following two research questions.

- Research question 1 (RQ1): Are the growing achievement-related disparities in all-cause and despair-related mortality among Swedish youths explained by changes in the selection of students into low achievement?
- Research question 2 (RQ2): Are these disparities mediated by low-achieving students’ subsequent socioeconomic disadvantages, net of changes in selection?

1.1. Theoretical framework and previous research

As stated, the association between health and education or academic achievement can come about through two distinct, albeit not mutually exclusive, processes: social causation and direct or indirect selection. The social causation-model draws on theories from sociology and social epidemiology, such as fundamental cause theory (Phelan et al., 2010) and the social determinants of health framework (Marmot and Wilkinson, 2005). It posits that education is associated with a range of resources – wealth, knowledge, social networks etc. – that can be leveraged to avoid risks, cope with stressors, and invest in better health. According to this model, low education increases exposure to chronic stressors and poor living conditions, restricts coping resources, and limits access to healthcare, which in turn undermines health and increases the risk of mortality (Phelan et al., 2010).

Three key corollaries follow from this model. First, more preventable forms of mortality – such as deaths of despair, which stem directly from individual actions – should exhibit greater inequalities (Phelan et al., 2010). Second, greater socioeconomic disparities in health-promoting resources within a given society should result in correspondingly greater disparities in such preventable mortality (Gutin and Hummer, 2021). Third, cumulative disadvantage over the life course, in the form of prolonged and repeated exposure to stressors or resource deficits, should have disproportionately adverse health effects (Corna, 2013; Kuh et al., 2003). Given the centrality of education for status attainment, this implies that early educational setbacks can be particularly detrimental by triggering cascading and compounding sequences of adversity across multiple life domains.

The selection-model can be subdivided into direct health selection and indirect selection. The direct health selection-model draws on, *inter alia*, human capital theory in economics (Becker, 2007; Grossman, 1972). From this perspective, health is an input in the production of human capital, and poor health, especially in childhood and adolescence, undermines individuals’ ability to invest in and advance their human capital stock through education (Haas et al., 2011). A key implication is thus that direct health selection ought to be stronger for health conditions that disproportionately inhibit socioeconomic advancement. A defining characteristic of diseases of despair is that they are mental and behavioral phenomena that are regulated by the brain. In short, like most psychiatric conditions, they involve dysfunction and disturbance of core cognitive as well as non-cognitive (e.g., emotional stability, perseverance, conscientiousness, or self-regulation) abilities (Stein et al., 2021). Thus, they can be expected to be particularly impairing in tasks requiring both cognitive and non-cognitive skills, such as education. Accordingly, virtually all psychiatric conditions – including depression and addiction – are negatively associated with later educational attainment and achievement (Dalsgaard et al., 2020; Esch et al., 2014).

Models of indirect selection (or spurious correlation) frequently draw on sociogenomic findings concerning the heritability of both education and health, with twin and sibling studies showing that the association between education and health is substantially reduced when adjusting for shared genes (Lundborg et al. (2016), Behrman et al., 2011; but see Lundborg et al. (2016)). An implication of this is that indirect health selection ought to be stronger for health conditions with a greater genetic component. Moderate-to-high genetic influence has been established for most psychiatric conditions related to despair (Sullivan

et al., 2012), suggesting that genetic selection may be relevant for deaths of despair as well.

As argued by Montez and Friedman (2015), there is compelling evidence that both causation and selection are relevant, but that the strength of the respective processes varies depending on contextual factors. The focal contextual factor in much of the literature on deaths of despair, including in this study, is time: the *raison d'être* for this body of literature is that despair-related mortality, and disparities in this type of mortality, has increased over time. The key issue is then to identify the underlying drivers of these trends.

Research on trends in despair has to date almost invariably drawn on social causation types of explanations. Since disparities in resources are assumed to translate into corresponding disparities in preventable mortality, it follows that growing resource disparities – as has been observed in, for example, the United States and Sweden – should result in growing mortality disparities as well (Gutin and Hummer, 2021). In line with this, one strand of research has emphasized so-called demand-side factors (King et al., 2022), arguing that structural transformations have generated growing alienation, community dissolution, and ultimately despair, especially in less educated population segments (Case and Deaton, 2022). Support for this comes from studies establishing links between despair and economic precarity (Bjorklund, 2023; Knapp et al., 2019), income inequality and stalled social mobility (Benny et al., 2023; Kuo and Kawachi, 2023; Loverock et al., 2024), deindustrialization (Autor et al., 2019; Pierce and Schott, 2020; Venkataramani et al., 2020), and social disintegration (Glei et al., 2024). Another strand has emphasized so-called supply-side factors and argued that the most parsimonious explanation for the rise of overdoses is a greater supply of drugs, especially prescribed and illicit opioids (Currie and Schwandt, 2021; Masters et al., 2017; Ruhm, 2022). Support for this comes from studies showing links between opioid misuse and marketing or availability of prescription opioids (Alpert et al., 2021; Finkelstein et al., 2022), healthcare intensity (Lin et al., 2020), and physician prescription patterns (Barnett et al., 2017). Supply-side factors can be viewed as a subtype of the broader social causation model insofar as the less-educated are disproportionately affected *because of* their lower education. For instance, fundamental cause theory posits that the diffusion of novel medical innovations is stratified, such that higher socioeconomic strata are better equipped to access and adopt them (Phelan et al., 2010). Analogously, the diffusion of novel health risks, such as synthetic opioids, may be inversely stratified (Gutin and Hummer, 2021), with lower socioeconomic strata being more susceptible due to greater neighborhood exposure, lower health literacy or or inadequate access to relevant treatments.

Less, if any, research on trends in despair has been devoted to factors related to selection. In general, selection processes are posited to be relatively more important the more that attainment or status is dependent on the functioning, or lack thereof, that is inherent to specific health conditions (McLeod and Pavalko, 2008). Importantly, several societal trends have increased the social value and utility of cognitive and/or non-cognitive abilities (Edin et al., 2022; Jokela et al., 2017) and thus made educational and socioeconomic success more contingent on precisely those abilities that are most acutely impaired by diseases of despair. This could, in turn, have brought about a stronger selection into both low education or socioeconomic status and despair. For instance, skill-biased technological change, the decline of lower skilled jobs, and the emergence of knowledge economies have increased the economic returns to both cognitive and non-cognitive abilities (Weinberger, 2014). Conversely, low ability has become more disadvantageous, possibly amplifying the sorting of vulnerable youths into lower social strata. Likewise, educational expansion can generate compositional changes within educational strata, such that the less educated are a shrinking and increasingly negatively selected group in terms of the type of abilities that are both conducive to educational success and impaired by despair-related conditions (Montez and Bisesti, 2024). Relatedly, increased social mobility since the mid-20th century has amplified the

importance of such abilities for socioeconomic status, which can reinforce the negative selection into low education (Mackenbach, 2012). Lastly, increasing rates of assortative mating based on achieved rather than ascribed characteristics may result in a greater clustering of genetic and social disadvantages, further reinforcing the link between low educational achievement or attainment and health risks (Leesch et al., 2024; Torvik et al., 2024).

1.2. The Swedish setting

The present study followed students for eight years after graduation from compulsory school at age 16. Compulsory school is comprehensive, with no selection of students into different tracks or programs. The next educational stage is the three-year upper secondary school, consisting of specialized vocational or academic and university preparatory programs. Upper secondary school is optional but close to 100 % of students apply at least for the first year. Students' compulsory school grade point average (GPA) carries high stakes as it is the main instrument used to sort students into different upper secondary schools or programs. Students with insufficient grades has since 1998 been barred from enrolling in the regular upper secondary programs and are instead referred to remedial programs, but often fail to attain an upper secondary degree. Both compulsory and upper secondary school are free of charge and publicly funded, largely through municipal taxation. Since the early 1990s, the organization of both educational stages has also been decentralized, with an increasing number of students attending private voucher schools.

2. Data and methods

2.1. Data and participants

All Swedish students graduating from the last year of compulsory school (school year 9; approximately 16 years old) in 1992, 1993, 2009 and 2010 were included in the study and followed for 8 years after graduation (until approximately age 24). The selection of these graduation cohorts was motivated by documented differences in achievement-related disparities in all-cause and despair-related mortality, with students graduating in the early 1990s exhibiting relatively small disparities, whereas those graduating in the late 2000s exhibited larger disparities (Högberg et al., 2025). Comparing these cohorts thus allows an assessment of changes over time in the effect of low academic achievement on mortality.

Individual-level data were obtained from Swedish administrative registers, including the National Cause of Death Register (Brooke et al., 2017), the National Patient Register (Ludvigsson et al., 2011) and various registers containing educational and socioeconomic data. The sample size with complete data on academic achievement was 424,715, although this was reduced in models adjusting for covariates (see details in Supplementary file A). The data were made available for research by the Umeå SIMSAM Lab (Lindgren et al., 2016).

2.2. Outcomes: all-cause mortality and deaths of despair

All-cause mortality was measured as death from any cause occurring one to eight years after graduation – specifically 1993–2001 for the early (1992–1993) and 2010–2018 for the late (2009–2010) graduation cohort. Deaths of despair were identified using the following International Classification of Diseases (ICD) codes, in line with Case and Deaton's (2015) original approach.

- Suicide: ICD10: X60–X84, Y87.0; ICD9: E950–E959.
- Drug overdose (including alcohol poisoning): ICD10: X40–X45, Y10–Y15, Y45, Y47, Y49; ICD9: E850–E855, E858, E860, E980.0, E935, E937, E939.

- Alcoholic liver disease and cirrhosis: ICD10: K70, K73, K74; ICD9: 571.

Only the underlying cause of death was used.

2.3. Exposure: low academic achievement

Participants' achievement was assessed using their compulsory school GPA, and converted into percentile ranks specific to each graduation year to account for different grading systems between the early (1992–1993) and late (2009–2010) graduation cohorts. There was a strong curvilinear association between GPA and mortality, with the approximately lowest 20 % of students having disproportionately high mortality risks (see Fig. A1 in Supplementary file A). The percentiles were therefore grouped into two categories – low GPA (1st–20th percentiles) and medium/high GPA (21st–100th percentiles) – so as to focus on the most at-risk segment of the population.

2.4. Moderator: graduation cohort

Graduation years were used to measure changes over time, with the early graduation years (1992–1993) grouped into one category (henceforth: early graduation cohort) and the late graduation years (2009–2010) grouped into another (henceforth: late graduation cohort).

2.5. Selection model: confounders

Complex phenomena such as trends in mortality are multifactorial (Shanahan et al., 2019), and we sought to include a broad range of potential pre-graduation confounders to capture selection effects (RQ1).

- Variables related to the youths themselves: sex, country of birth, age, birth month, and pre-graduation hospitalization for any health condition and for any psychiatric disorder.
- Variables related to the youths' mothers and fathers: country of birth, educational attainment, unemployment, social assistance receipt, pre-tax employment earnings, post-tax disposable income, pre-graduation hospitalization for any health condition and for any psychiatric disorder, and death from any cause.
- Variables related to the youth's municipality of residence: population size, population change, population density, average disposable income, average education level, and employment rate.

The values of time-variant variables were measured and summarized or averaged over all (parental death), five (hospitalization) or three (all other variables) years preceding the year of graduation. It should be noted that race or ethnicity is not recorded in Swedish registers. While country of birth (immigrant status) may serve as a proxy for this, we cannot do justice to the importance of race in the literature on despair (e.g., Case and Deaton, 2015; Siddiqi et al., 2019).

2.6. Social causation model: mediators

The following variables were used to investigate social causation, that is, mediation by post-graduation socioeconomic disadvantages (RQ2).

- Variables related to the youths themselves: educational attainment, educational participation (e.g., university enrollment), parental co-residence status, employment status, unemployment, NEET-status (not in employment, education or training), receipt of social assistance, receipt of disability benefits, receipt of sick pay, pre-tax employment earnings, and post-tax disposable income.
- Variables related to the youth's municipality of residence: population size, population change, population density, average income, average education level, and employment rate.

In line with the emphasis on cumulative disadvantage in the social causation model of health inequalities, the mediators were summarized (categorical mediators) or averaged (continuous mediators) for all years from graduation until the year of the measurement of the outcome.

Detailed information on the exact measurement of all variables, including summary statistics, is provided in Supplementary file A.

2.7. Statistical analysis

To study selection (RQ1), we fitted unadjusted and adjusted logistic regression models:

$$\ln\left(\frac{P(\text{Death}_{iy})}{1 - P(\text{Death}_{iy})}\right) = \beta_0 + \beta_1 \text{GPA}_i + \beta_2 C_i + \beta_3 (\text{GPA}_i \times C_i) + \beta_6 T_y \quad (\text{model 1a})$$

$$\ln\left(\frac{P(\text{Death}_{iy})}{1 - P(\text{Death}_{iy})}\right) = \beta_0 + \beta_1 \text{GPA}_i + \beta_2 C_i + \beta_3 (\text{GPA}_i \times C_i) + \beta_4 \bar{X}_i + \beta_5 (C_i \times \bar{X}_i) + \beta_6 T_y \quad (\text{model 1b})$$

Where $\ln\left(\frac{P(\text{Death}_{iy})}{1 - P(\text{Death}_{iy})}\right)$ is the natural logarithm of the odds of death for participant i in year y (1–8 years after graduation), β_1 is the main effect of achievement (GPA), β_2 is the main effect of graduation cohort and β_3 is the interaction between achievement and graduation cohort. The vector of confounders \bar{X}_i , averaged or summarized over pre-graduation years, was interacted with graduation cohort (β_5), thus allowing for heterogeneous selection effects across the cohorts. The focal parameter is β_3 , and expresses the differential effect of low achievement on mortality across graduation cohorts, without (model 1a) and with (model 1b) adjustment for cohort-specific selection effects. $\beta_6 T_y$ accounts for the baseline hazard function over time y (i.e., years since graduation). For brevity, and while acknowledging the strong assumptions required to infer causality from observational data, the adjusted association between achievement and mortality (β_1 and β_3) will in the remainder of the paper be referred to as an “effect”. In short, model 1b addresses RQ1 and accounts for selection effects by adjusting for pre-graduation factors as confounders.

To address RQ2, we used inverse odds ratio-weighting (IORW) for causal mediation analysis (Nguyen et al., 2015; Tchetgen Tchetgen, 2013). This is a non-parametric approach that compresses the information about the relationship between exposure and mediators, conditioned on confounders, into a set of weights. These weights are then used to estimate natural direct and indirect of the exposure by neutralizing all indirect pathways through the mediators to the outcome. Our application of IORW proceeded as follows. First, a logistic regression model for exposure (low achievement) given pre-graduation confounders and post-graduation mediators was fitted. The models were fitted separately for the early and late graduation cohort to allow for heterogenous confounding and mediating effects across cohorts:

$$\ln\left(\frac{P(\text{GPA}_i)}{1 - P(\text{GPA}_i)}\right) = \beta_0 + \beta_1 \bar{X}_i + \beta_2 \bar{M}_{iy} \quad (\text{model 2})$$

Where $\ln\left(\frac{P(\text{GPA}_i)}{1 - P(\text{GPA}_i)}\right)$ is the natural logarithm of the odds of low achievement (GPA) for participant i , \bar{X}_i again represents the vector of pre-graduation confounders for participant i , thus adjusting for selection effects, and \bar{M}_{iy} is the vector of post-graduation mediators for participant i in year y . Second, the inverse odds weights for both cohorts combined were computed as the inverse of the predicted odds from model 2 for the exposed (low achievement) group and set to 1 for the unexposed (medium/high achievement) group. Inverse odds weights were used in place of inverse odds ratio weights to improve efficiency (Nguyen et al.,

2015).

Third, the total effect, net of confounding by selection, was estimated as in model 1b; the natural direct effect (net of both selection and mediation by post-graduation socioeconomic disadvantages) by fitting a model equivalent to model 1b but weighted by the inverse odds weights derived from model 2; and the natural indirect effect of post-graduation socioeconomic disadvantages by subtracting the natural direct effect from the total effect. The natural direct effect is the effect of the exposure on the outcome if the mediators were held constant at the levels they would naturally take in the absence of the exposure. The natural indirect effect is the effect of the exposure on the outcome that operates through the mediators, by changing the mediators from the levels they would naturally take if the exposure were present to the levels they would naturally take if the exposure were absent, while holding the exposure constant. For brevity, direct and indirect effects will be used in place of natural direct and indirect effects.

Differences between odds ratios from models fitted on different samples or with different covariates capture both changes in residual variance across models and changes in the partial effects of the variables (so-called non-collapsibility), meaning that odds ratios cannot identify changes due to confounding or mediation separately from changes in the residual variance (Breen et al., 2018). The odds ratios from the logistic regression models were therefore transformed into average marginal effects, which are not affected by changes in the residual variance. Cluster-robust standard errors were used in all regression models to account for the dependence of repeated observations of the same individual, and bootstrapped standard errors (250 iterations) to derive standard errors of the natural indirect effects. Separate models were fitted for all-cause and despair-related mortality.

2.8. Supplementary and sensitivity analyses

Additional analyses were conducted to explore the robustness of the results. Linear probability models were used as an alternative way of accounting for the non-collapsibility of odds ratios across different models and samples (Breen et al., 2018). Additional models were also estimated with (i) a more conventional regression-based mediation analysis; (ii) the summarized or average measurement of the mediators employed in the main analyses replaced by yearly measurements; and (iii) additional confounders related to health at birth included, which were excluded from the main analyses since they were only available for native-born youths. Consequences of the operationalization of GPA were explored by (i) computing GPA percentiles separately for native- and foreign-born youths; and (ii) using alternative cutoff points to indicate low GPA. Consequences of the measurement of deaths of despair was investigated through the use of a broader indicator, including deaths due to any mental disorder (ICD10: F00–F99; ICD9: 290–319) and from injuries of undetermined intent (ICD10: Y16–Y34; ICD9: E980–989).

3. Results

Table 1 shows mortality depending on achievement and cohort. Given the large number of covariates (24 confounders and 19 mediators), summary statistics on all variables are presented in Supplementary file A. 1141 participants (0.27 % of the total sample) died during follow-up, 468 of whom died from despair-related causes. Risk ratios comparing low to medium/high-achieving youths were larger in the 2009–2010 than in the 1992–1993 cohort: 2.89 (0.53/0.18) vs. 2.15 (0.48/0.22) for all-cause mortality and 3.66 (0.31/0.08) vs. 2.42 (0.15/0.06) for despair-related mortality.

Fig. 1 presents the results of the selection models (models 1a and 1b), thus addressing RQ1. Full regression coefficients are presented in Table B1 in Supplementary file B. The degree of selection can be assessed by comparing the unadjusted (triangle-shaped markers) and adjusted (diamond-shaped markers) estimates. Focusing first on the two cohorts separately (top rows), the included confounders account for roughly half

Table 1

Number of deaths from all causes or despair depending on GPA and graduation cohort.

	Participants	All-cause mortality		Deaths of despair	
	N	N dead during follow-up	% dead during follow-up	N dead during follow-up	% dead during follow-up
<i>1992-1993 cohort</i>					
Low GPA	40,587	196	0.48 %	62	0.15 %
Medium/high GPA	153,377	344	0.22 %	97	0.06 %
<i>2009-2010 cohort</i>					
Low GPA	50,380	268	0.53 %	156	0.31 %
Medium/high GPA	180,747	333	0.18 %	153	0.08 %
Total	424,715	1141	0.27 %	468	0.11 %

Note. Only observations with complete data on confounders included. Abbreviations: GPA = grade point average.

(between 30 % and 61 %) of the association between low achievement and both all-cause (left panel) and despair-related (right panel) mortality, supporting the proposition of the selection model that low-achieving students are negatively selected in terms of health and health-related characteristics. However, the associations remain significant in both cohorts and the remaining half of the association is left unaccounted for. Note that despair-related mortality is rarer than all-cause mortality; hence the smaller coefficients with despair-related mortality as the outcome. The bottom rows show that the unadjusted interaction between achievement and cohort is positive and significant for both outcomes, indicating stronger associations between low achievement and mortality in the 2009–2010 cohort. Most importantly from the perspective of RQ1, adjustment for confounders does not reduce, and if anything slightly increases, the strength of the interaction, thus indicating that the stronger effects of low achievement in the later cohort cannot be explained by a stronger negative selection into the low achievement group.

Fig. 2 shows results of the weighted logistic (i.e., mediation) models, thus addressing RQ2. Note that “total effect” refers to models adjusted for all confounders, using only cases with complete data on all mediators; hence the slightly different effects compared to the adjusted models in Fig. 1. Starting with the results for the two cohorts separately (top rows), the direct effects are substantially smaller than the total effects. Thus, most of the effect of low achievement on all-cause and despair-related mortality within cohorts are due to indirect effects operating through the mediators. This supports the social causation model, positing that low achievements constrain access to health-promoting resources. The bottom rows display results for the interaction between achievement and cohort, and show that the direct effects of the interactions are smaller than the total effects. Thus, the stronger effects of low achievement on mortality in the later cohort can at least partly be accounted for by the combined influence of the mediators. This is in contrast with the selection models in Fig. 1, where adjustment for confounders resulted in somewhat larger interaction terms. The indirect effects are stronger for all-cause mortality, where they are significant and account for almost all of the total effect of the interaction term (88 %), than for despair-related mortality, where they are not significant and account for about half (51 %) of the total effect (see Table B3 in Supplementary file B for exact coefficients).

3.1. Sensitivity and supplementary analyses

The results are, with few exceptions, similar when measuring effects as odds ratios and using linear probability models (Supplementary file C); when using a more conventional, regression-based approach to analyze mediation (Supplementary file D); when measuring mediators

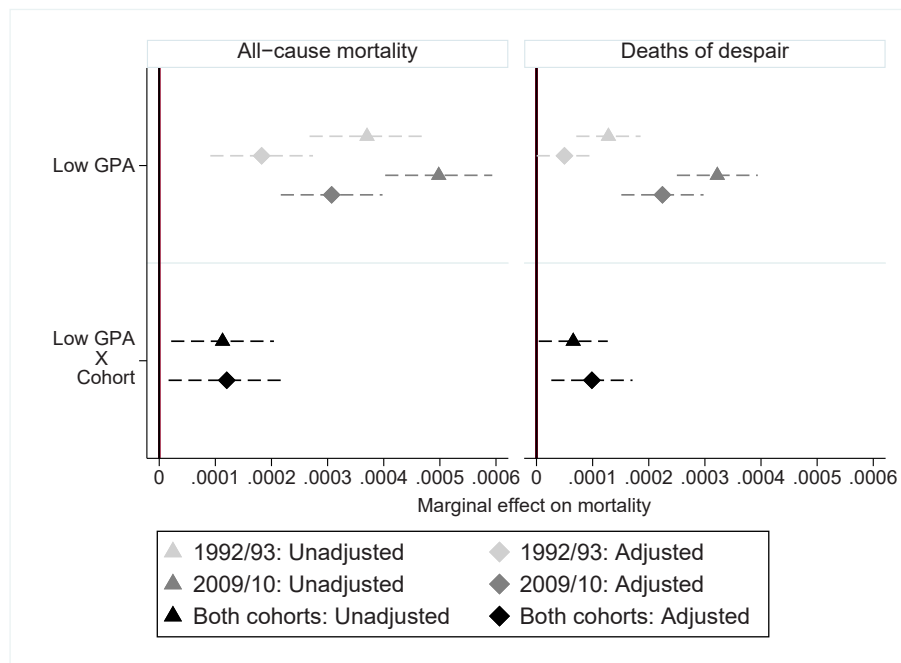


Fig. 1. Average marginal effects of graduation cohort and GPA with ACM or DoD as the outcomes. Selection models. Note. Figure shows average marginal effects calculated based on logistic regression models corresponding to models 1a and 1b. Abbreviations: GPA = grade point average. Horizontal dashed lines = 95 % confidence intervals. Unadjusted = sample restricted to observations with complete data on confounders (estimates from model 1a). Adjusted = model adjusted for confounders (estimates from model 1b). Low GPA X Cohort = interaction between low GPA and cohort.

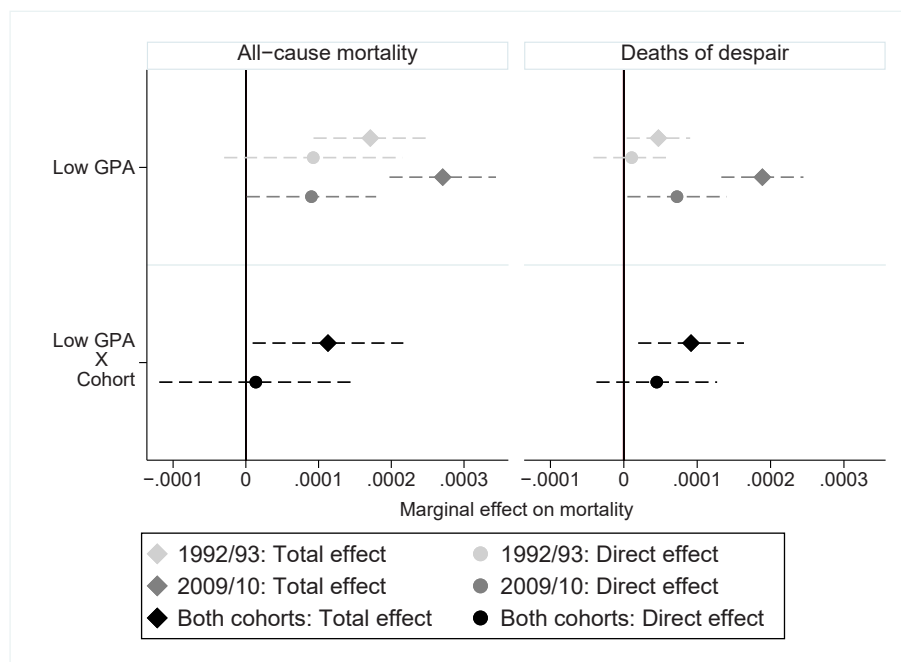


Fig. 2. Average marginal effects of graduation cohort and GPA with ACM or DoD as the outcomes. Mediation models. Note. Figure shows average marginal effects calculated based on logistic regression models. Abbreviations: GPA = grade point average. Horizontal dashed lines = 95 % confidence intervals. Total effect = adjusted for all confounders (model 1b) and conditional on availability of data on all mediators. Direct effect = adjusted for all confounders and mediators (using IORW). Low GPA X Cohort = interaction between low GPA and cohort.

yearly instead of summarizing or averaging them over the post-graduation period (Supplementary file E); when including additional confounders related to health at birth that were only available for native-born youths (Supplementary file F); when measuring low achievement only based on the grades of native-born youths and when using alternative cut-off points (10 and 30 lowest percentiles,

respectively) to define low achievement (Supplementary file G); and when using a broader definition of despair (Supplementary file H). The two exceptions are (i) results for foreign-born youths and (ii) results using the lowest 30 percentiles to define low achievement, which show that disparities did not change significantly over time.

4. Discussion

Against the backdrop of widening achievement-related disparities in all-cause and despair-related mortality in Swedish youths since the early 1990s, the present study investigated whether these disparities can be explained by changes in the selection into low achievement (RQ1), and if these disparities are mediated by changes in low-achieving youths' subsequent socioeconomic disadvantages, net of changes in selection (RQ2). To this end, we followed two cohorts of Swedish compulsory school graduates (graduating in 1992–1993 and 2009–2010, respectively), and compared mortality risks up to eight years after graduation.

As for RQ1, we found that roughly half of the association between low achievement and subsequent all-cause and despair-related mortality within each respective cohort was due to selection. However, the selection effect was largely similar in both cohorts and selection did not account for the stronger effect of low academic achievement on mortality in the later cohort. If anything, the larger achievement-related disparities in the later cohort became somewhat more pronounced when accounting for selection, at least for despair-related mortality. Selection was measured using indicators of pre-graduation health, socioeconomic status and demographic characteristics. In other words, neither stronger direct health selection (health causing achievement) nor stronger indirect selection (a third factor influencing both education and health), could explain the growing achievement-related disparities.

As for RQ2, we found that an even larger share – roughly two thirds – of the remaining association between low achievement and subsequent mortality within cohorts could be accounted for by the post-graduation mediators, meaning that most of the total effects of low achievement were due to indirect effects. Results concerning the growing achievement-related disparities were somewhat more mixed. The mediators accounted for almost all of the increased disparities in all-cause mortality and about half of the increased disparities in despair-related mortality, rendering both increases – that is, the direct effects of the interactions between achievement and cohort – statistically insignificant. On the other hand, the indirect effects of the mediators on the interactions between achievement and cohort were not themselves statistically significant, and most of the increased disparities in despair-related mortality remained unexplained. In keeping with the social causation model, mediation was measured using indicators of post-graduation cumulative socioeconomic disadvantages. In other words, the results indicate that low achievement to a greater extent constrains life-course prospects in a cumulative fashion, thereby amplifying its adverse health consequences.

These results have implications for the broader literature on health inequalities overall as well as for the more specific literature on temporal trends in mortality, particularly despair-related mortality. Beginning with (education-based) health inequalities, these are typically explained by two models: the social causation model and the selection model. The social causation model posits that lower achievement increases exposure to stressors and constrains access to health-promoting resources. Conversely, the selection model posits that individuals with poor health or predisposing characteristics face more difficulties in the education system (Grossman, 1972; Mackenbach, 2012; Montez and Friedman, 2015). All in all, we found clear support for both models in explaining the association between low achievement and mortality *within* the respective cohorts. However, to paraphrase Montez and Friedman (2015), instead of asking which model is “true” – it would be truly surprising if both were not at least partly true (McLeod and Pavalko, 2008) – it may be more fruitful to ask under which conditions, and to address which problems, the respective models are more or less applicable. The key social and historical “problem” motivating this study was the widening educational disparities in despair-related mortality, and to some extent all-cause mortality, observed in many high-income countries. Our results indicate that the selection model offers limited insights into this trend among Swedish youths, insofar as low-achieving youths in the later graduation cohort do not appear to

have been more negatively selected on characteristics that plausibly predict subsequent mortality. The social causation model, on the other hand, did have some explanatory power in that a marked share of the widening disparities could be attributed to stronger post-graduation socio-economic disadvantages.

As for temporal trends, the literature on deaths of despair has centred around demand- or supply-side perspectives (Bjorklund, 2023; King et al., 2022), both of which are subtypes of the broader social causation model but applied to the context of trends in despair-related mortality. Demand-side perspectives focus on structural transformations and socioeconomic factors, such as deindustrialization, economic precarity and inequality (Benny et al., 2023; Knapp et al., 2019; Kuo and Kawachi, 2023; Loverock et al., 2024; Pierce and Schott, 2020; Venkataramani et al., 2020), while supply-side perspectives stress the importance of changes in the price and availability of lethal drugs, especially opioids (Barnett et al., 2017; Finkelstein et al., 2022; Lin et al., 2020; Masters et al., 2017; Ruhm, 2022).

Our results partly align with the demand-side perspective, in that post-graduation socioeconomic disadvantages explained much of the educational disparities within cohorts as well as of the increase in these disparities over time. In the North American context, the demand-side perspective has focused on broad, macro-level transformations, particularly related to the labor market. Analogous, but weaker (Oesch and Piccitto, 2019), transformations have occurred in the Swedish labor market, with a decline in routine jobs combined with a more intense competition for the remaining jobs from a more highly educated workforce (Tählén and Westerman, 2020). Our results suggest that low-achieving youths find it more difficult to access this increasingly knowledge-intensive labor market, which may result in despondency, despair and ultimately death. The North American literature has paid less, if any, attention to changes in the education system, possibly because of its focus on midlife mortality. From the perspective of youths and school graduates, however, the education system is essential. The later cohort experienced a more decentralized and marketized Swedish education system, that had furthermore been subject to austerity cuts following the economic crisis of the early 1990s. They were exposed to greater segregation and inequalities between schools (SOU, 2019), which may have contributed to exacerbating the adverse consequences of low achievement later in life. The arguably most consequential changes, however, concern the higher demands for enrollment in, and graduation from, upper secondary school, which were introduced in the mid- and late 1990s. Since these reforms, 20–30 % of Swedish students in the bottom third of the compulsory school achievement distribution have been barred from accessing regular upper secondary school altogether, and 40–50 % have failed to graduate from it (SOU, 2019). Previous Swedish research show that these reforms negatively affected incomes and employment prospects of affected students (Diamond and Persson, 2016; Halapuu, 2021), and that students who do not graduate have higher all-cause mortality and suicide risks later in life (Döring et al., 2021).

One caveat is in order, however. While the indirect effects were moderately strong for all-cause mortality, they were more modest for despair-related mortality, at least in relation to the changing effects of low achievement over time. Thus, demand-side factors appear to be relatively less consequential for despair-related mortality than for other forms of mortality. This lack of specificity of the proposed mechanism raises doubts concerning the veracity of the demand-side perspective. A possible reason is that variations in both suicides (many of which involve drugs) and accidental overdoses are highly sensitive to the availability of drugs, in line with the supply-side perspective. Lacking detailed data on the specific drugs involved in overdoses, as well as data on drug accessibility (e.g., prescription patterns), our results do not directly speak to the importance of supply-side factors. Previous and more detailed analyses of death certificates show an increase in overdoses involving synthetic opioids such as fentanyl during the early 2010s (Auer et al., 2021). It should also be noted that overdoses

constitute a larger share of both all-cause and despair-related mortality in the late compared to the early cohort (see Table A3 in Supplementary file A).

4.1. Limitations

Estimation of causal effects, and *à fortiori*, natural direct and indirect effects, using observational data requires strong assumptions. Unbiased estimates of the effects of low achievement requires that all variables that confound the effect of achievement on mortality (i.e., direct and indirect selection) are observed, while unbiased estimates of natural direct and indirect effects in addition requires no confounding of the effect of achievement on the mediators or of the effects of the mediators on mortality (Nguyen et al., 2015). Furthermore, unbiased estimates also require that the exposure, confounders and mediators are measured without bias.

It is unlikely that the individual-level data at hand, though arguably more comprehensive than in most prior studies, satisfy these assumptions exactly. For instance, only data on inpatient care were available for both cohorts, and these were only available from 1987, that is, five years before the early cohort graduated. If psychiatric conditions that were treated in outpatient or primary care influenced achievement, or if earlier health conditions had long-term effects, selection effects may be underestimated. Relatedly, data restriction precluded inclusion of regional income inequality and race or ethnicity as confounders, which, since these have been linked to despair-related mortality (Auger et al., 2009; Case and Deaton, 2015), may lead to an underestimation of selection effects.

Relatedly, data restrictions precluded inclusion of regional income inequality which may lead to an underestimation of selection effects (Auger et al., 2009). Likewise, while we adjusted for country of birth as a proxy for ethnicity, this was a crude measure that only distinguished between native and immigrant background without accounting for demographic shifts within the immigrant population. The share of the total youth population with an immigrant background increased from 19 % in the 1992–1993 cohort to 26 % in the 2009–2010 cohort, while the immigrant share of the low-achieving group increased from 24 % to 34 %. Meanwhile, according to official statistics, the share of immigrants with a non-Western background (i.e., outside Europe, North America, or Australasia) also increased. Thus, over the study period, the immigrant population not only increased in size but also became more ethnically diverse and more low achieving, all of which may have influenced the link between academic achievement and health outcomes.

Lastly, though we operationalized deaths of despair in line with Case and Deaton's (2015) original work, we did not have data on additional alcohol-related causes of death (e.g., alcoholic cardiomyopathy) that have been used in subsequent research (Augarde et al., 2022; Camacho et al., 2024). Additionally, we only had data on underlying causes of death, likely resulting in an undercount of drug- and alcohol-related deaths compared to broader definitions including contributory causes. For instance, Durkin et al. (2010) found that alcohol was listed as a contributory cause in almost 9.5 % of all external causes of death in Northern Ireland. Similarly, Spark et al. (2023) found that despair-related mortality was 41 % higher when contributory causes were included, mainly because of a doubling of alcohol-attributable mortality. Selection effects may be underestimated insofar as selection processes are stronger for these additional alcohol-related or contributory causes.

5. Conclusions

This study examined the drivers of the growing achievement-related disparities in all-cause and despair-related mortality across cohorts of Swedish compulsory school graduates. It found that while both pre-graduation selection into low achievement and post-graduation socioeconomic disadvantages accounted for much of the association between

low achievement and mortality within cohorts, only post-graduation socioeconomic disadvantages could partly explain the increasing disparities over time. These findings suggest that low achievement in school has become more consequential for socioeconomic prospects later in life, thereby increasing its impact on access to health-promoting resources and exposure to socioeconomic stressors.

Much of the widening disparities in both all-cause and despair-related mortality remained unexplained, however. Future research should investigate additional pathways, such as psychological stressors, while incorporating detailed individual-level data on drug-use and accessibility so as to assess the relative importance of demand- and supply-side factors. Moreover, while adolescence and youth have largely been a blind spot in the literature on deaths of despair, life-course epidemiology suggests that these may be sensitive periods for the development of later health inequalities (Wadsworth, 1997), not least considering the critical transition from school to work. Our findings thus underscore the importance of considering education system characteristics, as well as the interplay between school and labor market dynamics from a life-course perspective, in research on trends in health inequalities and despair-related mortality. From a theoretical perspective, the findings reassert the call to move beyond the static selection-versus-causation-binary in the context of health inequalities (Montez and Friedman, 2015). A more fruitful approach is to identify the specific circumstances under which each model is more relevant or useful. Finally, from a policy perspective, the findings emphasize the need to lower barriers in the education system and to create viable educational and employment pathways for low-achieving students in an increasingly knowledge-intensive labor market.

CRediT authorship contribution statement

Björn Högberg: Writing – review & editing, Writing – original draft, Validation, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Formal analysis, Data curation, Conceptualization. **Simone Scarpa:** Writing – review & editing, Validation, Supervision, Methodology.

Ethics approval

All data analyses were performed in compliance with Swedish law and institutional guidelines. The use of data for this project was approved by the Swedish Ethical Review Authority (Dnr 2023-03999-01 and Dnr 2023-05360-02).

Declaration of interest statement and financial disclosure statement

The authors have no relevant financial or non-financial interests to disclose.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at <https://doi.org/10.1016/j.socscimed.2025.118130>.

Data availability

The authors do not have permission to share data.

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