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Directions for improving the policy relevance of inequality research: Lessons from social origin–specific health disparities

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ABSTRACT

This study examines how social science research can more effectively inform policy interventions aimed at reducing social inequalities. Focusing on health disparities by social origin, it identifies three directions: reformulating theoretical frameworks in terms of hypothetical interventions, strengthening the alignment between theory and analytical strategies, and employing methods that simulate the effects of interventions. I reformulate previous theories to derive three testable hypotheses concerning the equalization of educational attainment and returns to education across social origin groups. To evaluate these hypotheses, I analyze data from the Survey of Health, Ageing, and Retirement in Europe (SHARE) using a recently proposed random equalization decomposition approach. I compare the results of this method with those produced by conventional approaches—specifically, the product and difference methods—and demonstrate that only the random equalization decomposition provides quantities appropriate for testing intervention-based hypotheses. The results suggest that equalizing higher education rates across social origin groups consistently reduces health inequality for both women and men, and across all assessed age and cohort groups. Equalizing returns to higher education across social origin groups tends to increase rather than mitigate health inequalities, although these results are less conclusive. The study concludes by discussing further avenues to enhance the policy relevance of inequality research.

1. Introduction

How can the social sciences more effectively inform policies to reduce inequalities? Tackling this question is highly important because political commentators frequently criticise policymakers for failing to adequately address these disparities (e.g., Booth, 2024). At the same time, dating back to Engels' (1845) early quantitative work on social inequality, there is a long-standing research tradition in the social sciences, that has focused on explaining inequalities.

As the explanation of social inequalities lies at the very heart of social science research and remains a matter of public concern, social scientists should be uniquely positioned to inform policymakers about the most promising targets for intervention. However, recent research suggests that certain prevailing research practices constrain the ability of social science studies to generate such information (Jackson and VanderWeele, 2018; Lundberg et al., 2021). This study aims to explore pathways through which social science research can more effectively inform potential policy interventions to reduce inequalities.

To explore this issue, I examine the illustrative case of research investigating the mechanisms linking social origin—specifically, the resources of the family of origin—to health outcomes in later life. This field of research is selected because the COVID-19 pandemic has

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renewed public and academic interest in health inequalities (Kratz, 2021). The following section provides a brief review of research on how social origin influences health outcomes later in life. I identify three key avenues for improvement: (1) reformulating theoretical frameworks in terms of hypothetical interventions; (2) strengthening the connection between theoretical propositions and analytical strategies; and (3) employing recently developed methods that simulate the impact of hypothetical interventions.

To demonstrate how these steps can be implemented, I focus on self-rated health as an outcome and educational attainment as the main intervening mediating mechanism. I reformulate research questions and hypotheses explicitly in terms of hypothetical interventions. I then outline the identification assumptions under which these questions can be addressed empirically and discuss a non-parametric decomposition method (Yu and Elwert, 2025) designed to evaluate such interventions.

I use data from the Survey of Health, Ageing, and Retirement in Europe (SHARE), and compare findings obtained using the non-parametric decomposition method with those generated by conventional analytical techniques. The results demonstrate that the new method more effectively addresses the proposed research questions and hypotheses framed around hypothetical interventions. The study concludes by acknowledging its limitations and outlining directions for future research that can further bridge the gap between social science research and policy development.

2. Previous research on the mechanisms linking social origin to health

The explanation of origin-specific health disparities has received increasing attention in the scholarly debate (see Supplementary Table S1 for an overview). This literature review focuses on studies that highlight the role of social origin in shaping later-life health, specifically those that treat social origin as the main explanatory factor, examine at least one health outcome, and assess the mechanisms underlying this relationship. Measures related to social origin encompass socioeconomic conditions in childhood (Aartsen et al., 2019; VanderLinden et al., 2018; Landös et al., 2018; Pavela and Latham, 2016), childhood socioeconomic adversities (Montez and Hayward, 2014), childhood exposures (Kemp et al., 2022; Morton and Ferraro, 2020), early life disadvantage (Ferraro et al., 2016; O'Rand and Hamil-Luker, 2005), and childhood misfortune (Mustillo et al., 2021).

Health outcomes comprise cognitive functioning and cognitive decline (Aartsen et al., 2019), frailty (VanderLinden et al., 2018), disability trajectories (Landös et al., 2018), cancer onset (VanderLinden et al., 2018), trajectories of chronic conditions and multimorbidity (Pavela and Latham, 2016), the development of new health problems during adulthood (Ferraro et al., 2016), risk of heart attack (O'Rand and Hamil-Luker, 2005), chronic inflammation and mortality (Morton and Ferraro, 2020), obesity (Mustillo et al., 2021), and osteoarthritis and rheumatoid arthritis (Kemp et al., 2022).

Fig. 1 shows the common theoretical grounds of this literature. The aim of these studies often lies in adjudicating between the following three theoretical approaches: the sensitive period, the social trajectory, and the cumulative exposure model (Berkman, 2009; Kuh et al., 2003). While the sensitive period model implies that the link between social origin and health in old age does not arise due to social conditions during adulthood, the cumulative exposure model assumes that adult social conditions partly explain the association, and the social trajectory model posits that the association arises solely due to social conditions during adulthood.

To adjudicate between the sensitive period model, the social trajectory model, and the cumulative exposure model, previous research mainly relies on two methodological approaches (see Supplementary Table S1). The difference method appears to be the dominant approach in the literature, followed by the product method. The difference method uses a set of hierarchically nested regressions. Typically, Model 1 estimates the coefficient of a measure related to social origin. Subsequent models include (sets of) mediators that capture adult conditions, such as socioeconomic status and various risk behaviors, in the regression analyses. If the effect of social origin remains significant after including these intervening mediating mechanisms, researchers interpret this pattern as empirical support for the sensitive period model. If the effect is rendered non-significant, researchers conclude that the social trajectory model holds. If the effect is reduced but does not fully vanish, this pattern is interpreted as support for the cumulative exposure model.

The product method assesses indirect effects by estimating a regression model from social origin to specific mediators (X-M paths) followed by a model that estimates the association between these mediators and the health outcome of interest (M-Y paths). These paths are then multiplied to obtain indirect effects. Whether these indirect effects are significant can be tested by using the delta method (Sobel, 1987) or bootstrapping routines (Bollen and Stine, 1990). Direct effects are obtained by regressing the health outcome on the exposure while adjusting for all mediators. These analyses are frequently carried out using a structural equation framework in combination with the Mplus software (Muthén et al., 2017).[1]

3. Directions for improving the policy relevance

In what follows, I propose three pathways through which existing research practices can more effectively inform potential policy interventions aimed at reducing inequalities. First, there is a need to reformulate theoretical frameworks in terms of hypothetical interventions. Much of the literature on the sensitive period model has employed diverse labels—such as “independent association”

[1] While studies relying on the product method calculate significance tests for the indirect effects, studies relying on the most dominant approach in the literature (the difference approach) often neglect significance tests to assess whether the effect reductions (indirect effects) when (sets of) mediators are included in the regression framework are actually significant. As Gelman and Stern (2006) noted, underscored by Mustillo et al. (2018:1281), such differences (i.e., changes in statistical significance) may not be significant. As a consequence, such studies may erroneously conclude that either the social trajectory model or the cumulative exposure model holds, although the intervening mediating mechanisms are neither significant nor relevant.

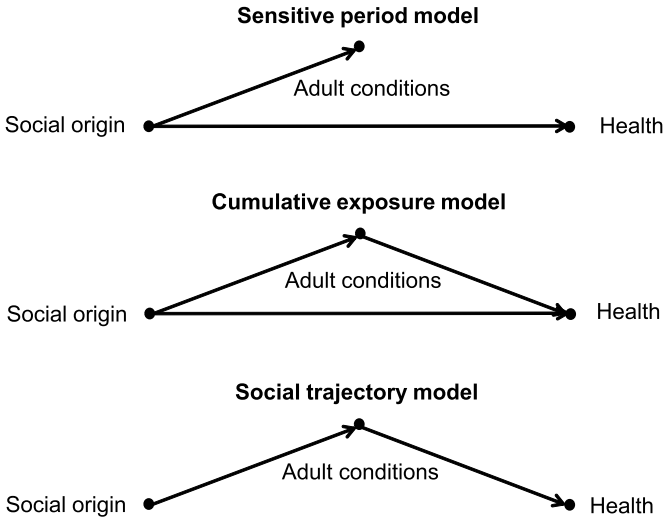

Fig. 1. The common theoretical approach of previous studies.

(Brandt et al., 2012), "lasting influence" (Pavela and Latham, 2016), "extraordinary long-term consequences not ameliorated by adult life circumstances" (Blackwell et al., 2001:1280), "long arm" (Hayward and Gorman, 2004; Pakpahan et al., 2017), "enduring effect" (O'Rand and Hamil-Luker, 2005; Schafer et al., 2011), "long-lasting effects" (Mazzonna, 2014), and "early imprints" (Ferraro et al., 2016).[2] While these terms capture important theoretical intuitions, they stop short of specifying the hypothetical interventions that would equalize the mediating mechanisms under study. As a consequence, existing results provide limited leverage for assessing the likely effectiveness of intervention strategies. Advancing the field therefore requires theoretical frameworks that explicitly connect hypotheses to well-defined intervention scenarios.

Second, the link between theoretical propositions and analytical approaches requires strengthening. Current formulations of the sensitive period, social trajectory, and cumulative exposure models remain ambiguous about which intervening mediating mechanisms should be equalized, and how, to align with the assumptions of each model. This lack of clarity hampers the comparability of empirical findings: for example, a study with only a few measures of adult life circumstances may conclude in favor of the sensitive period model, while another with a broader set of indicators may interpret its results as supporting the social trajectory model. Moreover, when interaction effects between social origin and adult conditions are present, even studies relying on the same mediators may reach conflicting conclusions because they apply different strategies for equalization. Future progress will benefit from theoretical models that more clearly connect their assumptions to the analytical practices required for their evaluation.

Third, the theoretical assumptions underlying the causal processes that link social origin to mediators—and ultimately to health—are often left implicit, even though clarifying them reveals analytical challenges that conventional methods cannot adequately address. Consider the example illustrated in Fig. 2A: when assessing how education contributes to the association between social origin and health in later life, controlling for educational attainment opens a backdoor path through childhood health, since childhood health affects both education (Basch, 2011; Case et al., 2002; Haas, 2006; Haas and Fosse, 2008; Forrest et al., 2013) and later-life health (Case et al., 2005; Pakpahan et al., 2017).[3] Consequently, failure to adequately control for childhood health will result in biased estimates of both the indirect effect through educational attainment and the remaining direct effect.

As shown in Fig. 2B, the situation becomes even more complex because social origin also influences childhood health (Currie, 2009; Chen et al., 2002). This structure—where a variable influences both the mediator and the outcome while being affected by the exposure—is known as exposure- or treatment-induced mediator–outcome confounding (VanderWeele et al., 2014; Wodtke and Zhou, 2020; Wodtke et al., 2020). As I will detail in Section 8.1, conventional approaches such as the difference method cannot accommodate such processes. To advance the field, research must therefore make explicit the assumed causal pathways linking social origin to mediators and health outcomes, clarify how different mechanisms relate to each other, and adopt analytical strategies capable of addressing the specific challenges posed by these causal structures.

4. Theory: equalizing education

Individuals' social origin is a powerful predictor of educational attainment (see, e.g., Jackson, 2013). These origin-based inequalities in educational opportunities have long-term implications: educational attainment is strongly associated with health outcomes in later life. Importantly, existing research suggests that this association is not solely driven by health-based selection

[2] See also Bhatta et al. (2018) for an earlier review.
[3] See Acharya, Blackwell, and Sen (2016); Imai et al. (2010); VanderWeele (2015) for further discussion of this type of bias.

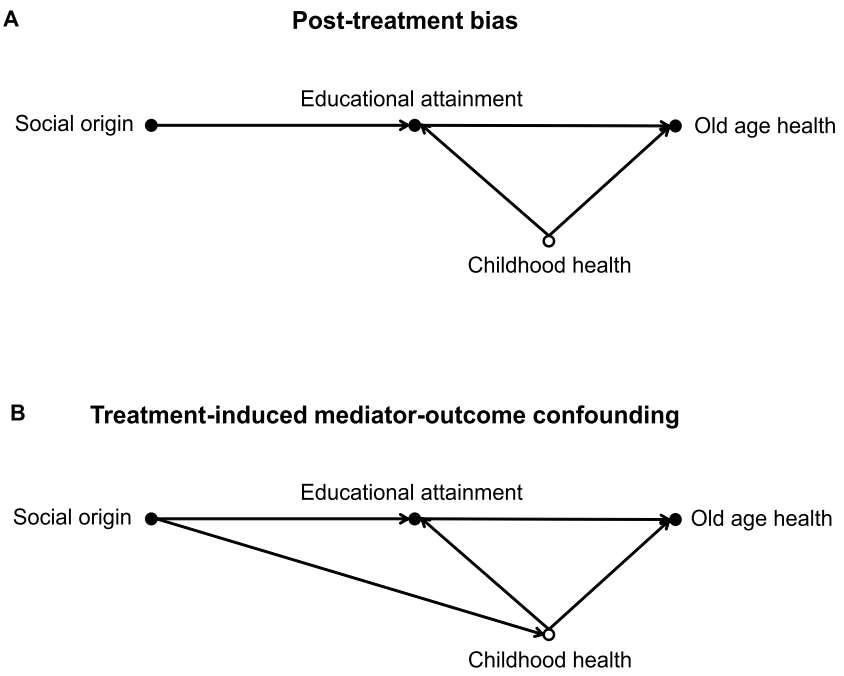

Fig. 2. Post-treatment bias and treatment-induced mediator-outcome confounding
Notes: Arrows order variables in time and show possible direct causal effects. Black dots indicate observed variables while empty circles indicate unobserved variables. Post-treatment bias arises when the identification strategy does not adjust for a common cause of educational attainment and health in old age. Treatment-induced mediator-outcome confounding arises when the identification strategy does not partialize out a common cause of educational attainment and health in old age that is affected by social origin.

mechanisms (e.g., Haas, 2006), but also reflects a causal influence of education on health (e.g., Davies et al., 2018). This body of research underpins the assumption that equalizing higher education attainment rates across social origin groups would reduce health inequalities.

H1. (Prevalence Hypothesis): Equalizing higher education attainment rates across social origin groups would reduce health inequalities.

Whether equalizing returns to education across social origin groups would narrow or widen origin-based health disparities is less clear. Theories of resource multiplication and added protection suggest that individuals from socioeconomically advantaged backgrounds benefit more from higher education than their disadvantaged peers (Ross and Mirowsky, 2011; Schafer et al., 2013). These frameworks argue that the advantages of education are magnified when combined with other forms of capital—such as well-connected social networks, cultural familiarity with institutional environments, and access to elite educational institutions. From this perspective, education serves to amplify pre-existing social advantages, reinforcing rather than narrowing disparities. In this context, equalizing educational attainment alone may not be sufficient to reduce health inequalities, unless the differential returns to education across social origin groups are also addressed.

This argument reflects a broader theoretical tradition arguing that individuals from high-status backgrounds can extract more value from their educational credentials (Ross and Mirowsky, 2006, 2011). Similarly, the theory of added protection (Schafer et al., 2013) suggests that education is more effective in promoting health when it is bolstered by supportive early-life environments. If such interactions are present, then origin-specific health disparities may become especially pronounced among those who attain high levels of education, as individuals from lower social origins may lack the non-material resources needed to fully capitalize on their educational attainment.

H2. (Resources-*as*-Amplifier Hypothesis): Equalizing the returns to higher education across social origin groups would reduce health inequalities.

In contrast to this perspective, theories of resource substitution and compensatory leveling offer a more optimistic outlook on the equalizing potential of education. According to this view, individuals from disadvantaged social origins benefit more from educational attainment because education compensates for the lack of parental resources and early-life advantages (Ross and Mirowsky, 2011; Schafer et al., 2013). For instance, if health-relevant knowledge and behaviors are transmitted either through parental investment or through formal education, then education becomes particularly valuable for those who lacked parental support. In this framework, educational attainment serves as a substitute for the advantages typically conferred by one's family of origin, allowing individuals to overcome structural disadvantages and achieve better health outcomes.

This logic is supported by empirical findings showing that individuals who attain higher education “against all odds” often

experience especially strong labor market returns (Brand and Xie, 2010). These findings reinforce the notion that the marginal value of education may be greatest among the socially disadvantaged, for whom it serves as a critical compensatory mechanism. In this scenario, equalizing the returns to higher education across social origin groups would not mitigate but rather increase health inequalities.

H3. (Resources-*as*-Equalizer Hypothesis): Equalizing the returns to higher education across social origin groups would increase health inequalities.

5. Current study

This study aims to examine how social science research can more effectively inform policy interventions aimed at reducing inequalities. Focusing on health inequalities by social origin, I reviewed the predominant theoretical approaches—the sensitive period, cumulative exposure, and social trajectory models—and show that they are not formulated in terms of hypothetical interventions. To address this gap, I reformulated the hypotheses explicitly around such interventions. Specifically, the Prevalence Hypothesis (H1) posits that equalizing higher education rates would reduce origin-based health gaps in later life, while the Resources-*as*-Amplifier Hypothesis (H2) and Resources-*as*-Equalizer Hypothesis (H3) offer competing expectations about whether equalizing returns to education would widen or narrow these gaps.

To test these hypotheses, I apply a random equalization decomposition approach, which generates estimable quantities that map directly onto the effects of these hypothetical interventions. Furthermore, this approach is well suited to deal with the challenges posed by the assumed causal processes underlying origin-related health inequalities. I compare the results of this approach with those obtained from the difference and product methods. The comparison shows that conventional methods do not yield quantities that allow for direct tests of the reformulated hypotheses. Moreover, they are not equipped to adequately address the role of childhood health when assessing how educational attainment contributes to the link between social origin and later-life health.

6. Data and measures

6.1. Data

This study uses SHARE data from waves 1 to 6 (for further information see Börsch-Supan et al., 2013) because of its favorable properties, making it the most frequently used data set for assessing origin-specific health disparities in old age (see Supplementary Table S1). SHARE is a multidisciplinary, biennial, cross-national panel survey on aging. The survey collects data on health and socioeconomic status and is representative for the national population of 50 and older. Retrospective life histories provide information on the previous life course including childhood conditions and parental characteristics. This retrospective data allows researchers to trace the pathways from social origin to health in old age.

I use data from 15 countries (Austria, Belgium, Czech Republic, Denmark, Estonia, France, Germany, Israel, Italy, Luxembourg, Netherlands, Slovenia, Spain, Sweden, and Switzerland). Cross-national comparability is ensured by harmonized questionnaire development and standardized fieldwork practices. I selected countries and respondents based on wave 5 participation, as this wave contains essential information on parental background and childhood conditions. Furthermore, I focus on non-institutionalized individuals and limit the analysis to the first observation for each respondent to define the study population in a balanced way.

Migrants may pose challenges for the estimation because educational attainment is often difficult to compare across countries of origin and destination (Markides and Eschbach, 2005). At the same time, migrants constitute an important segment of contemporary societies, and examining the underlying mechanisms is therefore of substantive interest for this group as well. To balance these considerations, I conduct the main analysis excluding the 5215 migrants from the sample and provide additional analyses in which migrants are included in the sample and examined separately. After removing cases with missing information on the variables of interest, the main analytical sample—excluding migrants—consists of 43,691 individuals. Descriptive statistics are presented in Table 1.

6.2. Measures

The outcome variable is self-rated health on a five-point scale ranging from 1 (*poor*) to 5 (*excellent*). Respondents were also asked how they perceived their health between the ages 0 and 15 using the same scale. I use this retrospective information to capture childhood health. Self-rated health is widely used in the literature on social determinants of health as it captures the multidimensionality of health and predicts mortality (Idler and Benyamini, 1997; Jylhä, 2009). I assume an underlying response continuum when reporting and therefore employ self-rated health as a continuous outcome variable. Research on health outcomes suggests that self-reported health conditions correlate highly with more objective health measures (Ferraro and Farmer, 1999). Furthermore, Willson and colleagues (2007) argued that self-rated health measures capture differences in health between groups independent of age.

Table 1
Summary statistics (SHARE).

	N	%	Mean	SD	Min.	Max.
Social origin						
High or medium	29,578	67.70				
Low	14,113	32.30				
Gender						
male	19,658	44.99				
female	24,033	55.01				
Education						
Primary/secondary	33,663	77.05				
Tertiary	10,028	22.95				
Birth cohort groups						
1927-1939	10,129	23.18				
1940-1945	8438	19.31				
1946-1953	13,998	32.04				
1954-1963	11,126	25.47				
Country						
Austria	3397	7.78				
Germany	3441	7.88				
Sweden	3024	6.92				
Netherlands	1929	4.42				
Spain	4073	9.32				
Italy	3526	8.07				
France	3262	7.47				
Denmark	3135	7.18				
Switzerland	2196	5.03				
Belgium	3995	9.14				
Israel	717	1.64				
Czech Republic	4396	10.06				
Luxembourg	648	1.48				
Slovenia	2166	4.96				
Estonia	3786	8.67				
Old age health			3.02	1.07	1.00	5.00
Childhood health			3.81	1.05	1.00	5.00
Age of respondent			62.56	8.52	50	84

Note: The analysis sample comprises a total of 43,691 individuals.

I generated a social origin index based on three domains—parental education, financial position, and housing situation—that mark parental socioeconomic conditions during childhood (Kratz and Bristle, 2019; Mazzonna, 2014). The indicator on parental education states the highest maternal or paternal educational attainment according to the International Standard Classification of Education (UNESCO, 2006) with six categories. Financial position is a binary measure indicating whether the family had few financial resources before age 15.[4] The housing indicator can be considered a wealth measure and is constructed by dividing the rooms available by the number of household members at age 10. I use principal component analysis to construct the social origin index, as it efficiently combines multiple indicators into a single measure that captures their shared variance without relying on arbitrary weighting.[5] Following Mazzonna (2014), I centered all indicators on the country mean value to adjust for the 15 country samples and to keep the relative positions within countries. I then obtained the social origin index from the first component of the PCA.

To facilitate interpretation, I then dichotomized the variable into low and high social origin. Consistent with most of the previous literature (see Supplementary Table S1), I construct the social origin index to measure disadvantaged social origin and dichotomize it as follows. I estimated terciles for the social origin variable and coded individuals as having a disadvantaged origin if they fall in the lowest tercile, with those in the middle and highest terciles forming the reference group.

Individual educational attainment is measured by the International Standard Classification of Education (ISCED-97). The six ISCED categories were divided into "primary or secondary" and "tertiary" education, where primary or secondary refers to no education, pre-primary, basic, lower secondary, upper secondary, as well as post-secondary non-tertiary education; tertiary refers to the first stage of tertiary education or higher attainment.

Further control variables include age, sex, country fixed effects, and cohort groups. Cohorts are grouped according to historical periods: those born between 1927 and 1939 (pre–World War II), 1940 to 1945 (during World War II), 1946 to 1953 (post-war period), and 1954 to 1963 (the Iron Curtain era, overlapping with the baby boom in some countries).

[4] Wave 5 contains a relative measure asking if the family has been "pretty well-off financially, about average, or poor" from birth to age 15. Wave 3 contains information on financial hardship up to a specific age. I combined information from both waves to construct a dummy indicator stating few financial resources if wave 5 respondents reported a poor financial situation or wave 3 respondents reported a period of financial hardship before the age of 15.

[5] Supplementary Section S2 provides further information.

7. Analytical strategy

I use a recently developed random equalization decomposition approach (Yu and Elwert, 2025), which is particularly well suited for testing the effectiveness of the formulated hypothetical interventions. Traditional decomposition techniques, such as the Kitagawa-Blinder-Oaxaca (KBO) decomposition (Blinder, 1973; Kitagawa, 1955; Oaxaca, 1973), do not support evaluation of hypothetical interventions (Lundberg et al., 2021; Opacic et al., 2025). In contrast, causal mediation analysis (VanderWeele, 2015) and random equalization decomposition approaches (Jackson and VanderWeele, 2018; Jackson, 2021; Lundberg, 2024; Opacic et al., 2025) enable simulation of hypothetical interventions to explain social inequalities.

I choose a random equalization decomposition approach for two main reasons. First, such approaches do not require thought experiments that manipulate social origin. Although such hypothetical interventions can provide valuable insights (Klein and Kühhirt, 2021; Kratz et al., 2022), they are often hardly practically implementable and therefore offer limited immediate policy relevance. Second, random equalization decomposition approaches rest on weaker unconfoundedness assumptions. The causal mediation framework—framing estimands in terms of the direct and indirect effects of social origin—requires that no unobserved confounders affect either the relationship between social origin and old-age health (U_1 in Fig. 3A) or the relationship between education and old-age health (U_2 in Fig. 3A).[6] By comparison, random equalization decomposition approaches require only that there are no unobserved confounders of the education–old-age health relationship (Fig. 3B).

To address these issues, scholars have proposed random equalization decomposition approaches that treat social origin as a demographic marker rather than a manipulable variable. This framework does not involve thought experiments to manipulate social origin. Furthermore, one must only assume that there are no unobserved confounders between educational attainment and old-age health (U2 in Fig. 3A). Among random equalization decomposition approaches, I select the method proposed by Yu and Elwert (2025) because it yields empirical quantities that allow us to disentangle the effectiveness of the previously formulated hypothetical interventions. In what follows, I adopt their notation.

Consider a binary variable $D_i \in \{0, 1\}$, where $D_i = 1$ indicates that individual i has attained higher education, and $D_i = 0$ indicates that they have not. Let Y_i^0 and Y_i^1 denote the potential old age health under the hypothetical states $D_i = 0$ and $D_i = 1$, respectively. The individual-level effect is defined as

$$\tau_i := Y_i^1 - Y_i^0$$

Assume that the population is partitioned into two mutually exclusive groups, $G_i = g \in \{a, b\}$, where group a denotes individuals from high social origin and group b denotes individuals from low social origin. I use the subscript g to denote group-specific expectations, for example:

$$E_g(Y_i) := E(Y_i \mid g).$$

For simplicity, I omit the subscript i hereafter. Assuming that the observed outcome Y satisfies the Stable Unit Treatment Value Assumption (SUTVA, Rubin, 1980):

$$Y = DY^1 + (1 - D)Y^0,$$

the observed health disparity between low and high social origin groups can be decomposed into four components (Yu and Elwert, 2025):

$$E_a(Y) - E_b(Y) = \underbrace{E_a(Y^0) - E_b(Y^0)}_{Baseline} + \underbrace{E_b(\tau)[E_a(D) - E_b(D)]}_{Prevalence} + \underbrace{E_a(D)[E_a(\tau) - E_b(\tau)]}_{Effect} + \underbrace{CoV_a(D, \tau) - CoV_b(D, \tau)}_{Selection}$$

The prevalence component directly tests the prevalence hypothesis by assessing how much equalizing higher education rates across social origin groups would reduce origin-based health disparities in later life. The effect component helps adjudicate between the resources-*as*-amplifier and resources-*as*-equalizer hypotheses by simulating how equalizing origin-specific differences in the impact of higher education on health would alter origin-based health disparities. The decomposition also produces two additional empirical quantities—the baseline and the selection components—that are less relevant for the present study but may be highly relevant in other contexts.

The selection component captures the contribution of differential selection into higher education between individuals from low and high social origins. If individuals from low and high social origins sort differently into higher education based on their returns to education, this component accounts for a substantial share of the health disparities between origin groups. Finally, the baseline component reflects the difference in health between individuals from low and high social origins after educational attainment has been equalized. It captures the origin-based disparity that is not attributable to differences in higher education attainment—that is, the residual disparity.

Identification of these components relies on two assumptions: conditional ignorability (or unconfoundedness)—requiring no

[6] While controlled direct effects require these assumptions, natural direct and indirect effects additionally require that no unmeasured confounders affect the relationship between social origin and education (see, e.g., VanderWeele, 2015).

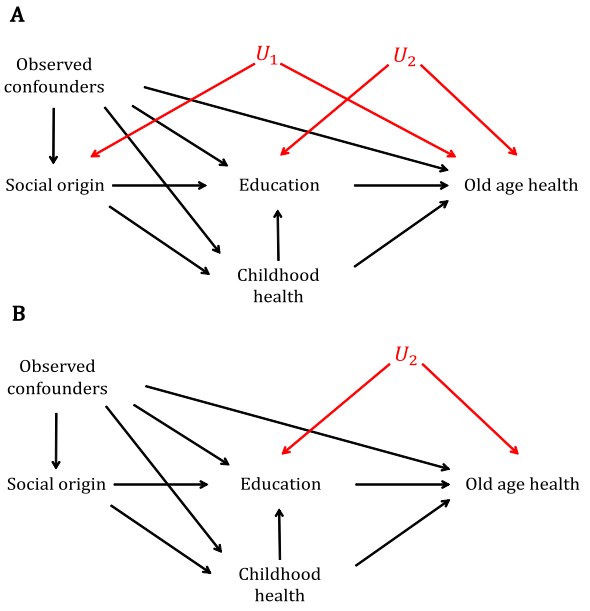

Fig. 3. No-unmeasured confounding assumptions
Notes: Arrows order variables in time and indicate possible direct causal effects. U_1 and U_2 represent unobserved confounders of the social origin-old age health and education–old-age health relationships.

unobserved confounders of the relationship between higher education and old-age health (Fig. 3B)—and overlap (or positivity)—ensuring that all individuals have a non-zero probability of attaining each education level. To provide some evidence that the overlap assumption may not be particularly problematic in the present case, Supplementary Fig. S1 shows the number of individuals who attained low and high levels of education in the advantaged and disadvantaged groups across different values of the covariates.

Estimating the decomposition requires fitting different nuisance functions.[7] Yu and Elwert (2025) recommend flexible machine learning methods with cross-fitting to reduce reliance on parametric assumptions. Accordingly, I estimate the nuisance functions using a generalized boosted models (GBM) algorithm. Under a specific set of assumptions—outlined in detail by Yu and Elwert (2025)—the resulting estimates are doubly robust, $\sqrt{n}$-consistent, asymptotically normal, and semi-parametrically efficient. The analyses were conducted in R version 4.4.2 with the cdgd package (Yu, 2024).

8. Results

The results section is structured as follows. I begin with an illustrative analysis showing how previous research has used the difference and product methods to adjudicate between the cumulative exposure, social trajectory, and sensitive period models. I then test the Prevalence Hypothesis (H1), the Resources-*as*-Amplifier Hypothesis (H2a), and the Resources-*as*-Equalizer Hypothesis (H2b) using the previously discussed random equalization decomposition approach. Finally, I examine whether the results of this approach vary by sex, cohort, and age group.

8.1. Conventional approaches

Fig. 4 summarizes the results of the difference method. The initial regression estimates the total origin-specific health disparity; subsequently, childhood health is added. Upon inclusion of childhood health, which is significant at the 5% level, the total disparity is reduced by 15%. A Sobel-Goodman test confirms that this reduction is statistically significant at the 5% level (ß = −0.0342, SE = 0.0020). In the next step, educational attainment is added. The effect of educational attainment is significant at the 5% level, leading to a further reduction of the health disparity by 22%. Again, a Sobel-Goodman test confirms the significance of this reduction (ß = −0.0441, SE = 0.0021). These results would be interpreted as consistent with the cumulative exposure model: the total social origin–health gap is significantly reduced when education is controlled for, yet a significant disparity remains after controlling for educational attainment (ß = −0.1529, SE = 0.0102).

Upon closer inspection, however, it becomes clear that this method leaves unresolved how childhood health should be handled when assessing the indirect effect of educational attainment. In this approach, I assess the reduction in effect from a model that already includes childhood health. This procedure estimates the additional mediating role of educational attainment once childhood health has

[7] See Yu and Elwert, 2025 for further details.

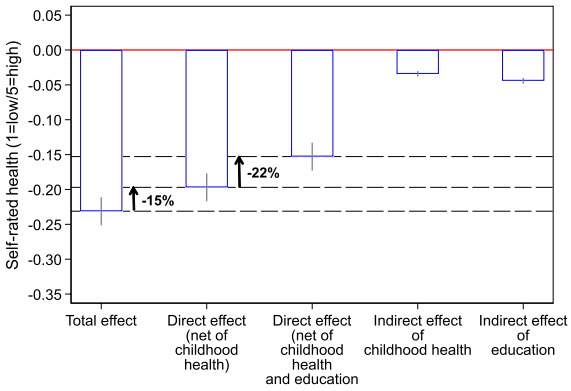

Fig. 4. Difference method (i.e., hierarchical regression procedure) to explain origin-specific health disparities
Notes: Control variables included in every model: gender, age, cohort groups and country fixed effects. Indirect effects are calculated using Sobel-Goodman tests. Supplementary Table S3 shows estimation details.
Source: SHARE Wave 1–6 release 6.1.0, own calculations

been accounted for. However, it does not isolate the indirect effect through educational attainment because part of the total effect that operates only through childhood health—but not through education—is also controlled away. If we include childhood health, we may inadvertently remove a portion of the total effect that operates through childhood health but not through education. If we exclude childhood health, we risk biased estimates of the effect of education, so the indirect effect of education may partly capture the mediating role of childhood health. Thus, when using the difference method to assess the mediating role of education, we cannot adequately account for childhood health. We are trapped: Bias arises whether childhood health is included in the regression or not. This analysis demonstrates that the difference method cannot address treatment-induced mediator–outcome confounding.

Furthermore, the method is unclear about which hypothetical intervention it implies. How, exactly, is educational attainment equalized? As previously outlined, social origin and education may interact in shaping health in old age, and depending on the specific intervention imagined, the results may differ. In sum, the results relying on the difference method have three shortcomings: ambiguity about the hypothetical intervention and thus the theoretical estimand, inability to address treatment-induced mediator–outcome confounding, and failure to account for interaction effects between social origin and mediating mechanisms.

Fig. 5 shows the results of the product method. All paths are statistically significant: social origin is significantly related to childhood health and educational attainment and also affects health "directly". Furthermore, both childhood health and educational attainment are significantly related to health in old age. Indirect effects suggest that the relationship between social origin and health in old age is mediated by childhood health (ß = −0.0334, SE = 0.0020) and by educational attainment (ß = −0.0450, SE = 0.0021). As with the difference method, these findings would be interpreted as consistent with the cumulative exposure model: part of the effect of social origin on health in old age is mediated by childhood health and part by educational attainment. When both pathways are considered, a significant direct effect remains. However, as with the difference method, it remains unclear how these coefficients relate to hypothetical interventions, and neither treatment-induced mediator–outcome confounding nor interaction effects are accounted for.

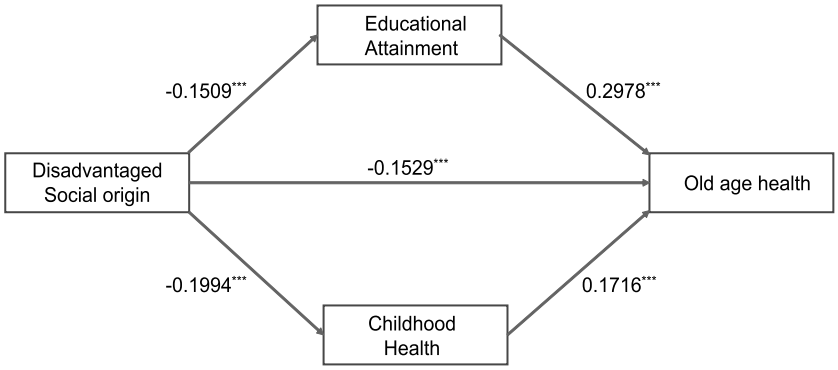

Fig. 5. Product method to explain origin-specific health disparities
Note: Control variables included in every model comprise gender and age. Supplementary Table S4 shows estimation details.
Source: SHARE Wave 1–6 release 6.1.0, own calculations.

8.2. Non-parametric decomposition results

Fig. 6 shows the results of the random equalization decomposition approach. To what extent would equalizing higher education rates across social origin groups reduce total health disparities? The prevalence component is negative and statistically significant, amounting to −0.043 (95% CI: −0.047, −0.038), which accounts for a substantial portion of the total disparity in self-rated health. Consequently, equalizing higher education rates between social origin groups would reduce the origin–health gap by roughly 17%. This intervention would decrease social inequalities in self-rated health and promote greater health equity. These results are consistent with the prevalence hypothesis (H1).

To what extent would equalizing returns to higher education across social origin groups reduce health disparities? The effect component is positive, not statistically significant, and equals 0.005 (95% CI: −0.002, 0.012). Consequently, equalizing returns to higher education between social origin groups would lead to a slight increase in disparities in self-rated health. This intervention would marginally widen social inequalities in self-rated health and reduce health equity. If anything, these results are rather consistent with the resources-as-equalizer hypothesis (H2b).

8.3. Heterogeneity analyses

To what extent would equalizing higher education rates across social origin groups reduce total health disparities among women and men? As shown in Supplementary Fig. S2, the prevalence component is negative and statistically significant for both sexes, amounting to −0.042 (95% CI: −0.048, −0.036) for women and −0.042 (95% CI: −0.048, −0.035) for men. This accounts for a meaningful share of the total origin–health gap, suggesting that equalizing higher education rates would reduce disparities in self-rated health for both groups. By contrast, the effect component is not statistically significant for women (0.000, 95% CI: −0.008, 0.009) and only slightly positive for men (0.014, 95% CI: 0.003, 0.024). These findings imply that equalizing returns to education would not reduce health disparities for women and would modestly increase them for men.

How do these patterns vary across birth cohorts? Supplementary Fig. S3 shows that for the 1927–1939, 1940–1945, and 1946–1953 cohorts, the prevalence component is consistently negative and statistically significant (ranging from −0.036 to −0.041), indicating that equalizing higher education rates across social origins would reduce disparities in self-rated health. For these cohorts, the effect component is small and not statistically significant, suggesting no clear evidence that equalizing returns would reduce disparities. However, for the youngest cohort (1954–1963), the effect component is positive and statistically significant (0.032, 95% CI: 0.011, 0.053), implying that equalizing returns to education would increase origin-specific health disparities in this group.

How do results vary by age? According to Supplementary Fig. S4, the prevalence component is negative and statistically significant across all age groups: −0.043 (95% CI: −0.050, −0.037) for ages 50–59, −0.040 (95% CI: −0.048, −0.033) for ages 60–69, and −0.035 (95% CI: −0.044, −0.026) for ages 70–84. This indicates that equalizing higher education rates reduces health disparities in all age groups, though the effect weakens somewhat in older adults. By contrast, the effect component is positive and statistically significant for the youngest group (0.023, 95% CI: 0.007, 0.038), but negligible and not statistically significant for older groups. This suggests that equalizing returns to education would increase health disparities among those aged 50–59 but would have little effect among older adults.

Do the results remain robust when individuals born abroad are included, and are similar mechanisms observed for this migrant group? As shown in Supplementary Fig. S5, the prevalence component is negative and statistically significant in both subsamples. For the sample in which migrants are not excluded, the prevalence component amounts to −0.046 (95% CI: −0.050, −0.042), while for the migrant-only subsample it is even larger in magnitude at −0.087 (95% CI: −0.107, −0.066). These estimates account for a meaningful

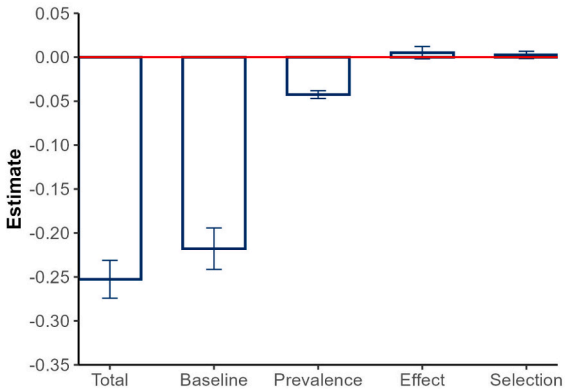

Fig. 6. Decomposition estimates of self-rated health.
Notes: Nuisance function models are estimated using gradient boosting machines. Error bars represent 95% confidence intervals. More detailed results are provided in Table S5.
Source: SHARE Wave 1–6 release 6.1.0, own calculations.

share of the total origin–health gap and suggest that equalizing higher education rates would substantially reduce disparities in self-rated health in both samples. The effect component is not statistically significant when migrants are not excluded—0.004 (95% CI: −0.003, 0.011)—and remains statistically indistinguishable from zero in the migrant-only sample—-0.009 (95% CI: −0.033, 0.015).

These findings imply that equalizing returns to education would not reduce origin-related health disparities in either subsample. In sum, the results are qualitatively similar whether migrants are included or excluded, yet they are more conservative when migrants are excluded. This pattern arises because the overall origin–health gap is more pronounced among migrants, and equalizing higher education rates would reduce this gap more strongly for migrants than for the non-migrant population.

Taken together, the subgroup analyses reveal a consistent pattern: the prevalence component is negative and statistically significant across sex, cohorts, and age groups, indicating that equalizing higher education rates reduces origin-specific health disparities. By contrast, the effect component is generally small and often not significant, but where it does matter—among men, the youngest cohort (1954–1963), and adults aged 50–59—it is positive, suggesting that equalizing returns to education would increase rather than reduce disparities.

9. Conclusion and discussion

This study examined how social inequality research can be better aligned with potential policy interventions. Using the case of origin-specific health disparities, I identified three directions: reformulating theoretical propositions as hypothetical interventions, strengthening the link between theory and analytical strategy, and employing methods that simulate intervention effects. To this end, I reinterpreted theories through a causal inference lens and tested the resulting hypotheses with SHARE data. The analysis showed that the random equalization decomposition approach produces quantities that correspond to hypothetical interventions, whereas conventional difference and product methods do not.

9.1. Summary of the results

Both the difference and product methods show that educational attainment is an important channel through which social origin affects health in old age, and that—after accounting for this mechanism—a significant direct effect of social origin on later-life health remains. Furthermore, results from the random equalization decomposition imply that equalizing higher education rates across social origin groups would reduce origin-specific health disparities, whereas equalizing returns to higher education may increase disparities in specific subgroups. Equalizing higher education rates would decrease health inequality across women, men, non-migrants, migrants, and all assessed age and cohort groups, while equalizing returns to education would increase inequality among men, the youngest age group, and the earliest-born cohort.

9.2. Theoretical implications

This study demonstrates that focusing on adjudicating between the sensitive period, social trajectory, and cumulative exposure models does not directly address hypothetical interventions. Thus, reinterpreting classical theories through a causal inference lens may be beneficial. For instance, this study reformulated resource substitution and resource multiplication theories to derive competing hypotheses about whether equalizing returns to education would increase or decrease origin-specific health disparities. Consistent with the Resources-*as*-Equalizer Hypothesis (H3), the results suggest that equalizing returns may increase such inequalities in specific subgroups. This pattern also corroborates sociological theories of resource substitution (Ross and Mirowsky, 2006, 2011) and compensatory leveling (Schafer et al., 2013), as well as the finding that individuals least likely to obtain a higher education degree profit most from their education (Brand and Xie, 2010).

9.3. Implications for the connection between theory and methodology

This study highlights the dilemma of adjudicating between the sensitive period, social trajectory, and cumulative exposure models. Conditioning on different sets of mediators or adopting alternative interventions to equalize them can lead to divergent conclusions about which model is supported. Yet these models are not explicit about which mediating mechanisms should be held constant—or how—to align with their theoretical assumptions. The findings illustrate this ambiguity: while equalizing higher education rates reduces origin-specific health disparities, equalizing returns to education tends to increase them for specific subgroups. These results underscore the need for theoretical models to specify assumptions about the effectiveness of hypothetical interventions and for analytical strategies to be explicitly designed to test such interventions.

9.4. Methodological implications

This study emphasizes that testing the effectiveness of hypothetical interventions to reduce social inequalities requires explicit assumptions about the underlying causal process. In the example at hand, scholars must be precise about how social origin affects mediators, how mediators interrelate, and how both ultimately affect health. Clarifying these assumptions guides covariate selection and makes transparent the no-unmeasured-confounding conditions required to test the effectiveness of hypothetical interventions.

Moreover, testing such interventions requires consideration of possible interaction effects. In this case, social origin and education interact in shaping health in later life. Such effects are consistent with a broad body of sociological literature showing that parental

characteristics and individual resources jointly shape later-life outcomes (Andersson, 2016; Andersson and Vaughan, 2017; Bauldry, 2014; Cheng et al., 2021; Kratz et al., 2022; Ross and Mirowsky, 2011; Zhou, 2024). This study demonstrates that, while the product and difference methods are not well suited to test hypothetical interventions when mediators are interrelated or when interaction effects exist, the random equalization decomposition approach applied here can address both challenges.

9.5. Policy implications

The results imply that equalizing higher education attainment across social origin groups would reduce health inequalities. If anything, they also point to the possibility that individuals from low social origins may benefit more from higher education than their high-origin counterparts in certain subgroups. Taken together, these findings suggest that affirmative action policies aimed at closing gaps in higher education attainment are likely to mitigate health inequalities later in life, whereas policies designed to increase the returns to higher education for low-origin groups may be less urgent.

9.6. Limitations and future research

Several limitations should be acknowledged. While the overlap assumption appears to be less problematic in our case, the results rely on the assumption of conditional ignorability (or unconfoundedness) and therefore do not eliminate concerns about unobserved confounding. Future research should build on this study by incorporating additional confounders of the education–old-age–health relationship to ensure that the results presented here are not affected by violations of the conditional ignorability assumption. Promising candidates include further parental characteristics, birth order, genetic predispositions, and personality traits, as well as childhood and adolescent risk behaviors and environmental risk factors.

Furthermore, endogenous selection bias may threaten the validity of the conclusions. Such bias may occur if respondents with low levels of social origin or education and simultaneously poor health in later life are less likely to complete the questionnaire, more likely to drop out of the survey, or more likely to die before the age of 50, when data collection began. Future research should use multiple imputation procedures and inverse probability weighting to assess robustness of the results when accounting for such forms of endogenous selection bias.

Another limitation is that social origin and high levels of education might influence how individuals evaluate their own health (Fforde et al., 2024). Those with low social origin and low education may overreport self-rated health compared to individuals with higher social origin or education, potentially biasing results. Future research should therefore complement self-reported outcomes with more objective measures of health in later life.

Recall bias may also affect reports of childhood health status. Individuals with different levels of education, social origin, and later-life health may recall their early health to varying degrees, potentially introducing systematic bias. Prospective data would help address this concern. Additionally, dichotomizing variables such as social origin (low vs. high) and educational attainment (higher education vs. no higher education) oversimplifies both the scientific findings and their policy implications. Future research should move beyond this binarization and examine additional contrasts in social origin and educational attainment.

Innovative approaches to construct measures of social origin may be employed (Mustillo et al., 2021), and further intervening mediating mechanisms deserve attention. For instance, social origin may affect risk behaviors, labor market processes, and family formation, all of which may contribute to origin-specific health disparities in old age. Such innovations in measurement should be combined with clarity about the hypothetical intervention being emulated to equalize the mediating mechanisms.

Finally, this study discusses how one promising random equalization decomposition approach allows for testing hypothetical interventions. There are also other random equalization decomposition approaches, such as the gap-closing estimand (Lundberg, 2024) or the approach of Opacic et al. (2025), that allow assessments of the effectiveness of clearly specified interventions. Systematically applying these analytical approaches could greatly expand our understanding of how social disparities evolve and help inform policymakers about effective ways to reduce (health) inequalities.

Data Note

The data used in this publication were made available by the Munich Research Institute for the Economics of Aging and SHARE Analyses (MEA-SHARE gGmbH). The SHARE data can be requested here: https://share-eric.eu/data/contact.

Replication files (Stata do-files and R code) are publicly available on the OSF project (Kratz, 2026): https://osf.io/vn825/.

Declaration of generative AI and AI-assisted technologies in the writing process

During the preparation of this work, the author used DeepL and ChatGPT in order to detect spelling and grammatical errors. After using these tools, the author reviewed and edited the content as needed and take full responsibility for the content of the publication.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.ssresearch.2026.103322.

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