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**COVER PAGE****Title**

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# Adolescent mental health and behavioural problems, and intergenerational social mobility: a decomposition of health selection effects

## Abstract

According to the health selection hypothesis, poor mental health and behavioural problems in adolescence limit socioeconomic attainment. But to what extent is health selection driven by prior social causation? This paper quantifies the extent to which health selection – here, restricted or downward intergenerational social class mobility due to poor mental health or behavioural problems – can be attributed to the influence of modifiable childhood risk factors. The UK National Child Development Study provides measures of socioeconomic deprivation and childhood adversities, as well as multiple-informant ratings of adolescents' mental health and behavioural problems, for which confirmatory factor analysis supports a three-factor model. Decomposition analysis is used to robustly assess the extent of attenuation of selection effects when conditioning on the childhood environment. Conduct problems, hyperactivity, and to a lesser extent emotional symptoms at age 16 are associated with individuals' chances of achieving (un)desirable mobility outcomes. When prior childhood risk factors are taken into account, the association of conduct problems with mobility is attenuated by around 50%, indicating a substantial role for confounding and earlier processes of social causation in the generation of this health selection effect. Further analyses indicate that this attenuation is greater for those from the most disadvantaged backgrounds and is mostly driven by the inclusion of indicators of generalised disadvantage such as crowded housing and low income. On the other hand, the effects of emotional symptoms and hyperactivity on mobility outcomes are not significantly accounted for by childhood risk factors. This study adds to the health inequalities literature by interrogating the empirical validity of the usual interpretation of health selection effects as indicating the causal priority of the onset of poor mental health.

## Keywords

UK; mental health; health selection; social causation; health inequality; adolescent mental health; social mobility; childhood environment

## 1. Introduction

Inequalities in health outcomes are ubiquitous, and this is true of mental as well as physical health. Explanations usually focus on the mechanisms of health selection and social causation (Dohrenwend et al., 1992; Miech, Caspi, Moffitt, Wright, & Silva, 1999; Power, Stansfeld, Matthews, Manor, & Hope, 2002). The social causation hypothesis holds that low socioeconomic status entails greater exposure to factors which negatively influence mental health. In short, SES affects health. According to the health selection hypothesis, mental health contributes to the sorting of individuals into SES positions, whether by influencing educational attainment or labour market outcomes. In this case, health affects SES. A recent formulation states that '[the health selection] hypothesis gives causal priority to the onset of mental illness as a factor preceding the disadvantaged placement of individuals into socioeconomic positions or social classes' (Muntaner, Ng, Vanroelen, Christ, & Eaton, 2013: 219). Another is that 'men and women with pre-existing illness drift down the social scale' (Power et al., 2002: 1989).

To what extent is health selection driven by prior social causation? This paper attempts to quantify the extent to which health selection – here, restricted or downward intergenerational social class mobility due to poor mental health or behavioural problems – can be attributed to the influence of modifiable (that is, at least partially amenable to social policy) childhood risk factors. These risk factors for poor mental health and restricted mobility – here, socioeconomic deprivation and adversities – may account for an apparent health selection effect in two ways (figure 1). The first is that adolescent mental health (AMH) may mediate the effect of childhood factors on SES in young adulthood. This reflects social causation occurring during childhood, and is therefore referred to as

*mediated social causation*. The second is that childhood factors may confound the relationship between adolescent mental health and SES in young adulthood. Despite similarly attributing causal priority to childhood environmental factors in accounting for the social gradient in mental health, this is referred to in the literature as the ‘indirect health selection’ hypothesis (DiStefano & Morgan, 2014; Wilkinson, 1986). This paper evaluates the joint contribution of these processes, but does not disentangle the two.

Health selection may occur both between and within generations. This paper focuses on intergenerational health selection. That is, when poor mental health restricts the intergenerational social mobility of an individual: her mental health prevents her from achieving the social class position relative to her parents’ which she otherwise would have attained. There is much reason to expect such selection: most mental disorders begin in adolescence and young adulthood, with half of all lifetime cases having onset by age 14 (de Girolamo, Dagani, Purcell, Cocchi, & McGorry, 2012; Kessler et al., 2005), and poor mental health in adolescence interferes with education in particular and the status attainment process in general (Miech et al., 1999; Power et al., 2002; Schoon, Sacker, & Bartley, 2003).

In sum, this paper examines the following two questions. First, are health selection effects upon intergenerational social mobility evident? Second, if health selection effects are evident, to what extent is health selection operating in the sense that poor AMH is independent of any measured risk factors, and to what extent, alternatively, does it simply represent mediation of, or confounding by, these risk factors?

## **2. SES and health: the importance of the transition to adulthood**

The reality of the health selection and social causation theories of health inequality is likely that they are best viewed as intertwined, with reciprocal causation occurring between SES and health over the life course (Kröger, Pakpahan, & Hoffmann, 2015; Mulatu & Schooler, 2002). The observation that social causation may precede and underlie health selection may therefore appear trivial, since *this* social causation may itself be driven by earlier health selection. However, though this picture of reciprocal causation may be accurate during adulthood, it is unlikely to apply during childhood and adolescence.

The most distinctive feature of the transition to adulthood is the change from economic dependence on parents or guardians, to economic dependence on oneself or one's partner. During childhood, since living conditions are determined by one's parents, there is no scope for health selection to occur, save in particularly severe cases in which a child's poor mental health affects parental SES. Schoon, Sacker & Bartley test this assumption on both the 1958 and 1970 British cohorts: 'In both cohorts the child's behaviour had no significant influence on family social circumstances' (2003: 1010). The first opportunity for health selection to occur is at first entry into the labour market as an economically independent adult. There is, however, scope for social causation to occur within childhood, for instance through parental SES and associated childhood adversities (CAs) (figure 2).

Put another way, the dependence of offspring on their parents postpones the possibility of health selection: the individual's SES does not become an outcome in its own right until late adolescence. On the other hand, one's mental health is an outcome constantly and immediately susceptible to influence. Parental SES is 'shielded' from poor mental health in childhood; mental health in childhood is not shielded from parental SES. Intergenerational health selection is therefore especially plausible because AMH, heretofore subject to social causation without consequences for one's SES, becomes, upon the transition to adulthood, consequential for market situation for the first time. While it may affect intermediate status attainment outcomes such as educational

achievement in school (e.g. Turney & McLanahan, 2015), these do not yet translate into an impact on children's living conditions, so there are no *reciprocal* effects between SES and health before the transition to adulthood. This implies that – at least within the child's generation – the association between mental health before the transition to adulthood and SES is not attributable to health selection. This limits the possible explanations of the attenuation of any apparent health selection effects once the childhood environment is controlled for. Mediated social causation – social causation that has occurred during childhood – and confounding (or 'indirect health selection') are the two possible explanations.

### 3. Previous work

The childhood environment is predictive of mental health in childhood and adolescence, though associations are stronger and more consistent for externalising problems, such as conduct problems and hyperactivity (Amone-P'Olak, Burger, Huisman, Oldehinkel, & Ormel, 2011; Bolger, Patterson, Thompson, & Kupersmidt, 1995). This body of research includes the natural experiment upon which Costello et al. (2003) capitalise, finding that an exogenous increase in income was associated with a substantial reduction in conduct and oppositional defiant disorders, while no effect on anxiety or depression was evident. This suggests that if health selection effects are evident, the extent to which they are accounted for by the childhood environment may vary with the dimension of AMH considered. While it is well-established that parenting behaviours and parent-child relationship quality account for much of the association between parental SES and AMH (Conger, Ge, Elder, Lorenz, & Simons, 1994; Leve et al., 2016), the mechanisms through which the childhood environment may differentially influence dimensions of AMH are less well understood.

It has consistently been found that measures of adolescent mental health or behavioural problems are associated both with SES in adulthood (A. Goodman, Joyce, & Smith, 2011; Kawakami et al.,

2012; Schoon et al., 2003) and with educational attainment (Breslau, Lane, Sampson, & Kessler, 2008; Kessler, Foster, Saunders, & Stang, 1995; J. McLeod & Fettes, 2007; J. D. McLeod, Uemura, & Rohrman, 2012), a key mediator in the status attainment process.

There is less consensus on the relative importance of different dimensions of adolescent mental health. A priori, externalising problems are especially likely to interfere with educational and labour market success because these characteristics conflict with the usual demands for success in these environments, such as obedience, conscientiousness, emotion regulation, and concentration. Moreover, the often highly-visible behaviours constituting externalising disorders may induce labelling effects whereby teachers gain negative impressions of a student's ability and come to hold and communicate lower expectations of them (J. McLeod & Fettes, 2007). Using the Dunedin Multidisciplinary Health and Development Study, Miech et al. (1999) found varying relationships between four types of mental disorders and a series of educational transitions. Neither anxiety nor depression at age 16 were significantly related to later educational attainment, whether (dichotomous) diagnoses or (continuous) symptom scales were used. On the other hand, conduct disorder was strongly associated with lower levels of achievement. Those with conduct disorder were less likely to earn a school certificate (in age 16 examinations), to earn a sixth form certificate, and to continue to tertiary education. Attention deficit disorder also negatively affected educational attainment, though results were only robust to the introduction of status attainment controls when the symptom scale rather than diagnosis was used. These results support an important distinction between internalising and externalising disorders in this context. While the findings of Breslau et al. (2011) also indicate this pattern, with disorders of conduct and inattention found to account for most of the effect of mental health on high school graduation in a US sample, Kessler et al. (1995) report that anxiety and depressive disorders also had a significant effect on a range of educational outcomes. Moreover, these results varied by sex, with anxiety disorders playing as strong a role as conduct disorders among women.



One of the analyses of Power et al. (2002) is to examine the effect of problem behaviour at age 16 on subsequent intergenerational social class mobility in the 1958 British birth cohort – the data used for this paper. Men and women with problem behaviour at age 16 were found to be more likely to be downwardly socially mobile, and less likely to be upwardly socially mobile. Problem behaviour here was indicated by falling in approximately the top 13% of the distribution of the total Rutter school behaviour scale (Rutter, 1967). Further analyses showed similar results using conduct and emotional sub-scales, though ‘this pattern was more pronounced for conduct disorder’ (Power et al., 2002: 1996). These authors do not, however, attend to the question of to what extent such selection effects may be accounted for by childhood risk factors.

It has been acknowledged, either explicitly (J. D. McLeod & Kaiser, 2004: 640; Stansfeld, Clark, Rodgers, Caldwell, & Power, 2011: 550), or implicitly in the identification strategy (Miech et al., 1999), that an effect of childhood or adolescent mental health on subsequent status attainment may simply be a reflection of causation that has occurred in childhood. For instance, Schoon et al. caution, in a discussion of their selection effect identification, that ‘[d]ifferences in the risk of later psychological well-being between more and less socially advantaged groups could result in part from the movement of young people with behavioural problems, who are more at risk of maladjustment, into less privileged social circumstances [health selection]. On the other hand, young adults who have been relatively disadvantaged in social and economic terms during childhood could have lower psychosocial well-being in part because they have acquired more behavioural problems as children and adolescents’ (2003: 1013).

This is often recognised, but the set of controls used in order to identify a health selection effect by conditioning on childhood risk factors is not always strong. Stansfeld et al. (2011), for instance, control for childhood socioeconomic position (manual vs. non-manual), sex and adulthood mental

health. Miech et al. (1999) control for family SES at age 15, sex, IQ, reading ability and school involvement. McLeod & Kaiser (2004) note that their data lacks information on father's SES and childhood adversities. Breslau and colleagues (2008; 2011) on the other hand control for a range of severe childhood adversities, though measures both of these and of psychiatric symptoms are based on retrospective self-report.

McLeod & Kaiser make an interesting contribution by explicitly quantifying the extent to which effects of mental health on education are accounted for by the inclusion of measures of the childhood environment: 'the effect of externali[s]ing problems [on high school degree receipt] was diminished by approximately 31 percent from the first model' (2004: 644). The equivalent figure for college enrolment was 37 percent, with the coefficient also no longer significant at the 0.05 level. As elsewhere, externalising problems are consistently associated with these outcomes. Internalising problems are significantly associated only with high school degree receipt, and this only when externalising problems are not included in the model, suggesting that comorbidity may explain the effect.

This paper incrementally extends this evidence in several ways. While studies using a strong set of controls have paid attention to educational outcomes in the US, this paper considers achieved social class position in the UK, using a nationally representative sample with strong, prospective measures of the childhood environment. Mental health is measured close to the transition to adulthood, at 16 – and by multiple informants – rather than, for instance, by retrospective self-report or by the mother. Latent variable modelling is used to control for error in the measurement of three distinct dimensions of adolescent mental health. Further, this analysis accounts for the possible bias introduced by comparing logistic regression coefficients across nested models, as McLeod & Kaiser do (Mood, 2010).

#### 4. Data & measures

##### *Data*

The National Child Development Study (NCDS) follows a British cohort made up of all births in England, Scotland and Wales in a single week in 1958, collecting data in a variety of domains. Data collected at birth and ages 7, 11, 16, 23 and 33 are used here. The NCDS includes a rich set of measures of the childhood environment, and measures of adolescent mental health from multiple informants. At age 33, 11,468 cohort members were interviewed (70.7% of the eligible sample). These participants are considered to be generally representative of the original cohort (Ferri, 1993).

##### *Dependent variable: intergenerational social class mobility*

Intergenerational social class mobility is used to indicate socioeconomic attainment. Both parental social class at age 16 (origin class) and own social class at age 33 (destination class) are indicated by the Registrar-General's Social Classification (RGSC) of current or most recent job. In the case of parental social class (measured in 1974), the father's class was used if available, otherwise the mother's. RGSC is divided into four categories: I&II (professional; intermediate), III non-manual (skilled non-manual), III manual (skilled manual), and IV&V (semi-skilled manual; unskilled manual). The two binary outcomes of upward (vs. not upward) and downward (vs. not downward) intergenerational social class mobility are determined according to this four-category system.

##### *Independent variables: adolescent mental health (AMH; age 16)*

Slightly modified versions of the Rutter behaviour scales are available in the data (Rutter, Tizard, & Whitmore, 1970). The purpose of these scales (adapted differently for teacher and parent response)

is 'the identification of broadly defined but clinically relevant behavioural disturbances' (Elander & Rutter, 1996a: 32). 13 items rated by both a parent and a teacher are used. For each item, the teacher and parent scores (0: 'does not apply', 1: 'applies somewhat', 2: 'certainly applies') are summed, creating 13 items ranging from 0 to 4, with ratings equally weighted between parents and teachers. This method aims to both minimise rater effects and acknowledge that home and classroom are contexts in which adolescents may behave differently (Elander & Rutter, 1996a). A validation study of the Strengths and Difficulties Questionnaire, a successor to the Rutter scales, finds that sensitivity in detecting psychiatric problems is greatest when parent and teacher ratings are combined (R. Goodman, Ford, Simmons, Gatward, & Meltzer, 2000).

The 13 items are allocated across three dimensions based on the scales' design and their use in the literature (Elander & Rutter, 1996b). The dimensions are conduct problems (*steals, destroys, disobedient, lies, bullies, fights, irritable*), emotional symptoms (*worries, fearful of new situations, solitary*), and hyperactivity (*squirmy, restless, can't settle*). To account for measurement error, a latent variable modelling approach is taken, and a confirmatory factor analysis conducted using the lavaan package in R (figure S1 in the supplementary material). For identification, the variances of the latent variables were fixed to 1. The diagonal least squares estimator – more specifically, weighted least squares with mean and variance adjusted (WLSMV) – is used, as is appropriate with ordinal variables (DiStefano & Morgan, 2014; Li, 2016). Model fit was good (robust test statistics: CFI = 0.976, TLI = 0.969, RMSEA = 0.052 [95% C.I. 0.050 - 0.054]). Other specifications, such as a two-factor internalising/externalising model, had poorer fit. Factor scores estimated from this model are used in the analyses.

*Independent variables: childhood socioeconomic deprivation (birth – age 16)*

22 dichotomous indicators of material hardship, from birth and ages 7, 11, and 16, are summed to give a childhood socioeconomic deprivation (CSD) score. These are listed in the supplementary material.

*Independent variables: childhood adversities (birth – age 16)*

Childhood adversities (CAs) are highly stressful events or conditions which are beyond the child's control (Clark, Caldwell, Power, & Stansfeld, 2010). The NCDS measures a good range of CAs prospectively. A particular strength is the variety of informants: the study records the prospective reports of parents or caregivers and teachers, and the independent assessment of health visitors to the family home (who also have access to relevant records).

Dichotomous indicators are used for each of seven CAs: taken into care, neglected appearance, family criminality, separation from the biological mother, separation from the biological father, bullied at school, and domestic tension. For a list of indicators of the included CAs, see the supplementary material.

## **5. Analytical strategy**

Logistic regressions are used to model the probability both of upward (versus stable or downward), and of downward (versus stable or upward) mobility. Cases in the highest and lowest origin class are excluded from the analysis of upward and downward mobility, respectively, since one cannot be upwardly mobile from the highest origin class and vice versa. In each case, I use the KHB method to compare a reduced model with a full model, whilst adjusting for the rescaling of the coefficients due to the need to fix the residual variance of the latent outcome (Karlson, Holm, & Breen, 2012, also see figure 3). In the reduced model, the adolescent mental health factors (included simultaneously

in all analyses) predict the mobility outcome, controlling for sex and origin class. In the full model, childhood socioeconomic deprivation (sum score) and childhood adversities (individually) are introduced as 'Z variables' and controlled for. Given temporal ordering, these Z variables are conceptualised as influencing both AMH and destination class, i.e. as confounders rather than mediators. For each of the AMH factors, the difference in coefficient between the reduced and full models represents the spurious component of the effect of AMH on the mobility outcome. This spurious component includes both the association generated by confounding ('indirect health selection', as childhood factors influence both AMH and destination class), and by mediation (social causation, as childhood factors influence AMH, and this in turn influences destination class). These two processes are not disentangled. However, they each give causal priority to childhood risk factors, rather than to AMH as in the health selection hypothesis. This method therefore allows a decomposition of the effect of AMH on social mobility into a 'true' selection effect, and a selection effect which is, rather, accounted for by earlier causation.

In terms of figure 3, the apparent selection effect represented by path A in the reduced model is decomposed. The 'true' selection effect is represented by path A in the full model; mediated social causation refers to the path B-A in the full model; confounding refers to the spurious association between AMH and social mobility generated by paths B and C in the full model.

Parental mental health, though a risk factor for AMH and partially modifiable, is not included among the CAs. This factor is expected to influence AMH through both environmental and genetic pathways. The genetic pathway, i.e. genetic transmission of poor mental health, is one source of the true health selection effect, and so parental mental illness is excluded in order not to overcontrol by conditioning out factors which may cause poor AMH independently of the environment. This necessitates a trade-off, since poor parental mental health is partly ameliorable, and so the selection effect estimates might attenuate by a greater amount were this variable treated as another CA, and

this would not be wholly due to conditioning on the genetic pathway. In addition to the justification in terms of not overcontrolling health selection, it is likely that, firstly, the conditions under which parental mental health is poorest and most impactful are highly correlated with the measured CSD and CA variables. Second, some of these CSD and CA variables may constitute pathways through which poor parental mental health affects AMH – e.g. the child appearing neglected – and so such environmental pathways may be partially controlled without the necessity of directly including parental mental health in the model.

A complete-case analysis would restrict the available sample to 3321 individuals. Instead, multiple imputation by chained equations is applied to maximise use of the available data, using the *mi* *ice* programme in Stata. Auxiliary variables include number of family moves at ages 7, 11 and 16, Bristol Social Adjustment Guides total score at ages 7 and 11 – a sum of teacher-rated ‘items of behaviour which in varying degrees, deviate from the norm or which may be symptomatic of emotional disturbance or social maladjustment’ (Shepherd, 2013: 1), parent-rated Rutter scale sum scores for conduct problems (*destroys, disobedient, fights, irritable*), emotional symptoms (*worries, fearful of new situations, miserable*) and hyperactivity (*squirmy, can’t settle*) at ages 7 and 11 (each item scored 0, 1 or 2 as above), and indicators of social class (RGSC, as at ages 16 and 33), unemployment, and economically inactive status at 23. 20 imputed datasets are generated. All cases are used in the imputations, but those dead by age 33 are excluded from the analysis.

As robustness checks, analyses are repeated a) using AMH factor scores from a CFA identical except that only teacher ratings of behaviours are used, b) using as a factor score the simple sum (0-4 for each item) of the indicators for each AMH factor (combining parent and teacher ratings as in the main analysis), and c) using a binary indicator for each AMH factor, with a cutoff at the 90<sup>th</sup> percentile of the factor score used in the main analysis. For each of these checks, multiple imputation is run separately, with 20 imputed datasets generated in each case.

## 6. Results

Descriptive statistics for the 3321 complete cases are shown in table 1. Due to standardisation of the latent variables in the CFA, the AMH factor scores each approximate a mean-centred normal distribution. The count of childhood socioeconomic deprivation items ranges from 0 to 21. The values at the 25<sup>th</sup>, 50<sup>th</sup> and 75<sup>th</sup> percentiles are 1, 3, and 6 respectively. As expected, childhood adversities are comparatively rare.

Table 2 is a mobility table for the complete-case sample. There is substantial intergenerational social class mobility, both upward and downward. In terms of absolute mobility, there is substantial growth in the professional & intermediate classes (I&II), and in the skilled non-manual group (IIINM). The number of skilled manual positions (IIIM) declines correspondingly, as the size of the semi-skilled & unskilled manual group (IV/V) remains stable. Further descriptive statistics, including means for the complete cases only, for all observed data, and for the multiply-imputed data, are shown in supplementary table S1. The large amount of observed data in non-complete cases supports the use of multiple imputation.

Results from the nested pair of logistic regression models with upward mobility as the outcome are shown in table 3, panel (a). In the reduced model, all three dimensions of AMH are associated with upward mobility, though the association is weaker in the case of emotional symptoms. Conduct problems and hyperactivity are each strongly related to mobility: a unit increase in the conduct problems factor score is associated on average with a 6.6 percentage point decrease in the predicted probability of achieving upward mobility, while the corresponding figure for hyperactivity is 8.5. For context, note that the sample mean probability of upward mobility is 0.57.



To what extent are the apparent health selection effects of the three dimensions of AMH attenuated when childhood risk factors are considered? The reduced model is compared with a full model including socioeconomic deprivation and various adversities in childhood as confounders. In this full model, the effect of conduct problems is much attenuated. 47.2% of the association between conduct problems and upward mobility is accounted for by these risk factors. This contrasts strikingly with the coefficients for emotional symptoms and hyperactivity, which do not differ between the reduced and full models, though the effect of emotional symptoms does become non-significant.

The results for the analysis of downward mobility (table 3, panel (b)) closely resemble those for upward mobility. For instance, against a 0.30 sample mean probability of downward mobility, the average partial effect of a unit increase in conduct problems is 5.1 percentage points in the reduced model, but 2.4 points in the full model. Two differences between the upward and downward mobility analyses are notable. First, that the effect of emotional symptoms, though of similar magnitude, is in this case significant in both full and reduced models. Second, that conduct problems become non-significant in the full model. However, most striking about this analysis is its similarity to the results for upward mobility: in addition to showing similar effect sizes, it is again found that around half of the selection effect associated with conduct problems in adolescence is accounted for by childhood risk factors, while no such attenuation is detected between the full and reduced model for emotional symptoms or hyperactivity.

Results of the robustness checks specified above (supplementary tables S2-S7) are very similar to those of the main analysis. In some models (upward mobility with specification a), and both outcomes with specification c)), the difference between the emotional symptoms coefficients in the reduced and full models was significant, though the change in the logit coefficient was no higher than 20%, well under half the corresponding figure for conduct problems. Similarly, in the models for

both mobility outcomes in specification c), the effect of hyperactivity did appear to attenuate significantly between models, though the change was no higher than 10%. Also, in the downward mobility analysis in specification a), the effect of conduct problems was non-significant (OR 1.21, 95% CI 0.99-1.48). The substantive conclusions appear robust across all four specifications.

## 7. Discussion

Growing up in deprivation and experiencing adversities such as bullying could partially account for the health selection effect of poor adolescent mental health and behavioural problems on socioeconomic attainment in adulthood. Previous research has raised this point in passing but not examined it in detail. This paper uses the NCDS to examine, first, whether selection effects are evident, and if so, second, to what extent they are accounted for by modifiable factors that place children at risk of poor mental health and restricted chances of upward mobility. The two mechanisms by which this may occur are what I have referred to above as mediated social causation and indirect health selection. In the former case, the childhood environment affects adolescent mental health, which in turn limits socioeconomic attainment. In the latter case, the childhood environment confounds the association between adolescent mental health and later socioeconomic attainment. Each of these mechanisms gives causal priority to stressors in the childhood environment, in contrast to the health selection hypothesis, which gives causal priority to poor mental health.

The analyses presented here suggest distinct patterns according to the aspect of adolescent mental health under consideration. Regarding the first question, conduct problems, emotional symptoms, and hyperactivity each appear to be independently associated with poorer mobility outcomes, though the effect of emotional symptoms is weaker. The results on the associations of conduct problems and hyperactivity with subsequent status attainment are broadly consistent with previous

work. On the other hand, the association with emotional symptoms has not always been found elsewhere. Miech et al. (1999) found that conduct disorder and attention deficit disorder were associated with subsequent educational transitions, while depression and anxiety were not. This pattern of externalising problems showing an association with status attainment and internalising problems not doing so is also found in the results of McLeod & Kaiser (2004).

As regards the second and main question, around half of the impact of adolescent conduct problems on one's mobility chances is accounted for by the socioeconomic context in which one is raised and the adversities one is subject to, which is inconsistent with an interpretation of the health selection effect which gives causal priority to the onset of poor mental health in driving health inequality. On the other hand, the associations of emotional symptoms and hyperactivity with social mobility do not appear to be attenuated, or at least are not attenuated to an extent at all comparable with the results for conduct problems, though it should also be recalled that the emotional symptoms association is substantively small. The lack of attenuation of the health selection effects of emotional symptoms and hyperactivity supports an interpretation of the health selection hypothesis giving causal priority to poor mental health. However, simply accounting for the influence of prior risk factors does not create causal estimates and these should not be interpreted as such.

It is not clear why an attenuation should be evident for conduct problems but not hyperactivity. One possibility is that the hyperactivity results reflect the strong genetic component in the related construct of attention-deficit hyperactivity disorder (ADHD) suggested by behavioural genetics research (Hudziak & Faraone, 2010: 731). To the extent that a dimension of mental health is directly influenced by genotype, its effect on mobility would not be expected to attenuate with controls for the risk factors. On the other hand, environment may moderate the consequences of genotype, such that we should still expect to see attenuation with the addition of the controls. However, the direct influence of genes explanation is consistent with the recent finding that 'neither chaos [i.e.

household disorganisation] nor SES moderated heritability, with consistent contributions from both genes and environment indicated across socioeconomic strata and levels of chaos' (Gould, Coventry, Olson, & Byrne, 2017). Results on gene-environment interactions for ADHD have been mixed, with 'substantial inconsistencies in the literature regarding even the most well associated genetic and environmental risk factors' (Pennington et al., 2009: 85).

Relatedly, previous research indicates that SES does moderate the association between genes and antisocial behaviour, with environmental factors playing a greater role for those in more disadvantaged environments (Tuvblad, Grann, & Lichtenstein, 2006). Supplementary tables S8 and S9 show results disaggregated by class of origin, which echo this finding. This analysis reveals that in both upward and downward mobility analyses, the attenuation in the association of conduct problems with mobility is greatest in the lowest origin class, suggesting that the environmental risk factors play a greater role in limiting mobility for those most disadvantaged in childhood.

This paper has several strengths, such as the use of latent variable modelling to control for measurement error, and a robust method for making comparisons of coefficients across nested non-linear probability models. It also has important limitations. First, it should be noted that a complete-cases analysis ( $N = 3321$ , shown in supplementary tables S10 and S11) showed highly similar results except that the effect of conduct problems on upward mobility was non-significant (OR 0.83, 95% CI 0.66-1.04), and emotional symptoms showed no effect on mobility (upward mobility: OR 0.91, 95% CI 0.81-1.04; downward mobility: OR 1.13, 95% CI 1.00-1.29). The inclusion of a particularly strong set of auxiliary variables (such as measures of mental health at ages 7 and 11) associated with the probability of being a complete case may explain this discrepancy, if those suffering from conduct and emotional problems are at an elevated risk both of attriting and of undesirable mobility outcomes. Comparing the multiple imputation and complete-cases analyses, the odds ratio point

estimates were very similar, and there was an identical pattern of which effects on mobility attenuated with the inclusion of the risk factor variables.

Second, this study may suffer from omitted variable bias with regard to stressors in the childhood environment. For instance, the most severe forms of childhood adversity, such as physical or sexual abuse, are not prospectively measured. However, previous research suggests that data on less severe aspects of the childhood environment may proxy for such traumatic events: 'parental death, being sent away from home, or child maltreatment (such as sex abuse, physical abuse, and serious neglect) tend to occur in the context of high cumulative adversity' (Schilling, Aseltine, & Gore, 2008: 1147). Relatedly, the decision to model only modifiable risk factors as confounders entailed excluding variables such as parental mental health, which affect children's mental health through multiple pathways and are to some extent, though not wholly, modifiable.

Third, interpretation of the AMH factor scores is slightly complicated by the fact that the SDs fall below 1, despite the standardisation of the latent variables from which they are estimated. Thus, a unit is not precisely equivalent to a standard deviation. This is due to clumping at the minimum values, which reflects the cases for whom no problems whatsoever were reported (8.2% of those observed on the AMH items). In other words, the observed AMH factor scores have positive skew (conduct problems: .84, emotional symptoms: .62, hyperactivity: .87), though in each case kurtosis close to 3 demonstrates similarity to a normal distribution (conduct problems: 3.31, emotional symptoms: 3.07, hyperactivity: 3.23). Above the median (conduct problems: 0.05, emotional symptoms: -.017, hyperactivity: -.051) these factor scores *are* normally distributed, with unit standard deviation. Recall also that robustness check b) uses simple sum scores of AMH items and gives the same conclusions as the main analysis.

Supplementary tables S12 and S13 reproduce the results from table 3 and show results for men and women separately. There is no clear gendered pattern to the results and gender interactions were consistently non-significant.

Supplementary table S14 details to what extent each of the childhood environment controls accounts for the attenuation of the conduct problems coefficient. The major contributions come from groups of the CSD variables: low income, crowding, and housing tenure (not owner-occupier) especially. The CSD variables together account for around 85% of the attenuation in both the upward and downward mobility analyses, with the CA variables accounting for the remaining 15%. This highlights child poverty and housing conditions as important targets for intervention, and suggests, both in terms of policy and research, that at least in this context the presence of specific childhood adversities may have a limited importance once generalised socioeconomic disadvantage is taken into account. On the other hand, this analysis lacks measures of the most severe CAs, and the CSD measures may in part be capturing their effects.

This study adds to the health inequalities literature by interrogating the empirical validity of the usual interpretation of health selection effects as indicating the causal priority of the onset of poor mental health. In the case of conduct problems, this interpretation of the health selection hypothesis ought to be tempered by the consideration that earlier social causation underlies a considerable portion of the association of conduct problems with undesirable mobility outcomes. Moreover, supplementary analyses revealed that this is the case to a greater extent among those from the most disadvantaged social origins. Efforts to increase social mobility might usefully be targeted here, and in particular at risk factors for behavioural problems among that portion of the population.

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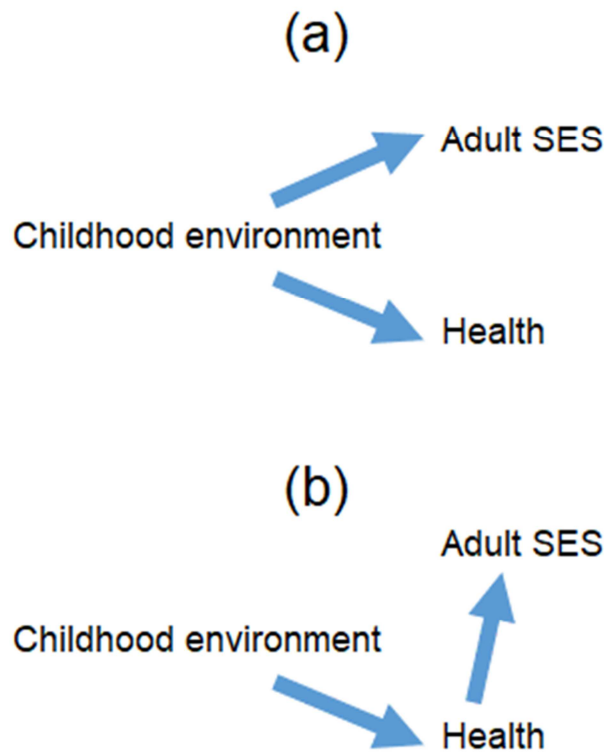
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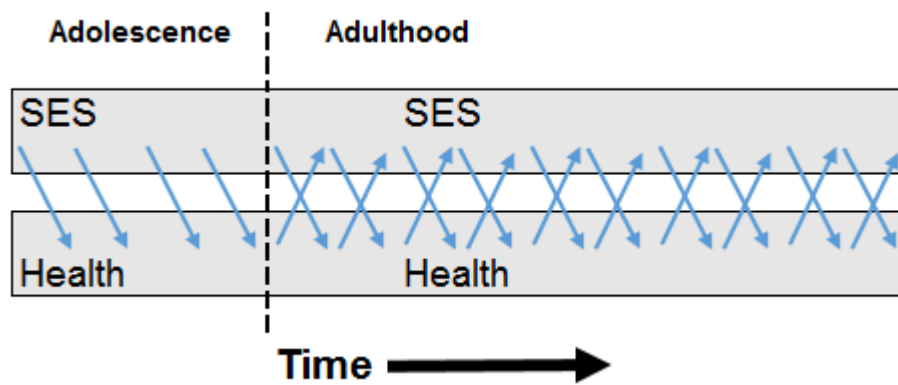
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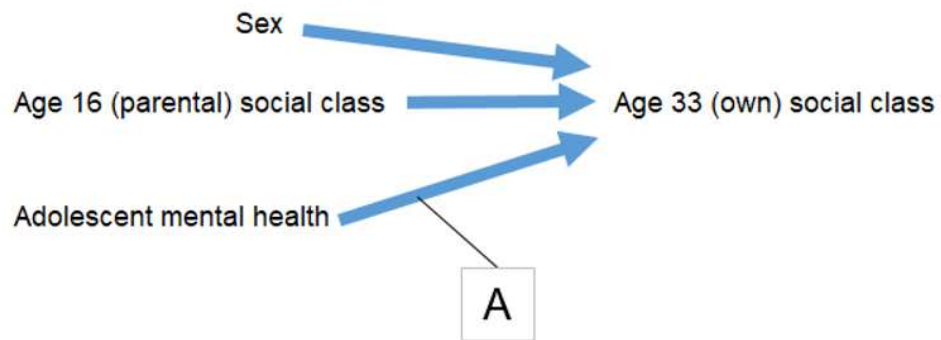


**Figure 1** (a) Confounding / 'indirect health selection' (b) Mediated social causation

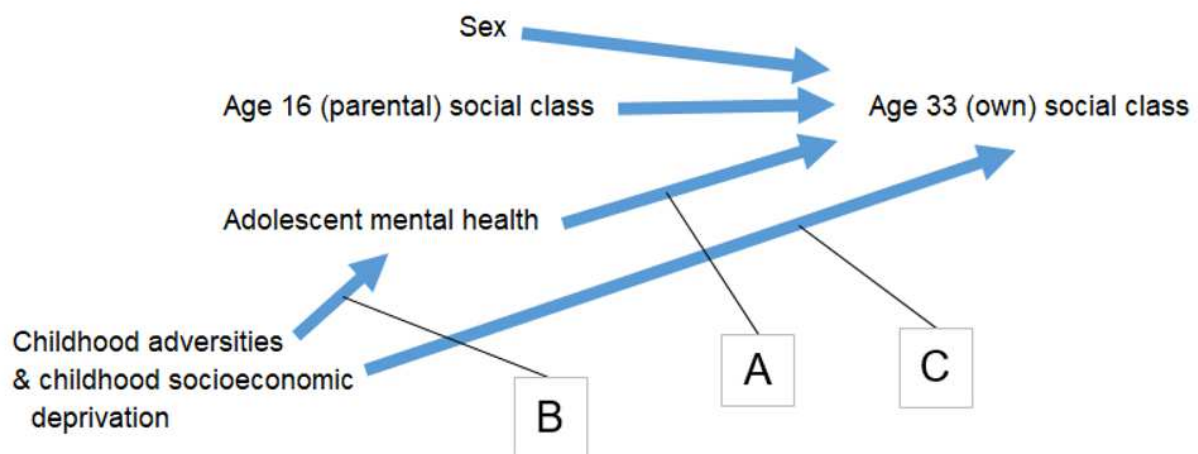


**Figure 2** Conceptual model of life-course causal relations between SES and health

# Reduced model



# Full model



**Figure 3** Reduced and full analytical models

**Table 1** Descriptive statistics, complete cases only,  $N = 3321$

Variable	Mean	SD	Min	Max
Female	0.51	0.50	0	1
Adolescent mental health				
Conduct problems	0.02	0.70	-0.75	4.14
Emotional symptoms	0.01	0.71	-0.96	3.32
Hyperactivity	0.04	0.70	-0.73	3.32
Childhood socioeconomic deprivation	3.90	3.37	0	21
Childhood adversities				
Taken into care	0.02	0.15	0	1
Neglected appearance	0.04	0.20	0	1
Family criminality	0.04	0.20	0	1
Separation from biological mother	0.04	0.19	0	1
Separation from biological father	0.08	0.26	0	1
Bullied at school	0.06	0.25	0	1

Domestic tension 0.03 0.18 0 1

**Table 2** Intergenerational social class mobility, complete cases only

Origin social class (age 16)	Destination social class (age 33)				Total	%
	I/II	IIINM	IIIM	IV/V		
I/II	545	225	99	100	969	29
IIINM	151	108	57	40	356	11
IIIM	422	340	332	300	1394	42
IV/V	134	138	161	169	602	18
Total	1252	811	649	609	3321	
%	38	24	20	18		

687 **Table 3** Upward & downward mobility: nested logistic regression models, KHB decomposition

		(a) Outcome: upward mobility							(b) Outcome: downward mobility								
		OR	95% C.I.		<i>p</i>	Δ (%)	APE	95% C.I.		OR	95% C.I.		<i>p</i>	Δ (%)	APE	95% C.I.	
Conduct problems	Reduced	0.74	0.65	0.85	<0.001		0.066	0.096	0.036	1.30	1.14	1.49	<0.001		0.051	0.025	0.077
	Full	0.85	0.74	0.98	0.028		0.035	0.066	0.004	1.13	0.98	1.30	0.081		0.024	0.003	0.051
	Difference	0.87	0.84	0.90	<0.001	47.2	0.031			1.15	1.11	1.19	<0.001	53.4	0.027		
Emotional symptoms	Reduced	0.93	0.87	1.00	0.047		0.016	0.032	0.000	1.11	1.02	1.20	0.014		0.020	0.004	0.036
	Full	0.94	0.87	1.01	0.094		0.014	0.030	0.002	1.10	1.01	1.19	0.028		0.018	0.002	0.034
	Difference	0.99	0.97	1.01	0.388	14.7	0.002			1.01	0.98	1.04	0.454	9.7	0.002		
Hyperactivity	Reduced	0.68	0.59	0.79	<0.001		0.085	0.117	0.053	1.39	1.20	1.62	<0.001		0.064	0.036	0.093
	Full	0.67	0.57	0.78	<0.001		0.089	0.121	0.056	1.42	1.22	1.65	<0.001		0.068	0.039	0.097
	Difference	1.02	0.99	1.05	0.129	-5.0	0.004			0.98	0.96	1.01	0.204	-5.3	0.003		

688 Sample size (number potentially upwardly/downwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 13184-13301 (b) 13343-13469.

689 Not shown: concomitant variables (full & reduced models): female, origin class; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.

690 APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.

691 Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson, Holm et al. 2012: 294,  
 692 equation 12c).



ACCEPTED MANUSCRIPT

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# Adolescent mental health and behavioural problems, and intergenerational social mobility: a decomposition of health selection effects

## Highlights

- Adolescent mental health and behavioural problems affect socioeconomic attainment.
- To what extent does the childhood environment account for this association?
- Half the effect of conduct problems is accounted for by earlier risk factors.
- No such attenuation for emotional symptoms or hyperactivity.
- Smaller effects on mobility of emotional symptoms than conduct or hyperactivity.