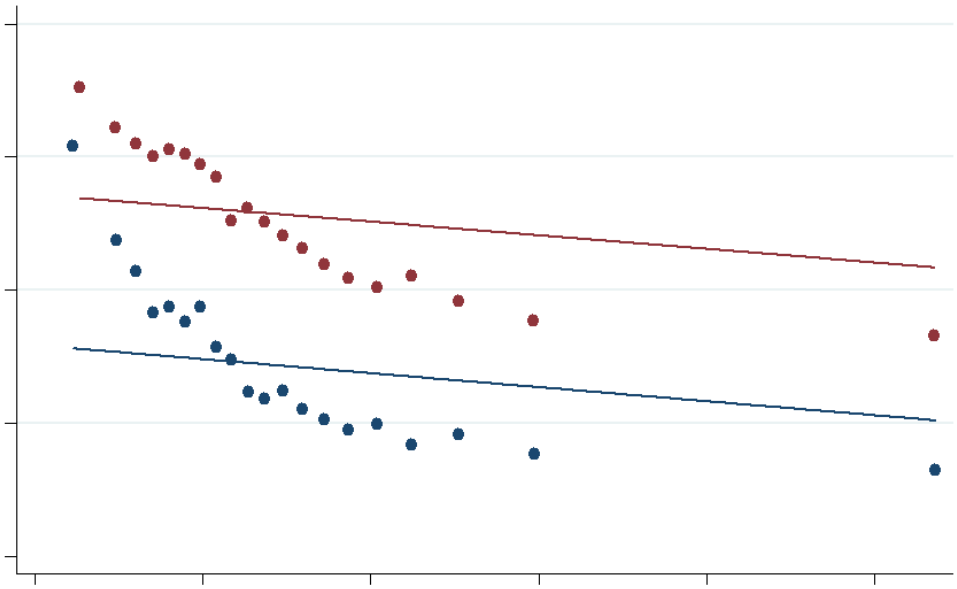
The Social Gradient in Mental Health

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Trinity term 2019



Acknowledgements

I am grateful to everyone who supported me in the completion of this work. Special thanks to those who are helpful without ever seeing the results or knowing whether it made a difference.

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Abstract

The aim of this thesis is to contribute to the understanding of the generative processes underlying the social gradient in mental health. I present three relatively self-contained empirical chapters, each attempting to answer questions which the current literature leaves unanswered. I first consider three questions regarding the role of the childhood environment. It is unclear whether the association which is often reported between low childhood socioeconomic status and adult mental health is accounted for by a greater exposure to various noneconomic childhood adversities – direct and relatively extreme stressors such as sexual abuse, neglect, or being frequently bullied. Next, through what pathways do these aspects of the childhood environment affect mental health in adulthood? And given evidence suggesting that the impact of certain stressors on mental health varies by gender, what contribution can the childhood environment make to explaining the gender difference in symptoms of depression and anxiety? Health selection and social causation are usually viewed as the two primary explanations for health inequalities. In my second empirical chapter, I ask: to what extent is health selection driven by prior social causation? In my third: when and how do social inequalities in mental health arise? In past research, the *how* has received considerably more attention than the *when*. This chapter aims to attend to both elements of the question, hypothesising that different mechanisms for the generation of mental health inequality may take precedence at different life course stages.

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1 Introduction

Marmot & Bell state that '[w]herever there are health outcome data disaggregated by indicators of disadvantage, there are health inequalities' (2012: S5). This is not true (as, incidentally, this thesis will show), but nor is it a great exaggeration. Social inequalities in health have been documented in a wide range of contexts and using a variety of measures of both social disadvantage and health outcomes (Feinstein, 1993; Kröger et al., 2015; Mackenbach, 2012; Mackenbach et al., 2008; Präg & Subramanian, 2017).

This is true of mental as well as physical health (Allen et al., 2014; Fryers et al., 2005; Lorant et al., 2003; Miech & Shanahan, 2000; Muntaner et al., 2004, 2013). For instance, Lorant et al.'s (2003) meta-analysis finds that across several measures of socioeconomic status (SES) and depression, low-SES individuals are significantly more likely to be depressed (overall random effects OR 1.81, 95% CI 1.57-2.10, $p < 0.001$), and a dose-response relation holds for education and income.

In broad terms, the aim of this thesis is to contribute to the understanding of the generative processes underlying this social gradient in mental health: why is it that people in lower socioeconomic positions are more likely to suffer from poor mental health? What processes, at what stages of the life course, are important for bringing this fact into being?

Before briefly introducing the empirical chapters, I discuss the conceptualisation of mental health and give an introductory overview of the sociology of mental health.

1.1 The concept of mental health

'Mental health' lacks a shared referent, even in the scholarly literature. It is helpful, firstly, to register three distinct but related, broadly coherent domains of mental health: a) positive mental health, flourishing, mental well-being, subjective well-being; b) psychological distress, low mood, symptoms of depression and/or anxiety; c) mental disorder, mental illness, specific psychiatric diagnoses. Payton (2009) explores the relationships between these. While no pair can be regarded as strictly on a shared continuum – such that, for instance, high positive mental health is simply the absence of

mental illness – the domains are highly correlated (0.77 for distress and disorder, –0.65 for distress and positive mental health, and –0.51 for disorder and positive mental health).

Without denying the importance of rarer disorders such as schizophrenia, personality disorders, or eating disorders, the focus of this thesis (and what I generally mean by 'mental health') is psychological suffering manifested as symptoms of (non-psychotic) depression and (generalised) anxiety – the 'common mental disorders' (Goldberg & Huxley, 1992). I am thus concerned not with positive mental health, but with a (major) subset of distress and disorder. I do not focus on the distinction *between* depression and anxiety, as the two are highly comorbid, conceptually similar, and often not differentiated in measurement scales.

The conceptual relationship between distress and disorder is debated. The psychiatric paradigm conceives of mental disorders as discrete entities, either present or absent in an individual at a given time. On this account, symptoms regularly cluster in predictable ways. Thus an experienced psychiatrist writes: 'Recogni[s]ing common patterns can make even ordinary clinicians seem like mind readers. Asking a patient who reports lacking hope, energy, and interest, "Does your food taste like cardboard, and do you awaken at four a.m.?" is likely to elicit "Yes, both! How did you know?"' (Nesse, 2019: 47). The psychiatric epidemiology literature (Fergusson et al., 2013; Kessler et al., 2010; Stansfeld et al., 2008, 2011) generally adheres to this schema. Structured interview instruments are used and binary diagnoses calculated algorithmically.

On the other hand, there is a long tradition, including among sociologists, of rejecting a conception of psychological problems as discrete disorders (Mirowsky & Ross, 1989; Scheff, 1966; Szasz, 1962). Under the distress conception of mental health, what psychiatrists call mental disorder is simply 'the distribution's tail' (Mirowsky, 2007: 301): the extremity of a continuous distribution, fundamentally the same phenomenon as at other points on the distribution, and an outcome of the same causal processes. The quantitative sociological literature on mental health tends to adhere to this view, whether explicitly or (usually) implicitly.

Whether or not the 'distribution's tail' assertion holds is an empirical question. The distinction between the 'common mental disorders' and others is important in this respect.

Kessler points to taxometric methods (of which 'sociologists appear to be unaware') which 'make it possible to test the hypothesis that dimensional symptom scale data are derived from an underlying discrete entity known as a taxon (Grove & Meehl, 1993)' (2002: 175). These methods 'search for consistent inflection points in associations among the symptom measures in dimensional scales that would indicate the existence of a taxon rather than a latent continuum' (Kessler, 2002: 175). A large review of such studies reported that:

> Promising but still not definitive evidence of psychological taxa was confined to the domains of schizotypy, substance use disorders and autism. This review indicates that most latent variables of interest to psychiatrists and personality and clinical psychologists are dimensional, and that many influential taxonic findings of early taxometric research are likely to be spurious.
> (Haslam et al., 2012: 903).

Other reviews also find that the evidence supports the hypothesis of depression as a continuum (Flett et al., 1997; Solomon et al., 2001). Kessler (2002) reports preliminary findings from a taxometric analysis in which he also finds no evidence for taxa in nonspecific psychological distress. He nonetheless urges the use of categorical measures as well as continuous. However, his arguments are rooted in the pragmatic concerns of treatment and clinical decision-making. For my purposes, a distress conception of mental health, measured primarily using continuous symptom scales which have their origins in screening for possible clinical caseness, is most suitable. This allows variation in severity to inform the investigation rather than be discarded.

There are further points of contention. Another position in the distress/disorder debate is that the two are conceptually distinct, and each *exist*, but those whose professional concerns involve the labelling of psychological states as 'disordered' have far overstepped their legitimate role, encroaching on normal, everyday negative emotions – distress – with the language of medical diagnosis (Davies, 2013). For instance, Horwitz (2007) holds that the American Psychiatric Association's *Diagnostic and Statistical Manual* – the standard diagnostic reference work – conflates normal responses to certain stressful situations, and actual pathology or dysfunction (that is, disorder). It is entirely normal and natural, on this account, to feel overwhelming pain and sadness if one suffers a major setback in life, such as the death of a partner. This would be distress without disorder.

This has three implications which are potentially of concern here. First, it implies that mental disorders (including depression and anxiety) *exist*, and are qualitatively different from distress. Indeed Aneshensel cites research by Karp (1996) showing that 'some people report experiencing depression as qualitatively different from ordinary distress' (2002: 240). It may then be that the causal processes underlying 'true' depression and anxiety are distinctive and considering such cases the 'distribution's tail' will lead to mismeasurement and misspecification. On the other hand, the taxometric evidence cited above suggests otherwise.

A second implication one might draw is that distress is not a problem. Rather, based on evolutionary theory, it has been argued that the capacity for low mood has conferred an adaptive advantage in the past, and that the mechanisms by which it did so are still in fact to a large extent useful in the modern world: 'communicating a need for help, signalling yielding in a hierarchy conflict, fostering disengagement from commitments to unreachable goals, and regulating patterns of investment' (Nesse, 2000: 14). Distress might best be viewed as a bodily signal analogous to the physical pain experienced if one puts one's hand too close to the fire: *stop what you are doing*. '[T]he psychiatry residents I have trained tell me they find it invaluable to ask their depressed patients, "Is there something very important that you are trying to do that you can't succeed at and can't give up?" (Nesse, 2019: Epilogue).

Even within this evolutionary framework it can be argued that our evolved predispositions conflict in important ways with the modern world, such that distress is very often *not* helpful. As Nesse elsewhere observes, 'modern social structures often seem to induce motivational structures that leave individuals sacrificing much in life to pursue major efforts to reach huge goals, whose attainment is uncertain and whose alternatives are few and unsatisfactory' (2004: 1343). Disengagement under this motivational structure may be much more difficult. This is reflected in the prominence of situations of 'entrapment' in the development of depression in Brown's research – he notes 'the difficulty we have as a species in escaping from punishing situations' due to role commitments (Brown, 2002: 270). To take an extreme example of the difficulties of disengagement in the modern world, whereas in the past postnatal depression might have brought a new mother to abandon a baby in a context in which prospects for raising it to

reproductive age no longer appeared good, this is now essentially an inconceivable option.

Whether or not it constitutes a valuable signal which may aid in bringing about productive change, distress as experienced is unpleasant and undesirable. The evolutionary psychiatry perspective implies that the social distribution of distress proxies the social distribution of unpropitious situations, status loss, and unreachable goals (Brown, 2002; Nesse, 2019). The social distributions both of distress itself, and of situations which lead to distress – whether distress is functional or dysfunctional in those situations – are of sociological and substantive interest.

Accepting the evolutionary perspective does not, then, entail viewing distress as unproblematic. This perspective does however raise important questions beyond the scope of this thesis. To what extent should distress be relieved at a superficial level through medication? Under what conditions should individuals be encouraged to allow distress to guide them (for instance, out of an unsatisfying relationship or an unwinnable status competition), and what policy measures can sensibly enable distress to fulfil this communicative role?

The third implication is that high distress may not correlate well with disorder. If disorder truly reflects some kind of pathology, perhaps with a stronger biological basis, whereas distress is the natural response to environmental stressors, then conflating the two would be an error. In response, I return to the findings of Payton (2009) cited above on the high correlation between measures of distress and (diagnostically assessed) disorder. Other evidence also shows that high scores on distress scales have been shown to be valid in discriminating those with clinically assessed psychiatric morbidity (Goldberg et al., 1997; Rodgers et al., 1999).

Finally, I would reiterate that the basis for viewing psychiatric diagnoses of depression and anxiety as distinct entities rather than the result of arbitrary cut-points on a continuum is not strong (but the commercial and professional incentives to do so, of pharmacological enterprises and psychiatrists respectively, are strong (Davies, 2013)). Consider the exchange below, between an interviewer and Robert Spitzer, chairman of the DSM-III taskforce charged with solving psychiatry's 'reliability crisis' of the mid-20th century:

> *Carlat*: How did you decide on five criteria as being your minimum threshold for depression?
> *Spitzer*: It was just consensus. We would ask clinicians and researchers, 'How many symptoms do you think patients ought to have before you would give them the diagnosis of depression?', and we came up with the arbitrary number of five.
> *Carlat*: But why did you choose five and not four? Or why didn't you choose six?
> *Spitzer*: Because four just seemed like not enough. And six seemed like too much [Spitzer smiles mischievously].
> (Carlat, 2010: 53-54).

Under the system Spitzer designed, and on which most psychiatric epidemiological measurement is still essentially based, two individuals can be diagnosed with major depressive disorder without sharing a single symptom (Monroe & Anderson, 2015). According to some, the reliability crisis in psychiatry continues to persist (Aboraya, 2007; Aboraya et al., 2006), though others argue that the evidence base for this claim is sparse (First, 2007). As even Kessler – perhaps the most eminent figure in psychiatric epidemiology – puts it, 'the diagnostic criteria for mental disorders stipulated in the American Psychiatric Association's [DSM] often had as much to do with votes by committees as scientific evidence (Kirk & Hutchins, 1992)' (2002: 172). If pathological depression and anxiety are binary states, distinct both from one another and from their absence, it has not been possible to identify their occurrence with both reliability and broadly-accepted validity.

Given my focus on psychological suffering manifested as symptoms of depression and anxiety, the above considerations lead me to conceptualise mental health as a continuum. It would be harder to argue that there is, for instance, a continuous distribution of schizophrenia in the population. For suffering characterised by the commonplace symptoms of depression and anxiety, however, this is the position I take. 'Sub-threshold' distress is of substantive interest, as are gradations of what might be termed 'super-threshold' distress. As elsewhere, dichotomising discards informative variation. In my first empirical chapter, I use both a continuous and categorical operationalisation of the outcome, simply because this measure – the malaise inventory – is dichotomised in every application of it which I have come across.

In the literature review that follows and in later discussions of existing research, I make little distinction according to whether authors view mental health problems as continuous or categorical. It appears rare that this alters conclusions.

1.2 The sociology of mental health

Before outlining the three empirical chapters, I present a very brief overview of some aspects of the sociology of mental health. I focus on how dimensions of social location are associated with poor mental health as conceptualised above, and draw on relevant evidence from a range of disciplines. This cannot of course be exhaustive, but I hope to give a broadly representative overview of the literature and highlight some of the stronger evidence available.

1.2.1 Sex

Women consistently show higher rates of depression and anxiety (Kessler et al., 1993, 2005; McLean et al., 2011). A stylised fact cited at the beginning of many papers is that adult women are twice as likely as men to be depressed (Cyranowski et al., 2000; Hyde et al., 2008; Nolen-Hoeksema & Girgus, 1994). The proposed explanations can be organised around the ideas of differential *exposure* to depressogenic factors between men and women, and differential *vulnerability* to them.

Biological explanations highlight anatomical and physiological bases for differential vulnerability. The gender difference in depression emerges in puberty (Angold et al., 1998), and has been linked to hormonal changes that can lead to limbic system hyperactivity; that is, heightened emotional responsiveness (Parker & Brotchie, 2004, 2010). This is reflected in women's tendency to report greater levels of neuroticism from puberty onwards (De Bolle et al., 2015; Schmitt et al., 2008). Parker and Brotchie contend that such a predisposition to a behavioural pattern of vigilant defence particularly conferred an evolutionary advantage among women '(in their role as mothers)', whereas it would have hindered men engaged in resource-gathering, 'where fearlessness would have group survival advantages' (2010: 433-434).[1]

[1] One might make the argument without invoking the controversial theory of group selection (Nesse, 2019; West et al., 2008).

Psychosocial and cultural explanations for the gender disparity, by contrast, tend to foreground the stresses experienced particularly by women. For example, women in a US sample were more likely to have experienced various forms of interpersonal and sexual violence such as rape. Adjusting for exposure to these stressors explained a modest part of the gender disparity, but there was no evidence for differential vulnerability (Dunn et al., 2012). On the other hand, Kessler (2000) in a similar analysis finds that while women experience greater levels of sexual trauma, other kinds of traumatic stressor are more likely to be experienced by men, and after adjustment for all such events the gender difference is little changed.

Others have examined more quotidian differences in stressors. Hochschild (1989) estimated thirty years ago that US women were working approximately an extra month than their spouses each year due to their entry into the labour force and the continuing norm that they perform the bulk of household labour. In the years since then, 'a slow and incomplete convergence of women's and men's work patterns' has taken place (Kan et al., 2011: 234). Accordingly, Marchand et al. (2016) examined the role of exposure to work and family stressors, as well as work-to-family and family-to-work conflict, finding that controlling for these factors rendered the gender difference in depressive symptoms nonsignificant, albeit with cross-sectional data. Work-family-conflict – measured with items such as *my work takes up time that I'd like to spend with family/friends* – was an important mediator, indicating the importance of role conflict to women's increased risk of depression.

Piccinelli and Wilkinson review a variety of explanations and report only limited evidence for several, including biological factors (gonadal hormones show a 'partial effect, although smaller than that of environmental variables' (2000: 487)), social support, vulnerability, and coping style. These authors endorse explanations based around women's social roles: partnered women without a job take on the 'frustrating' and 'devalued' role of housewife, while those who enter the labour market face the sort of 'role overload' and 'role conflict' described above (2000: 488). However the only evidence cited here is indirect, namely several findings from certain cultures (such as the Old Order Amish and the orthodox Jewish community in London) 'where high value is attached to the female role' and the gender difference in depression is either very limited

or absent altogether (*ibid*.). Like Kessler (2000), Piccinelli and Wilkinson report inconsistent support for the idea that women are exposed to a greater number of adverse life events. They suggest however that women are at greater risk of sexual abuse in childhood and may be more sensitive to its effects, based on analogous findings with respect to parental divorce (Rodgers, 1994) and some other childhood adversities (Veijola et al., 1998), concluding that 'gender differences in the association between adverse experiences in childhood and adult depression have been poorly investigated' (2000: 487). I extend this line of evidence in my first empirical chapter.

Differential exposure and vulnerability, and roles for biological, psychological, social, and cultural factors are not mutually exclusive, and likely interact in complex ways to produce the observed differences. A number of models have been proposed to integrate this extremely large body of research and explain the emergence of the gender disparity in depression (Cyranowski et al., 2000; Hankin & Abramson, 2001; Hyde et al., 2008; Nolen-Hoeksema & Girgus, 1994). Nolen-Hoeksema and Girgus (1994) argue that gender differences in traits and behaviours that are present before puberty constitute an increased vulnerability, which is then met with a disproportionately challenging early adolescence. The vulnerability factors in this model include a more ruminative, unassertive, self-focused style of responding to stress, and a pattern of group interaction prioritising cooperation. In adolescence women are confronted with, for example, 'the more domineering interaction styles of their male peers in heterosexual dating relationships and in competition for college and careers' (1994: 439), as well as abuse, harassment, and concerns over body image. Cyranowski et al. (2000) particularly focus on women's 'heightened affiliative need' during adolescence, arising partly from hormonal influences (oxytocin), and sensitising them to negative life events with interpersonal consequences.

The model of Hankin and Abramson (2001) is similar to that of Nolen-Hoeksema and Girgus (1994), emphasising differences in cognitive vulnerability and stressful life events; it is distinctive in positing that initial increases in depression can generate further negative life events, especially interpersonal conflict, and that such a cycle may contribute to the emergence of the gender difference. For instance 'increases in depression can lead to more dependent negative life events as depressed individuals seek reassurance excessively and are rejected by others (Joiner & Coyne, 1999)' (2001: 780).

Hankin and Abramson speculate on the development of girls' cognitive vulnerability to depression, citing qualitative evidence of socialisation patterns in which for instance 'mothers discussed sadness in longer and more detailed conversations with daughters than sons … [and] mothers are more controlling of their daughters than sons' (2001: 785). Greater exposure to childhood sexual abuse is again suspected to play a role, generating cognitive vulnerabilities to depression (such as generalised feelings of powerlessness and hopelessness) particularly among women. This link is also proposed by Hyde et al. (2008), whose model also emphasises the development of cognitive vulnerabilities such as an external locus of control ('negative cognitive style'), rumination, and 'objectified body consciousness' that comes with the expression of secondary sex characteristics.

As Hyde et al. (2008) argue, many pathways lead to depression, and it appears that women are at greater risk for many of these. Further research might usefully focus on how it is that socialisation influences the cognitive vulnerabilities which these chiefly psychological integrative theories highlight. For instance, girls learn that they are not expected to show the same levels of independence as boys and consequently appear to develop weaker instrumental coping skills (Cyranowski et al., 2000). Sociologists could usefully examine the macro and micro contexts within which this learning takes place. How might changing norms around women's educational and labour force participation, and role in society in general, influence this learning process and potentially lead to greater cognitive resilience among young women?

The gender difference is not the focus of this thesis. As stated above however, the contribution I make in this area (in the first empirical chapter) is to explore further the role of childhood adversities in this gender difference: sexual abuse in particular is repeatedly highlighted as a life event to which women may be more exposed and more vulnerable.

1.2.2 Age

Different life stages come with different sources of stressors and protective factors. Adolescents must negotiate peer pressures, unstable intimate relationships, and the emergence of an identity and social role. Adults bear responsibilities, including caring increasingly not just for their children but also their own parents. The elderly face

loneliness, isolation, and functional limitations. Accordingly, the age distribution of poor mental health defies succinct description. Rather, it is problem-specific and measurement-dependent. For instance, specific phobias largely arise during childhood. Considering the diagnostic categories of major depressive disorder and generalised anxiety disorder, the 25th, 50th, and 75th percentiles of age-of-onset in a nationally representative US sample were 19, 32, 44, and 20, 31, 47 respectively (Kessler et al., 2005).

With regard to levels of depressive and anxious symptoms, sharply contrasting findings have been reported. Most studies are of a particular age range or cohort, prohibiting comparison across age. Some US studies report that older adolescents experience more depressive symptoms on average than adults (Wight et al., 2004), or find U-shaped distributions over age, with symptoms lowest at approximately age 40-60 (Kessler et al., 1992; Mirowsky & Ross, 1992; Sutin et al., 2013). Others report a reverse U-shaped distribution over age (Dhingra et al., 2011; Tomitaka et al., 2018). Lang et al. (2011) find the reverse-U pattern in the UK, but only among the lowest income quintile. Recent UK evidence indicates that the proportion of women with high symptom scores is greatest for the 16-24 age group and otherwise follows the reverse-U pattern (though only weakly), while among men the proportion varies little across the age range from 25-64 (McManus et al., 2016). This remains an open issue. The distribution over age may vary with context and measurement instrument (particularly the weight given to somatic items which may independently worsen with age). It is possible that the association between age and depressive symptoms is not robust simply because it is small in magnitude (Kessler et al., 1992; Tomitaka et al., 2018).

1.2.3 Education

Researchers have consistently observed an association between education and mental health (Dohrenwend et al., 1992; Fryers et al., 2003). Some evidence suggests that this association strengthens with age, suggesting that better education may set individuals on more advantageous mental health trajectories across the life course (Miech & Shanahan, 2000). Protective effects of education may be relatively direct, as through social status, better coping and problem-solving skills, and a sense of control over one's environment

(Schieman & Plickert, 2008), or more contingent – through access to material resources, social support, and a desirable occupation.

Mechanisms for a causal effect of education are intuitively somewhat more doubtful than for certain other dimensions of SES. One can easily envisage how high income yields a level of control that can shield an individual from many stressors. The benefits of education are less tangible. Accordingly, many researchers have wondered whether the association between education and mental health is confounded by factors such as ability and social background. A large study of identical twins found that while simple OLS estimates reproduced the negative association between education and depression, within-twins models found almost a zero association (Halpern-Manners et al., 2016). Sociogenomic findings confirm that education and depression share common genetic influences (Boardman et al., 2015). Other work suggests that mental health problems which occur in childhood and adolescence (and often persist into adulthood) may restrict individuals' educational attainment (Breslau et al., 2008; Kessler et al., 1995; Miech et al., 1999).

1.2.4 Social class and related concepts

Social class as defined by Goldthorpe and colleagues (Goldthorpe, 2007) has clear conceptual implications for individuals' feelings of depression and anxiety. Individuals in service class occupations characteristically enjoy greater earnings, job security, earnings stability, and prospects of advancement (Goldthorpe & McKnight, 2006). While it has been suggested that a social class gradient in mental health is not a robust finding (Fryers et al., 2003), the null associations reported by Fryers et al. are based on use of the Registrar General's Social Classification, which as discussed below lacks a coherent theoretical rationale. Even so, it often does exhibit a gradient in mental health (Power et al., 2007; Richards & Paskov, 2016), as does social class measured with the NS-SEC (Chandola & Jenkinson, 2000).

Beyond differing returns and stability, occupations of different social classes also differ with regard to dimensions of the work itself which are consequential for mental health. A meta-analytic review concluded that 'job strain [high demands combined with low decision latitude], low decision latitude, low social support, high psychological demands,

effort-reward imbalance, and high job insecurity predicted common mental disorders' (Stansfeld & Candy, 2006: 443). It is not clear that 'salariat' work straightforwardly corresponds to favourable work characteristics and vice versa, but in some important respects this does appear to be the case. For instance, difficulty of monitoring (of employees by employers) is one of two main dimensions structuring the determination of type of employment contract. This is intuitively cognate to the concept of job autonomy or decision latitude. Likewise, on a 'working class' labour contract paid by the hour and with little prospect of promotion or progression, high effort is liable to go unrecognised and unrewarded beyond token gestures. Indeed, the gradient in depression and anxiety among civil servants according to their employment grade was largely accounted for by the combination of decision latitude, job demands, and work social support (Stansfeld et al., 1998).

Social class has an important indirect impact on mental health through its structuring of the distribution of economic status, and particularly unemployment (Gallie et al., 1998). Richards and Paskov (2016) report a social class gradient in symptoms of depression and anxiety, but also find that this is accounted for when economic status is controlled. Much evidence suggests that transitions out of employment, as well as the status of being unemployed itself, have a negative impact on mental health, as individuals lose a range of psychosocial and material benefits associated with employment (Brand, 2015; Frasquilho et al., 2016; Krug & Eberl, 2018; Paul & Moser, 2009; Steele et al., 2013).

Occupations may also be organised into alternative dimensions of social position, such as social status (Chan & Goldthorpe, 2007) and prestige (Treiman, 1977). Less research has examined the independent associations between these dimensions and mental health; more often they are incorporated alongside measures such as education and income into socioeconomic indices (Link et al., 1993; Muntaner et al., 2004; Wohlfarth, 1997). However, based on more idiographic work highlighting the strong impact of humiliating experiences on depression – such as 'put downs' (Brown, 2002: 259; Brown et al., 1995), it is likely that low status or prestige would place individuals at risk of symptoms of depression and anxiety, especially in the context of high variance in these dimensions at the workplace or societal level.

1.2.5 Income

Income is another dimension of social position which robustly correlates with depression and anxiety (Fryers et al., 2003; Sareen et al., 2011; Schlax et al., 2019). The relationship is non-linear, a unit of income mattering more for those with less (Hounkpatin et al., 2015). Different mechanisms underlying the association have been proposed, since income not only allows individuals to purchase goods and services (the absolute income hypothesis), but also confers status (the relative income hypothesis). In a test of models predicting current and future depression with various specifications of income – income utility, income rank, deviation in income from the mean of those with a similar level of education, and all three combined – a model with income rank alone was found to show superior fit, and results replicated with a different data set using wealth rather than income (Hounkpatin et al., 2015).

Some evidence suggests that income has a causal effect on mental health, however the range of studies harnessing natural experiments and credible instruments is rather limited, especially compared with the body of research which has examined the effect of income on physical health (Kawachi et al., 2010). An unexpected increase in pension income reduced depressive symptoms among adults living with pensioners in South Africa (Case, 2004); however this took place in a context of material scarcity and may not generalise to Western countries. In the UK, individuals who experienced a positive exogenous shock to their income through lottery and other gambling wins of between £1,000 and £120,000 (compared both with those who reported no such income, and those who reported smaller wins) saw an improvement in symptoms of depression and anxiety, but only *two* years after the win (Gardner & Oswald, 2007). Apouey and Clark (2015) replicate this result using more waves of the BHPS and controlling for individual fixed effects, finding that under some specifications there was also a positive impact on mental health one year after a win. Again however external validity is a concern, since one-off wins are conceptually distinct from a higher income as usually understood: *consistently* higher income in the form of an increase to wages or benefits. Reeves et al. (2017) and Kronenberg et al. (2017) each used the introduction of the UK minimum wage to explore this issue, but reached conflicting conclusions regarding the effect of this increase on symptoms of depression and anxiety among low-wage workers.

1.2.6 Contextual factors, family, and social support

Countless other factors at a range of levels – household, neighbourhood, network, community, nation (Allen et al., 2014; Bhugra et al., 2018) – act as social determinants of mental health. I offer here only the most sparse review of selected variables.

Attention should be drawn to certain non-obvious vulnerability factors and 'provoking agents' identified by Brown and Harris in their rich study of the origins of depression in samples of women (1978). Women were at greater risk of developing depression when exposed to an event such as a loss or a long-term interpersonal difficulty, when they lacked an intimate tie to confide in, had three or more young children at home, or lost their own mother before the age of 11.[2] In a different context, Small has highlighted the importance of 'someone to talk to', but found that the protective effects of being able to discuss one's problems do not necessarily require doing so with a close tie; in fact a weak tie or total stranger is often preferred (Small, 2017). The benefits of social support accord with the well-evidenced role of rumination in the development of mental health problems (Nolen-Hoeksema, 2000). Social support is somewhat socially stratified (Matthews et al., 1999).

A recent large-scale cross-national effort yielded the conclusion that individual-level factors accounted for 86.5% of the variance in depression and country-level factors the remaining 13.5% (Rai et al., 2013). This is striking given that 53 countries were included from across the scale of national development. Income inequality has been posited as an important national-level factor – a debate I will not enter here – but showed no association with depression in this study. Gallie (2004) suggests an important cross-national determinant of mental health by demonstrating that patterns of household formation relate closely to social isolation.

1.3 Outline of the thesis

I present three relatively self-contained empirical chapters.

[2] Being 'working class' was another such vulnerability factor.

The health inequalities literature is primarily concerned with two explanatory mechanisms: social causation (SES affects health) and health selection (health affects SES). The quality of the evidence for each of these is discussed piecemeal throughout this thesis, but particularly in Chapter 4. In Chapters 2 and 3 I seek to clarify aspects of these mechanisms with regard to mental health in the UK, taking advantage of the rich prospective measurement of the National Child Development Study cohort. The two mechanisms are treated rather separately in these two chapters.

In Chapter 4 I consider the two together, and aim to examine the extent to which they each contribute to the generation of the social gradient in mental health, with household income the measure of SES in this case. The systematic review of the health inequalities literature by Kröger et al. (2015) intriguingly found that, among studies which tested both the selection and causation hypotheses, those judged to be of higher methodological quality were more likely to find support for selection. The grading of methodological quality was based on whether studies dealt with random measurement error by using measurement models; with missing data by multiple imputation or full information maximum likelihood estimation; with confounding by using methods which capture time-invariant unobserved heterogeneity; whether they made estimates of health selection and social causation comparable by standardising regression coefficients; and whether both directions of causality were modelled.

While not guided exclusively by a concern to tick every box on this list, in Chapter 4 I attempt a methodologically rigorous evaluation of the strength of social causation and health selection in the contemporary UK, and moreover explore the extent to which they each contribute to the generation of the social gradient in mental health across the life course. I also give consideration to a third possibility – that health and SES do not causally affect one another, but have common causes. This has come to be referred to as the indirect health selection hypothesis (Foverskov & Holm, 2016; Hoffmann et al., 2018; Krug & Eberl, 2018), (although that term apparently holds a range of slightly different meanings (Ki et al., 2011; West, 1991)).

1.3.1 Aim of Chapter 2

In this chapter I focus on the social causation hypothesis, and in particular the role of the childhood environment. My first aim is to assess whether the association which is often reported between low childhood socioeconomic status (CSES) and adult mental health is accounted for by a greater prevalence of various noneconomic childhood adversities (CAs) – direct and relatively extreme stressors such as sexual abuse, neglect, or being frequently bullied.

Second, through what pathways do these aspects of the childhood environment affect mental health in adulthood? In particular, are the effects of early conditions mediated through the status attainment process, or – as the critical period hypothesis suggests – are there direct effects which persist independently of later life socioeconomic status?

Third, given evidence suggesting that the impact of certain stressors on mental health varies by gender, I test what contribution the childhood environment can make to explaining the gender difference in mental health.

Empirical results on each of these issues are mixed. I address these questions using a nationally representative prospective cohort study complemented by retrospective measures of the most serious childhood adversities.

1.3.2 Aim of Chapter 3

Whereas the previous chapter examines social causation, whereby stressors inherent in or associated with low SES lead to poor mental health and thus bring about the social gradient, this chapter interrogates the other mechanism regularly cited for the generation of health inequalities: health selection, according to which those with pre-existing poor mental health, because of this fact, drift to lower SES positions. I ask: to what extent can intergenerational health selection be accounted for by social causation which occurs earlier – during childhood?

1.3.3 Aim of Chapter 4

In this chapter I draw on several strands of the health inequalities literature to attempt to answer the following question: when and how does the association between income and

mental health problems arise? In past research, hypotheses relating to the *how* – social causation, health selection, or indirect health selection – have received considerably more attention than the *when* (Kröger et al., 2015). This chapter aims to attend to both elements of the question, hypothesising that different mechanisms for the generation of mental health inequality may take precedence at different life course stages.

Previous work has generally focused on testing for the *presence* of two mechanisms which each contribute to the formation of social inequality in mental health – social causation and health selection. This chapter further explores the extent to which each matters *for the generation of this inequality*, and at what stages of the adult life course they may do so. Further, in light of past research, this chapter uses a method that adjusts for unobserved heterogeneity – enabling direct testing of the indirect health selection hypothesis, and focuses on employment transitions due to their close links with health.

2 Adversities and socioeconomic status in childhood: gendered patterns of association with adult psychological distress in a British birth cohort

2.1 Introduction

The UK government estimates that £30-£40 billion is lost to its economy each year through mental ill-health (Department for Work and Pensions & Department of Health, 2009). Understanding the antecedents of conditions with such high social and economic costs is of great importance. While it is well-established that the childhood environment is associated with mental health outcomes in later life, questions remain as to the nature of this relationship.

This chapter attempts to clarify and make more precise the broad proposition that the childhood environment affects mental health in adulthood, using as its outcome a measure at age 33 of psychological distress, or more specifically, symptoms of depression and anxiety – the 'common mental disorders' (Stansfeld et al., 2016).

Figure 1 illustrates the conceptual relationships at issue. First, the primary question with which this chapter is concerned: what aspects of the childhood environment matter for later mental health? In particular, it is unclear whether growing up in a lower social class household is associated with poorer mental health in adulthood because of generalised socioeconomic disadvantage, or because of the greater prevalence in such contexts of childhood adversities, a set of highly stressful events or conditions which are correlated with but distinct from material conditions. From a policy standpoint, it matters whether particular types of adversity account for this association and could thus be targeted in isolation with a view to reducing both the burden of and inequality in mental health.

Second, through what pathways do these aspects of the childhood environment affect mental health in adulthood? In particular, are the effects of early conditions mediated through the status attainment process, or – as the critical period hypothesis suggests – are there direct effects which persist independently of later life socioeconomic status? Third,

do these processes vary between men and women? There is evidence suggesting that the impact of certain stressors on mental health varies by gender. Empirical results on each of these issues are mixed, as discussed below. I address these questions using a nationally representative prospective cohort study complemented by retrospective measures of the most serious childhood adversities.

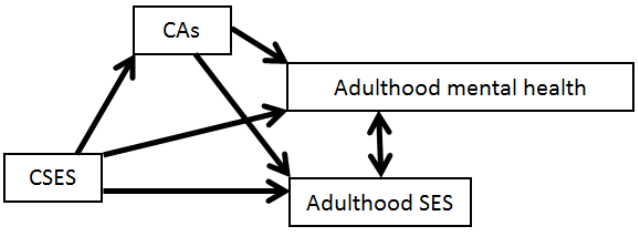


Figure 1 Model of childhood influences on adulthood mental health
Note: CSES = Childhood socioeconomic status, CAs = Childhood adversities.

2.2 Prior evidence & hypotheses

2.2.1 Childhood socioeconomic status (CSES) and childhood adversities (CAs) as antecedents of poor mental health in adulthood

Two generally separate bodies of evidence – mostly within psychiatric epidemiology – have investigated associations with mental health in adulthood of, firstly, childhood socioeconomic status, and secondly, childhood adversities (also referred to as adverse childhood experiences (ACEs)).

In the case of CSES, results have tended to indicate an association with mental health in adulthood (Elovainio et al., 2012; Gilman et al., 2002; Greve, 2013; Luo & Waite, 2005; Power et al., 1997; Quesnel-Vallée & Taylor, 2012). For instance, principal components loading on family income and parental employment, and on parental education, were strongly related to alcohol use disorder, other mental disorders, and comorbidity of the two in an Australian birth cohort (Salom et al., 2014). Gilman et al. (2002) report that manual parental occupation at birth and age 7 is associated with close to twice the odds of lifetime risk of major depression in a US sample, compared with non-manual parental occupation. With similar measures, Power et al. (1997) find similar strengths of association in the UK.

However there have been results to the contrary (Mäkinen et al., 2006; Veijola et al., 1998), and results have varied with the operationalisation of CSES or the mental health outcome in question. In a New Zealand birth cohort no bivariate association was found between parental occupation and major depressive disorder or anxiety disorder, though parental occupation was associated with tobacco dependence and alcohol or drug dependence (Melchior et al., 2007). In the US National Comorbidity Study Replication, neither parental occupation nor parental education was associated with the onset of mental disorders, while financial hardship (defined as ever having received government financial assistance) was (McLaughlin et al., 2011). With parental education as a measure of CSES, both positive and inverse relationships with adult mental health have been found (Lahelma et al., 2006; Ochi et al., 2014). In this chapter CSES is operationalised as social class, as a broad and relatively stable indicator of social advantage.

Many types of childhood adversity have been investigated in relation to mental health in adulthood. As with physical health, there appears to be a robust relationship (Felitti et al., 1998; Moffitt & The Klaus-Grawe 2012 Think Tank, 2013). Kessler et al. (2010) estimate the population attributable fraction of all mental disorders worldwide due to CAs at close to 30%.

In this literature, CAs are often considered jointly, in sets whose size and composition varies considerably between studies. Separation from either parent – whether due to death, divorce, or another reason – and parental mental illness or substance abuse are usually included (Chase-Lansdale et al., 1995; Gilman et al., 2002; Schepman et al., 2011). Other CAs often measured are parental violence or criminality, neglect, physical or sexual abuse, low birth weight, physical illness, and being bullied at school (Green et al., 2010; Kessler et al., 2010; McLaughlin et al., 2012; Oshio et al., 2013; Wojcik et al., 2013). Other stressors are less frequently measured and show less consistent associations with later outcomes, such as exposure to a natural disaster or having to repeat a year of school (Schilling et al., 2008). The most consistent and strongest childhood predictors of mental disorder in adulthood are parental mental illness, parental substance misuse, parental criminality, family violence, physical or sexual abuse, and neglect (Green et al., 2010; Kessler et al., 2010; Schilling et al., 2008). Parental mental illness, physical abuse, and sexual abuse are each independently associated with approximately twice the hazard

of onset of mental disorder in the World Health Organisation's large, cross-national World Mental Health Surveys (Kessler et al., 2010).

Despite the correlations between low CSES and CAs (McLaughlin et al., 2011), few studies have assessed their independent associations with mental health in adulthood. Veijola et al. (1998) examine 21 childhood factors and the risk of adult depression, but offer no definition or operationalisation of 'low social class of father', for which they report no association with the outcome. McLaughlin et al. explore multiple dimensions of CSES and conclude that while an association appears between financial hardship and the onset of mental disorders, this 'remained significant only [for onset in] childhood once we controlled for CAs, suggesting that exposure to CAs explains the association between financial hardship and onset of mental disorders in adolescence and adulthood' (2011: 1094). On the other hand, results from Danish register data suggest associations between father's unemployment in the year before birth and risk of schizophrenia and personality disorders, and between being in the lowest income quartile and risk of affective disorders (Greve, 2013). In sum, results have been mixed (Colman et al., 2014; Melchior et al., 2007; Quesnel-Vallée & Taylor, 2012).

Where effects of CSES have been found independent of CAs, the set of CAs has tended to be limited (Lundberg, 1993; Sadowski et al., 1999; Salom et al., 2014) – for instance to parental mental illness, single-parent status, and mother's age (Gilman et al., 2002). On the other hand, studies showing that CAs account for the apparent effect of CSES often depend wholly upon retrospective measures of CAs (Mäkinen et al., 2006; Ochi et al., 2014), which have been found to upwardly bias associations between CAs and mental disorder in adulthood, though the extent of the problem is debated (Moffitt & The Klaus-Grawe 2012 Think Tank, 2013; Susser & Widom, 2012).

It remains unclear, then, whether the effect of CSES on adult mental health is in fact accounted for by CAs, or whether on the other hand the generalised socioeconomic disadvantage of CSES exerts an effect independent of CAs. The answer may vary across measures, outcomes, and contexts. This question has not been investigated using a nationally representative UK sample, which is the first aim of this chapter.

2.2.2 Life course models: the critical period, status attainment mediation, and null hypotheses

If the childhood environment may exert an effect throughout the life course, it is important to attend to how this occurs (Ben-Shlomo & Kuh, 2002). Which of several hypotheses receives support has implications for action on the social determinants of mental health.

A first hypothesis is of a critical or sensitive period early in the life course, such that early – even prenatal – experiences associated with low SES have a persistent effect on mental health, 'regardless of subsequent life circumstances' (Hertzman, 1999: 86). The mechanisms envisaged here are biological and involve more-or-less permanent effects that exert an ongoing direct influence on mental health: Hänsel et al. write, for instance, that 'the second and third year of life seem critical for setting the stage for a pro-inflammatory phenotype later in life' (2010: 119). Much evidence now links inflammation and depression (Bullmore, 2018). Stress-response systems are particularly implicated: 'accumulating evidence suggests that childhood adversity is associated with dysregulation in the autonomic nervous system and the hypothalamic-pituitary-adrenal axis' (McLaughlin et al., 2010: 1092). Further developmental pathways linking CAs with later poor mental health include social information processing biases, difficulties with emotion regulation, and disruptions in reward processing (McLaughlin et al., 2019). Support for this hypothesis would direct policy towards material and psychosocial support during pregnancy and childhood, the aim being to inculcate resilience and avoid stressful experiences whose deleterious effects echo through the life course. In this chapter, results suggesting substantial effects of childhood circumstances on adult mental health *independently of adult SES* would support this hypothesis.

A second hypothesis is of pathway effects, whereby early socioeconomic disadvantage has an indirect effect on later mental health, acting through the status attainment process and later life low socioeconomic status. This may also be understood as a process of cumulative advantage or disadvantage. The apt policy response would involve improving the childhood environment, as part of a broader effort to promote equality of opportunity, as well as general improvements to the psychosocial environment through the life course in order to reduce the mental health penalty associated with low SES in adulthood. In

this chapter, support for this hypothesis corresponds to a reduction in the effect size of childhood factors once adult SES is controlled.

The null hypothesis, according to which there is no association – even bivariate – of adult psychological distress with the childhood environment, would be consistent with the view of Melchior et al. that 'adult experiences are key in determining people's risk of adult depression and anxiety' (2007: 972). Under this null hypothesis, early intervention strategies appear mistargeted with respect to this particular outcome, though they may nevertheless relieve contemporaneous distress.

Reviewing the literature on CAs, George writes that 'these studies convincingly demonstrate that early stressors continue to have direct effects on mental health 50-60 years later' (2013: 593), while there is also evidence of associations between CSES and mental health in adulthood which persist net of adult SES (Gilman et al., 2002; Power et al., 2007), as well as contrary findings supporting the null hypothesis (Harper et al., 2002). However there is a paucity of studies testing the joint effects of CSES, a range of childhood adversities, and adult SES on adult mental health. Melchior et al. (2007) present apparently the only such test, but as noted above, no bivariate association between childhood SES and mood disorders was found. The second aim of this chapter is therefore to explore whether these childhood factors exert effects which persist with the inclusion of adult SES in the multivariate model, and hence whether, or to what extent, they are mediated by the status attainment process.

2.2.3 Variation by gender: the vulnerability and exposure hypotheses

The overall prevalence of mental disorder is estimated to be similar for men and women when all types of disorder are considered; however, women have odds around 50% greater than men for lifetime prevalence of any mood disorder (Kessler et al., 2005). That disorders tend to be so strongly patterned by gender suggests that the causal processes at work are not identical for men and women. For this reason the analysis is stratified by gender and interaction terms are used to test for gender differences in associations between risk factors and adult mental health.

The psychological distress outcome used in this chapter most closely corresponds to a diagnosis of clinical depression (see below), and so a greater prevalence is expected among women. One explanation for this is that particular factors increase the risk of depression more strongly for women than for men. Supporting evidence has been found with regard to stressful life events (Maciejewski et al., 2001). This is referred to as the *vulnerability hypothesis* (Marchand et al., 2016), though it should be borne in mind that the focus is on differential vulnerability *to depression*, and it is possible that any group's differential vulnerability to one disorder is offset by relative resilience to another. Slopen et al. (2011), however, reject the simple narrative that stressful life events are more likely to precede depression among women, and alcohol dependence among men.

An alternative explanation for these gender differences is that men and women are differentially *exposed* to risk factors whose effect may or may not vary by gender. Women consistently report higher rates of childhood sexual abuse (Evans et al., 2017; Fuller-Thomson et al., 2013), one of the CAs most strongly linked with later psychological distress for both men and women. The third aim of this chapter is to add evidence to the debate on the vulnerability and exposure hypotheses.

Before presenting empirical findings, I discuss the conceptual boundary between CSES and CAs.

2.3 Defining childhood adversity

The definition of a CA implicit in the majority of the literature is an a priori severe childhood stressor, whether chronic or acute. Hence, the list of potential CAs theoretically extends to include any substantive childhood stressor. However CAs are seldom explicitly defined. Typically low CSES is excluded, under an implicit assumption that low parental education, social class or occupational prestige – for instance – are only contingently experienced as stressful by the child, if at all. Consider this theorisation of adverse childhood experience:

> We have identified ACE as a set of traumatic and stressful psychosocial conditions that are out of the child's control, that tend to co-occur and often persist over time ... We have restricted ACE to intra-familial events or conditions in the child's immediate environment causing chronic stress

> responses. In our definition ACE is distinguished from events or conditions linked to the socioeconomic and material environment. (Kelly-Irving et al., 2013).

This conceptual clarity is helpful. The distinction between noneconomic CAs and 'the socioeconomic and material environment' is taken as the basis of the definition used in this chapter. There are other strengths: that CAs are *out of the child's control* is necessary to avoid endogeneity. By contrast, Hatch et al. (2010), for instance, include individual characteristics such as high anxiousness or aggressiveness in childhood and low ambition and truancy at age 16 in multivariate models alongside social context indicators such as parental separation or divorce. Such variables may be on the causal pathway from early environment to poor mental health in adulthood. As such, they might be informatively included in a sequential model, in order to assess the extent to which they mediate CAs or CSES, but otherwise the effects of variables measuring the early environment are obscured.

On the other hand, several points within the definition of CAs above are contingently true of CAs – but not usefully a part of their definition. They do *tend to co-occur*, but if a single CA occurs – even *usually* occurs – in the absence of others, this is not cause to exclude it from the definition. The same point applies to the fact that CAs *often persist over time*. A single exposure to a stressful event might have far-reaching effects.

Moreover, that CAs are events or conditions *causing chronic stress responses* begs the question of the mechanism through which they influence adult mental health, presupposing a form of critical period model. A specific physiological response may or may not be implicated in the association of interest.

In this context, it seems unnecessary to exclude events or conditions which occur outside the family context, such as bullying by peers or abuse by another adult. Extra-familial events are conceptually similar to adversities that occur within the family context, insofar as the distinction of interest is essentially between socioeconomic disadvantage, and severe stressors which are noneconomic in character and usually relate to the quality of interpersonal interaction.

Severe financial hardship has been included in several studies, conceived of alternatively as low CSES (Lahelma et al., 2006) and as a CA (McLaughlin et al., 2011). I follow Kelly-Irving et al. (2013) in their exclusion of socioeconomic factors from the set of CAs. However the occurrence of severe financial hardship is not easily categorised as a measure of CSES either, since it usually refers to a period of acute hardship rather than a more stable attribute such as social class. I assume that episodes of severe financial hardship may be regarded as a possible intermediary mechanism through which CSES conceived of as social class exerts its effects, alongside factors such as lower wages and job insecurity.

I therefore define a childhood adversity as an event or condition occurring during childhood which can reasonably be expected to be experienced as directly and severely stressful by the child, and which is out of his or her control.

2.4 Data

The National Child Development Study (NCDS) follows a British cohort made up of all births in a single week in 1958. Data collected at birth and ages 7, 11, and 16 provide prospective measures of CAs and CSES, while psychological distress and adult SES are measured at age 33 (sweep 5). Retrospective measures of certain CAs are from the biomedical survey conducted at age 44-46.

At age 33, 10,986 cohort members were interviewed (70.6% of the target sample). These subjects are considered generally representative of the original sample (Ferri, 1993). Some evidence from Denmark based on a comparison of survey responses with national register data suggests that differential attrition does not bias associations between childhood circumstances and adult mental health (Osler et al., 2008). Moreover, complete-case analysis (using only cases for which all variables are observed) is valid when the probability of being a complete case depends only on the covariates (White & Carlin, 2010). This is plausible with the model applied here – the same sorts of disruptive conditions which are linked to poor mental health in adulthood are also likely to be related to the probability of dropout in adulthood.

On the other hand, partly due to the use of multiple indicators for most CAs and the use of some retrospective measures taken in mid-adulthood, the number of available complete cases is limited to 1,827 men and 1,963 women. I therefore apply multiple imputation (by chained equations), and results are based on 20 imputed datasets. Despite the low number of *complete* cases, a much larger number of respondents are observed on most of the analysis variables, as suggested by the descriptive statistics in Table 1. Multiple imputation allows the analysis to incorporate this information. Moreover, I include in the imputation procedure a wide range of auxiliary variables which have been found to be associated with attrition in the NCDS (Atherton et al., 2008; Hawkes & Plewis, 2006; Seaman et al., 2012). I also include as auxiliary variables several measures of mental health from a range of ages, which are expected to be predictive of the dependent variable. The auxiliary variables I use are listed in the Appendix to this chapter. The imputations are run separately for men and women to allow the valid testing of interactions by gender (Tilling et al., 2016).

Cohort members who were dead at the age 33 sweep (mostly cases of perinatal mortality) were excluded.

2.5 Measures

2.5.1 Childhood SES

Childhood SES is based on the father's occupation at birth, according to the Registrar-General's Social Classification (RGSC). Taking the measurement from birth rather than a later time during childhood has some advantages. First, it measures socioeconomic circumstances during (or very close to) the theoretically especially important prenatal and early years periods. Second, the possibility that some characteristic of the child might influence parental SES is minimised.

RGSC is divided into four categories: I/II (professional; managerial and technical), III non-manual (skilled non-manual), III manual (skilled manual), and IV/V (semi-skilled manual; unskilled manual). The collapsing of the ends of the distribution is a standard convention (Power et al., 1997, 2002), and one which answers Rose & Pevalin's (2000:

1125) criticism of Chandola (2000) that the RGSC has a strong tendency to show top-to-bottom differences if classes I and V are separated from their neighbours, since they are small groups representing approximately 5% at each end of the distribution. The original variable includes a fifth category: 'not applicable / no male head of household.' Cases in this category were assigned the social class of the mother, and in case this was missing, the social class of the mother's father.

An important limitation of the RGSC is that it lacks a detailed or clear underlying rationale, being based initially, so far as it has been possible for historians to piece together the process, on a 'commonsense' division of occupations 'into a top, a middle and a bottom' based on the level of skill generally involved in their practice (Szreter, 1984: 533). However, the RGSC has been widely used with this sample and other British data, and is the only available measure of social origins in the early waves of the NCDS.

Some previous work has examined the childhood determinants of the mental health of this sample, but it dichotomises CSES into manual and non-manual and uses it as a control, not reporting the coefficients (Clark et al., 2010; Hatch et al., 2010; Power et al., 2002).

2.5.2 Childhood adversities

The NCDS measures a wide range of CAs, some prospectively and others retrospectively (both in some cases). 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). The retrospective reports are made by the cohort member using a computer, as part of an interview.

Ever in care =1 if according to the parent or caregiver the cohort member has ever been in local authority or foster care (measured at age 7, 11, and 16).

Neglected =1 if the teacher reports that the child appears 'underfed' or 'scruffy and dirty' (age 7), or 'undernourished' or 'slovenly, dirty' (age 11).

Family criminality =1 if a family member (other than the cohort member) is in contact with a probation officer (age 7), or probation services or police (age 11 or 16), or if a family member is in prison (age 11).

Separated from biological mother =1 if, at either 7, 11, or 16, the 'person acting as mother' (age 7 and 16) or the 'child's mother figure' (age 11) is coded as anything other than 'natural mother.'

Separated from biological father =1 if, at either 7, 11, or 16, the 'person acting as father' (age 7 and 16) or the 'child's father figure' (age 11) is coded as anything other than 'natural father.'

Family mental illness =1 if any of the following are reported: the family is in contact with a psychiatric social worker or mental welfare officer; mental illness/neurosis is listed as a family difficulty by the health visitor (age 7); the family is in contact with mental health services other than child guidance; the father, mother, or another adult in the household has a chronic condition described as 'psychiatric' (age 11); or the father, mother, or another adult in the household has a psychiatric diagnosis (age 16).

Bullied =1 if according to the parent or caregiver the child is 'frequently' bullied by other children (age 7 or 11).

Domestic tension =1 if the health visitor reports domestic tension as a family difficulty (age 7).

Sexually abused =1 if the cohort member answers 'Yes' to 'I was sexually abused by a parent (or parent figure)' (age 45).

Physically abused =1 if the cohort member answers 'Yes' to 'I was physically abused by a parent – punched, kicked or beaten with an object, or needed medical treatment' (age 45).

2.5.3 Adult SES

Adult SES, at age 33, is based on the cohort member's current or last job (or that of their partner, whichever is highest: Erikson's (1984) 'dominance method'), coded to the 1990 version of the Goldthorpe class schema (Goldthorpe, 1980), the precursor to the National Statistics Socio-economic Classification. Since the variable is measured in 1991, this version of the schema precedes the slight modification which explicitly made employment relations the central factor differentiating the classes (Erikson & Goldthorpe, 1992). Instead, occupations are grouped into social classes based on similarities in the work and market situations associated with them (Lockwood, 1958; Pevalin & Rose, 2002). The former refers to an individual's position within the hierarchy of authority in the workplace, the latter to the character of the rewards of one's work – income, job security, and prospects of advancement. As such, it is not surprising that before and after the conceptual modification and elaboration of Erikson and Goldthorpe (1992), 'the allocation of individuals to locations in the schema are calculated in the same way' (Evans, 1992: 213).

This measure has seven categories: I (higher-grade professionals and administrators, managers in large establishments, large proprietors), II (lower-grade professionals and administrators, managers in smaller establishments, higher-grade technicians, supervisors of non-manual employees), III (routine non-manual), IV (small proprietors and other self-employed), V (lower-grade technicians, supervisors of manual workers), VI (skilled manual), and VII (semi- and unskilled manual workers) (Goldthorpe, 1980).

2.5.4 Outcome: psychological distress

Psychological distress at age 33 is assessed by the malaise inventory (Rutter et al., 1970), a 24-item self-completion questionnaire which assesses symptoms of depression and anxiety. Each of the 24 items is scored *yes* or *no*. The items are listed in full in the Appendix to this chapter. A couple of examples are 'Do you feel tired most of the time?' and 'Do you often feel miserable or depressed?'

The malaise inventory shows acceptable internal consistency in this sample and in others, and discriminates moderately well those with current case-level depression and other affective disorders (McGee et al., 1986; Rodgers et al., 1999). For example, Johnston et al. use British Cohort Study data including reports of medically-treated mental health

conditions, and estimate that (using a subset of nine malaise items), 'every additional 'yes' response … increases the probability of having a depressive-type disorder by 7 percentage points' (2013: 1079).

In line with other work using the malaise inventory, this outcome is dichotomously coded, with seven or more symptoms indicating high distress (Clark et al., 2010; Hope et al., 1999; Power et al., 2002). Where six or fewer items are missing, values are prorated. For example, if five items are missing, a score of six or higher indicates high distress. This operation is applied to 139 cases, most of which are only missing one or two items. A supplementary analysis treats the malaise inventory as a continuous variable.

2.6 Analytical approach

First, a series of multivariate logistic regressions is estimated for the outcome of having seven or more malaise symptoms. At this stage, analyses are run separately for men and women. To explore the unadjusted relationships, the first model includes only CSES, and a second model includes all the CAs but not CSES. Then, the question of whether CAs account for the observed association between CSES and adult mental health is addressed by including the two sets of variables together, in the third model. To test whether effects persist net of adult SES, a fourth model adds this control. Apparent gender differences are tested explicitly with interaction terms in a pooled model. As a robustness check, I replicate these steps taking the malaise inventory as a continuous variable and using linear regression.

In order to both illustrate effect sizes and examine whether comparisons between odds ratios across models are affected substantially by unobserved heterogeneity (Mood, 2010), average marginal effects are also presented. The continuous-outcome analyses also serve as a check on any such problems, as they are unaffected.

To further address the question of gender differences and, in particular, the question of whether differences in vulnerability or exposure account for the greater proportion of women scored as having high distress, I apply a Oaxaca-Blinder decomposition (Blinder, 1973; Oaxaca, 1973). I use the user-written Stata package *oaxaca* (Jann, 2008). This sets

up a series of counterfactuals, exploring what effect it would have on the gender difference in the outcome, if the groups' levels of (exposures to) the independent variables were the same, and what effect it would have if their vulnerabilities (i.e. the coefficients) were the same.

2.7 Results

Table 1 describes the data. For each variable, it shows the percentage coded positively (among those observed on that measure), the number observed, and (among those coded positively on each measure) the proportion with a high malaise score. Childhood adversity variables are shown disaggregated into each age at which they are measured. (In the subsequent analyses these age-specific measures are combined such that each CA is dichotomous, with a value of 1 for a CA measure at *any age* indicating the presence of that CA). Gender differences in the levels of variables which are significant at the 0.05 level are indicated.

Most notably, the proportion scoring above the threshold for high malaise is almost twice as high among women. Retrospective reports of having been sexually abused as a child are significantly higher among women. Prospective reports of having been taken into care (by age 16), of neglect (at ages 7 and 11), of having been separated from one's biological mother (by age 16), and of being bullied (at age 11) are significantly higher among men. The distribution of adult SES also varies between the sexes. The proportions of individuals with a high malaise score at age 33 by social class illustrate the social gradient in mental health. In this case the pattern is not strictly monotonic, but there are clear steps between classes I and II, III to VI, and – with a strikingly higher prevalence – class VII, the semi- and unskilled manual workers. Before adjustment for covariates, women falling in this category have approximately 75% greater risk of high psychological distress than men of the same social class, and seven times the relative risk compared with men of class I.

Figure 2 shows the distributions of scores on the malaise inventory for men and women, with the conventional cut-off point indicated. In each case, the number of symptoms is highly right-skewed. 57.12% of men and 43.99% of women reported either zero or one

symptoms. The mean score for men is 2.06, and for women 2.85. The non-integer values with very small percentages are the prorated values for cases in which some items are missing, as described above.

Table 2 shows the distribution of the total number of CAs by CSES. The prevalence of CAs follows a clear social gradient, with higher exposure to adversity in lower SES households.

Table 1 Descriptive statistics, observed data

		Men			Women		
		%	N observed	Malaise ≥7 (%)	%	N observed	Malaise ≥7 (%)
Outcome (age 33)	Malaise ≥7†	6.87	5571	-	12.22	5770	-
Childhood SES (at birth)	I/II	17.38	8347	4.47	17.47	7935	6.26
	III non-manual	10.05	8347	5.46	10.11	7935	11.23
	III manual	50.07	8347	7.49	49.44	7935	12.61
	IV/V	22.50	8347	7.56	22.99	7935	15.82
Childhood adversities	Ever in care (age 7)	2.28	7413	19.59	2.16	7076	24.74
	" (age 11)	3.63	6896	19.73	3.19	6611	25.19
	" (age 16)†	3.44	7449	20.71	2.73	7062	25.00
	Neglected (age 7)†	4.62	7790	14.62	3.34	7445	20.95
	" (age 11)†	4.46	7754	12.57	2.91	7394	24.60
	Family criminality (age 7)	1.83	6562	10.94	2.00	6309	30.38
	" (age 11)	1.79	7754	19.35	2.02	7394	22.73
	" (age 16)	3.15	7449	10.74	3.31	7062	24.55
	Separated from biological mother (age 7)	3.09	7433	12.98	2.83	7091	10.57
	" (age 11)	3.93	6989	12.05	3.42	6703	11.04
	" (age 16)†	5.41	5912	13.50	4.56	5658	14.14
	Separated from biological father (age 7)	7.12	7433	10.63	7.22	7089	15.20
	" (age 11)	9.14	6988	11.50	10.11	6704	15.33
	" (age 16)	13.11	5911	9.26	13.74	5656	14.41
	Family mental illness (age 7)	4.06	6994	18.71	4.23	6687	21.76
	" (age 11)	2.88	7754	13.29	3.04	7394	13.33
	" (age 16)	1.81	7449	14.94	1.74	7062	22.47
	Bullied (age 7)	5.67	7271	13.33	5.00	6943	21.67
	" (age 11)†	4.60	6759	18.95	3.45	6516	22.50
	Domestic tension (age 7)	6.34	6361	16.29	5.76	6116	23.21
	Sexually abused (R)†	0.52	4610	13.64	2.83	4658	28.44
	Physically abused (R)	6.00	4581	12.33	6.20	4642	22.44
Adult SES (Goldthorpe Social Class) (age 33)	I†	27.37	5386	3.49	25.64	5547	7.09
	II†	19.42	5386	4.71	21.24	5547	8.28
	III†	16.52	5386	7.08	29.60	5547	11.89
	IV†	5.48	5386	5.08	2.88	5547	12.58
	V†	4.64	5386	8.03	2.65	5547	12.24
	VI†	12.92	5386	7.39	6.45	5547	14.85
	VII†	13.65	5386	14.34	11.54	5547	24.69

Note: Malaise refers to the 24-item malaise inventory. A score of 7 or higher indicates high psychological distress. † indicates a gender difference in the prevalence percentage significant at the 0.05 level. (R) indicates item measured retrospectively, at age 45.

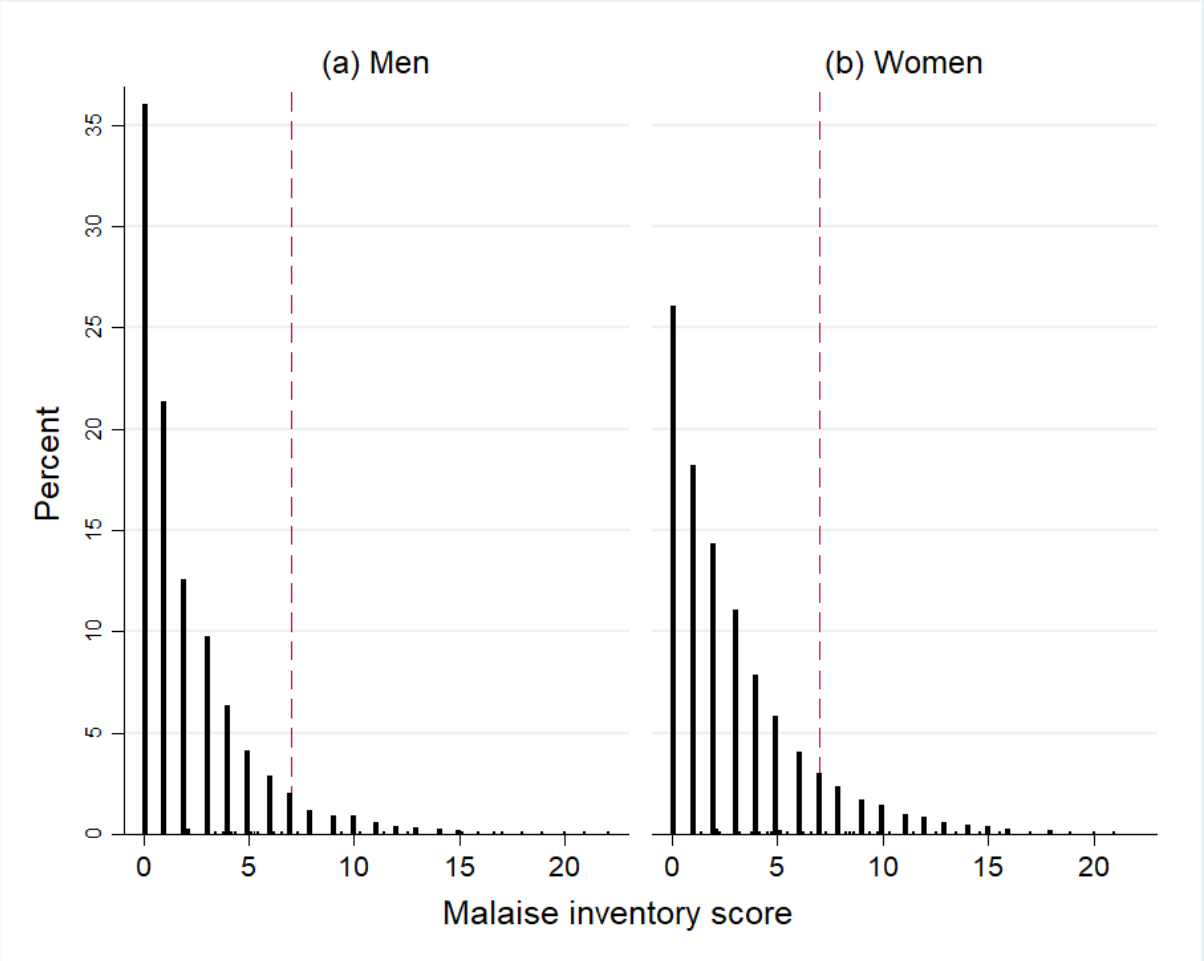


Figure 2 Distribution of malaise inventory scores, by sex
Note: dashed line indicates the conventional cut-off, at *7 or above*.

Table 2 Distribution of number of childhood adversities by childhood SES

	Men			
	Childhood SES			
Number of CAs	I/II	III non-manual	III manual	IV/V
0	79.04	73.03	66.57	58.82
1	15.18	19.92	23.18	23.53
2	4.34	4.56	6.37	10.54
3	1.20	1.66	2.31	4.17
4	0.24	0.00	1.29	2.21
5	0.00	0.00	0.09	0.49
≥6	0.00	0.83	0.18	0.25

	Women			
	Childhood SES			
Number of CAs	I/II	III non-manual	III manual	IV/V
0	79.12	68.42	66.52	55.78
1	16.71	23.25	22.68	23.13
2	2.78	4.82	7.11	10.43
3	0.93	2.19	2.61	7.03
4	0.23	0.44	0.54	1.81
5	0.23	0.44	0.27	1.36
≥6	0.00	0.44	0.27	0.45

Table 3 Logistic regression results (outcome: high psychological distress as indicated by 7 or more malaise symptoms), men

	(1)	(2)	(3)	(4)
CSES: I/II	*Ref.*		*Ref.*	*Ref.*
CSES: III non-manual	1.17		0.99	0.93
	(0.28)		(0.25)	(0.24)
CSES: III manual	1.80***		1.48*	1.22
	(0.29)		(0.25)	(0.21)
CSES: IV/V†	1.82***		1.20	0.89
	(0.32)		(0.23)	(0.17)
Ever in care		1.80**	1.81**	1.65*
		(0.34)	(0.35)	(0.34)
Neglected		1.59**	1.59**	1.33
		(0.23)	(0.23)	(0.20)
Family criminality		1.27	1.24	1.16
		(0.28)	(0.27)	(0.25)
Separated from biological mother		1.20	1.19	1.20
		(0.21)	(0.21)	(0.21)
Separated from biological father		1.04	1.05	1.02
		(0.14)	(0.14)	(0.14)
Family mental illness		1.79***	1.80***	1.70***
		(0.28)	(0.28)	(0.27)
Bullied at school		2.20***	2.17***	2.03***
		(0.30)	(0.29)	(0.28)
Domestic tension		1.52*	1.53*	1.46
		(0.31)	(0.31)	(0.29)
Sexually abused		1.00	0.96	0.88
		(0.64)	(0.61)	(0.55)
Physically abused		2.31**	2.28**	2.19**
		(0.60)	(0.60)	(0.59)
Adult SES: GSC I				*Ref.*
Adult SES: GSC II				1.28
				(0.25)
Adult SES: GSC III				1.83**
				(0.35)
Adult SES: GSC IV				1.41
				(0.43)
Adult SES: GSC V				2.03*
				(0.57)
Adult SES: GSC VI				1.89***
				(0.35)
Adult SES: GSC VII				3.76***
				(0.75)

Note: coefficients are odds ratios. Standard errors in parentheses.
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$
† model 4 coefficient significantly differs by sex at 0.05 level.

Table 4 Logistic regression results (outcome: high psychological distress as indicated by 7 or more malaise symptoms), women

	(1)	(2)	(3)	(4)
CSES: I/II	*Ref.*		*Ref.*	*Ref.*
CSES: III non-manual	1.91**		1.76**	1.64*
	(0.40)		(0.37)	(0.35)
CSES: III manual	2.10***		1.83***	1.51**
	(0.30)		(0.27)	(0.22)
CSES: IV/V†	2.70***		2.06***	1.60**
	(0.45)		(0.35)	(0.27)
Ever in care		1.49*	1.43	1.40
		(0.28)	(0.27)	(0.27)
Neglected		1.61**	1.52**	1.29
		(0.24)	(0.23)	(0.20)
Family criminality		1.34*	1.28	1.19
		(0.19)	(0.19)	(0.18)
Separated from biological mother		1.09	1.08	1.05
		(0.19)	(0.19)	(0.20)
Separated from biological father		1.09	1.07	1.04
		(0.10)	(0.10)	(0.10)
Family mental illness		1.31*	1.31*	1.25
		(0.18)	(0.18)	(0.17)
Bullied at school		1.79***	1.71***	1.65**
		(0.27)	(0.26)	(0.25)
Domestic tension		1.52**	1.50**	1.46*
		(0.22)	(0.22)	(0.21)
Sexually abused		2.13**	2.08**	2.04**
		(0.50)	(0.49)	(0.50)
Physically abused		1.81**	1.76**	1.69*
		(0.36)	(0.35)	(0.35)
Adult SES: GSC I				*Ref.*
Adult SES: GSC II				1.17
				(0.18)
Adult SES: GSC III				1.56**
				(0.21)
Adult SES: GSC IV				1.69
				(0.47)
Adult SES: GSC V				1.53
				(0.43)
Adult SES: GSC VI				1.84**
				(0.35)
Adult SES: GSC VII				3.60***
				(0.52)

Note: coefficients are odds ratios. Standard errors in parentheses.
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$
† model 4 coefficient significantly differs by sex at 0.05 level.

Table 3 shows the regression results for men, with the dichotomised version of the outcome. In model 1, with CSES the only predictor, it is apparent that the likelihood of poor mental health in adulthood is higher among those from the lower two social classes of origin (e.g. IV/V versus I/II OR 1.82). Model 2, including only CAs, suggests that exposure to being taken into care, neglect, family mental illness, being bullied, domestic tension, and physical abuse are each independently important. Some CAs which have elsewhere been shown to be influential do not here register an independent association with mental health in adulthood among men.

In model 3, including both CSES and CAs together, the CSES coefficients appear considerably attenuated (e.g. IV/V versus I/II OR 1.20). By contrast, the coefficients for the CAs are almost identical in models 2 and 3. Among men, a large part of the association between CSES and mental health in adulthood appears to be accounted for by the greater prevalence of CAs in low SES households in childhood.

In the final model – with adult SES added to model 3 – current (adult) SES is shown to be strongly associated with psychological distress. The CSES coefficients are further attenuated, reflecting the intergenerational persistence of SES. Some CA coefficients – ever in care, neglected – are modestly attenuated with the inclusion of adult SES. However in most cases there is only a negligible change in the CA coefficients, suggesting that their effects do not occur through the status attainment process. This is particularly true of coming from a family in which a parent suffered mental illness, being frequently bullied at school, and being physically abused by a parent or other adult.

Table 4 shows the results for women. Again in model 1 CSES is associated with the outcome, but the point estimates are clearly larger than for men. Each of the other CSES categories is significantly different from RGSC I/II, and the odds ratios are considerable. A woman born into what approximately corresponds to the lowest SES quintile is estimated to have nearly three times the odds of experiencing high psychological distress at age 33 compared with a woman born into the approximate top quintile (IV/V versus I/II OR 2.70). In model 2, again most of the CAs show an independent association with the outcome. The most apparent contrast with the results for men is that among women, having been sexually abused shows a substantial association with high psychological distress, though the standard error is relatively large.

In model 3, as for men there is some attenuation of the CSES coefficients. However in contrast with the results for men, all three categories remain significant and continue to show substantial associations with the outcome. Again, the CA coefficients are not much changed once CSES is included. Strikingly, the odds ratio for the contrast between the highest and lowest CSES categories is of a similar magnitude to that for sexual abuse.

When adult SES is added, in model 4, the CSES coefficients are surprisingly persistent, though they are again attenuated (e.g. IV/V versus I/II OR 1.60). A pooled model confirms that the coefficient for the lowest CSES category differs significantly between men and women at the 0.05 level. In this model, however, the gradient across all four CSES categories is gone and the substantive contrast is between the highest origin class and all of the others, suggesting a particular protective effect of high origin social class among women, when adult SES is taken into account. This further suggests that the distinctively large coefficients for CSES IV/V in the earlier models reflect both a higher concentration of CAs in such households, and higher chances of reaching a low adult social class.

In addition to the significant interaction of gender with the lowest CSES category, sexual abuse appears to affect women more strongly, but cases are too few and standard errors too large to reject the null that the association does not differ for men and women. With regard to the CA coefficients, as for men those adversities which show stronger associations with the outcome change very little from model 2 to 4. Finally, as expected, adult SES is strongly associated with concurrent mental health.

Table 5 Average marginal effects from logistic regression models (outcome: high psychological distress as indicated by 7 or more malaise symptoms), men

	(1)	(2)	(3)	(4)
CSES: I/II	*Ref.*		*Ref.*	*Ref.*
CSES: III non-manual	0.79 [-1.68, 3.26]		-0.02 [-2.75, 2.71]	-0.44 [-3.50, 2.61]
CSES: III manual	3.58 [1.84, 5.33]		2.53 [0.54, 4.52]	1.30 [-0.92, 3.52]
CSES: IV/V	3.66 [1.62, 5.70]		1.09 [-1.15, 3.33]	-0.71 [-3.08, 1.66]
Ever in care		3.94 [1.33, 6.56]	3.98 [1.31, 6.64]	3.32 [0.56, 6.08]
Neglected		3.12 [1.24, 5.00]	3.10 [1.18, 5.02]	1.89 [-0.02, 3.80]
Family criminality		1.60 [-1.29, 4.49]	1.46 [-1.42, 4.34]	0.97 [-1.82, 3.76]
Separated from biological mother		1.22 [-1.06, 3.50]	1.19 [-1.09, 3.47]	1.21 [-1.06, 3.47]
Separated from biological father		0.25 [-1.53, 2.02]	0.30 [-1.50, 2.10]	0.13 [-1.65, 1.91]
Family mental illness		3.92 [1.81, 6.02]	3.92 [1.82, 6.02]	3.50 [1.44, 5.57]
Bullied at school		5.30 [3.51, 7.10]	5.19 [3.39, 6.99]	4.65 [2.82, 6.48]
Domestic tension		2.82 [0.09, 5.55]	2.85 [0.13, 5.56]	2.50 [-0.13, 5.14]
Sexually abused		0.00 [-8.60, 8.62]	-0.29 [-8.90, 8.31]	-0.87 [-9.18, 7.45]
Physically abused		5.60 [2.16, 9.04]	5.49 [2.01, 8.97]	5.14 [1.65, 8.63]
Adult SES: GSC I				*Ref.*
Adult SES: GSC II				1.12 [-0.68, 2.92]
Adult SES: GSC III				3.21 [1.07, 5.36]
Adult SES: GSC IV				1.69 [-1.52, 4.90]
Adult SES: GSC V				4.02 [0.22, 7.82]
Adult SES: GSC VI				3.45 [1.25, 5.66]
Adult SES: GSC VII				9.70 [6.69, 12.70]

Note: average marginal effects (multiplied by 100), with 95% confidence intervals in brackets.

Table 6 Average marginal effects from logistic regression models (outcome: high psychological distress as indicated by 7 or more malaise symptoms), women

	(1)	(2)	(3)	(4)
CSES: I/II	*Ref.*		*Ref.*	*Ref.*
CSES: III non-manual	5.55 [1.89, 9.21]		5.07 [1.18, 8.96]	4.82 [0.63, 9.01]
CSES: III manual	6.58 [4.53, 8.63]		5.53 [3.32, 7.75]	3.85 [1.38, 6.32]
CSES: IV/V	9.81 [6.92, 12.70]		6.92 [3.93, 9.90]	4.47 [1.38, 7.55]
Ever in care		4.35 [0.33, 8.37]	3.89 [-0.14, 7.92]	3.52 [-0.46, 7.51]
Neglected		5.17 [1.97, 8.38]	4.51 [1.29, 7.74]	2.67 [-0.61, 5.94]
Family criminality		3.17 [0.03, 6.30]	2.68 [-0.44, 5.79]	1.82 [-1.29, 4.92]
Separated from biological mother		0.93 [-2.87, 4.73]	0.83 [-2.98, 4.64]	0.54 [-3.39, 4.46]
Separated from biological father		0.95 [-1.11, 3.00]	0.69 [-1.35, 2.74]	0.46 [-1.59, 2.51]
Family mental illness		2.94 [0.02, 5.87]	2.92 [0.02, 5.82]	2.32 [-0.58, 5.22]
Bullied at school		6.33 [3.14, 9.52]	5.81 [2.62, 9.00]	5.25 [2.12, 8.38]
Domestic tension		4.53 [1.40, 7.67]	4.37 [1.23, 7.50]	4.02 [0.94, 7.10]
Sexually abused		8.21 [3.14, 13.27]	7.94 [2.88, 13.01]	7.52 [2.36, 12.69]
Physically abused		6.42 [2.05, 10.79]	6.09 [1.76, 10.43]	5.55 [1.21, 9.90]
Adult SES: GSC I				*Ref.*
Adult SES: GSC II				1.24 [-1.19, 3.67]
Adult SES: GSC III				3.99 [1.70, 6.29]
Adult SES: GSC IV				5.00 [-1.03, 11.04]
Adult SES: GSC V				3.86 [-1.74, 9.47]
Adult SES: GSC VI				5.94 [1.91, 9.97]
Adult SES: GSC VII				15.78 [12.08, 19.47]

Note: average marginal effects (multiplied by 100), with 95% confidence intervals in brackets.

Since odds ratios are influenced by the amount of unobserved heterogeneity in a model, 'attenuation' and 'persistence' are more justifiably invoked by examining changes across models in average marginal effects (Mood, 2010). The average marginal effects from each model are shown in Table 5 and Table 6. Multiplied by 100 as shown, these can be interpreted as percentage point changes in the probability of high psychological distress. These results tell the same story as the odds ratios presented above. It is clear that, averaged across all individuals in the female sample, CSES is associated with a substantial probability difference. A woman born into the lowest parental social class has a probability around 4.5 percentage points higher of experiencing high psychological distress at age 33, net of a wide range of childhood adversities, and net too of her current social class. This is comparable to the difference between one's current, adult social class being VI, the second lowest, rather than I, the highest.

I repeat the regression models (as linear regressions) using a continuous operationalisation of the outcome. Results are shown in supplementary tables in the Appendix (Tables S1 and S2). The findings closely follow those shown above from the dichotomous operationalisation of the mental health outcome. Two differences are noteworthy. First, in the model for men, the continuous results show a large, positive coefficient for reporting having been sexually abused. By contrast, in the logistic regression results the odds ratios are close to 1 for men. The instability in these estimates is not surprising if we consider that only 24 male respondents report this CA. It is by some distance the least frequent(-ly reported) CA for either sex, in addition to being one of the variables with the most missing data. This warrants agnosticism in the interpretation of the coefficients for sexual abuse among men. Second, in the continuous analyses, a second coefficient differs significantly between men and women: adult social class VII. Among women, this class position is associated with 1.83 more positive answers to items on the malaise inventory (1.30 for men), compared with being in the highest social class in adulthood. As regards the two research questions examined so far, this does not alter the findings.

We have seen that there are some clear differences between men and women both in the levels of exposure to the risk factors measured here, and in those risk factors' strength of association with poor mental health in adulthood. Lastly, I explore to what extent the higher prevalence of psychological distress at age 33 amongst women can be attributed

to differential exposure versus differential vulnerability, using a Oaxaca-Blinder counterfactual decomposition. The results are essentially the same whether the dichotomous or continuous version of the outcome is used. For ease of interpretation I present the latter. Results are shown in Table 7.

Table 7 Oaxaca-Blinder decomposition of the mean difference in number of malaise symptoms

Mean malaise score: women	2.96	[2.88, 3.03]
Mean malaise score: men	2.17	[2.09, 2.24]
Difference	0.79	[0.68, 0.90]
attributable to endowments	-0.01	[-0.08, 0.05]
attributable to coefficients	0.84	[0.72, 0.96]
attributable to interaction of endowments and coefficients	-0.04	[-0.11, 0.04]
Endowments		
Neglected	-0.01	[-0.02, -0.00]
Bullied at school	-0.01	[-0.02, -0.00]
Sexually abused	0.05	[0.01, 0.08]
GSC II	-0.01	[-0.01, -0.00]
GSC VII	-0.02	[-0.03, -0.01]
Coefficients		
CSES: I/II	-0.05	[-0.09, -0.01]
CSES: IV/V	0.06	[0.01, 0.11]
GSC: VII	0.06	[0.01, 0.10]
Constant	0.88	[0.69, 1.07]

Note: 95% confidence intervals in brackets. For interpretation refer to text. Under *endowments* and *coefficients*, only values significant at the 0.05 level are reported. All independent variables from model 4 are included in the estimation.

The first two rows report the means of the number of malaise symptoms for men and women. These differ slightly from the values given earlier because they are estimated from the multiply imputed data. Below this is the difference between the two group means. *Attributable to endowments* refers to the expected change in the mean of malaise symptoms for men, under a counterfactual in which men have the prevalence of (that is, the level of exposure to) the independent variables which is observed for women. That is, what difference would it make to the gender difference in psychological distress, if as many men as women were exposed to sexual abuse (etc.)? *Attributable to coefficients* refers to the expected change in the mean, under the counterfactual in which the coefficient for each of the independent variables for men is that which is estimated for women. That is, what if childhood SES (etc.) had as strong an association with adult mental health among men as it does among women? *Attributable to interaction of endowments and coefficients* 'account[s] for the fact that differences in endowments and coefficients exist simultaneously between the two groups' (Jann, 2008: 455). Below this, I report the values *attributable to*, disaggregated into the contribution of each independent variable (showing only those which are significant at the 0.05 level).

At first glance, the results appear to support the vulnerability hypothesis: the mean difference in symptom count is wholly attributable to differences in the coefficients between men and women. However, the disaggregated results show that none of the measured independent variables in fact accounts for much of the difference. Rather, the difference is almost wholly attributed to the constant term; that is, to men and women's differing intercepts. Neither the exposure hypothesis nor the vulnerability hypothesis is supported. The observed variables do not explain the gender difference in psychological distress in adulthood.

2.8 Discussion

This chapter sought to address some questions regarding the relation of the childhood environment to mental health in adulthood for which the existing evidence is mixed. I aimed to explore, first, whether the association between low socioeconomic status in childhood and poor mental health in adulthood was in fact accounted for by the greater prevalence in low-SES households of certain highly stressful noneconomic events or

conditions. Second, I aimed to explore whether any associations between childhood conditions and mental health in adulthood were independent of adult SES or mediated by adult SES. Third, I sought to explore whether different levels of exposure or vulnerability to low childhood socioeconomic status and childhood adversities could account for the stark gender difference in the level of psychological distress (measured as symptoms of depression and anxiety) at age 33.

With regard to the question of which aspects of the childhood environment matter for mental health in adulthood, these results indicate the importance of both a wide range of childhood adversities and childhood SES for women, but more exclusively childhood adversities for men. For men, the bivariate CSES coefficients are relatively small, and once CAs are brought into the model, are largely attenuated. On the other hand, for both men and women, the coefficients of the childhood adversity measures generally attenuate very little across models. As expected, almost all of the childhood adversities show substantial associations with later poor mental health. Being neglected – that is, appearing so to a teacher, having a parent suffering from mental illness, being frequently bullied at school, living in a house fraught with 'domestic tension' (according to a health visitor), and being sexually or physically abused are each strong predictors of high psychological distress in adulthood.

Are these associations independent of later life SES, as the critical period hypothesis states, or mediated by the status attainment process? Or do they not matter at all, conditional on adult SES? For men, the already-attenuated CSES coefficients attenuate further and become indistinguishable from zero when adult SES is added to the model. For women, there is some attenuation in the CSES coefficients when adult SES is added, but a substantial part of the association remains. The attenuation here suggests that low CSES contributes to poor mental health in adulthood partially through the intergenerational transmission of status: those with low origin social class are likely to have low destination social class, and low destination social class is associated with poor mental health. There is thus modest support for the status attainment mediation hypothesis.

Of course, support for status attainment mediation as a pathway does not preclude the possibility of critical or sensitive period effects which are not mediated in this way.

Indeed, among women, there remains a substantial and statistically significant association between CSES and psychological distress in the full model including adult SES. This suggests a lasting influence of social background on women which is accounted for neither by the measured CAs nor by the status attainment process. Indeed, the coefficient for the lowest CSES group differed significantly between men and women in the full model including adult SES. No previous study has found such an association to hold independent of a broad range of CAs including even the most extreme, and independent also of one's own adult SES. Power et al. (2002) note the apparent vulnerability of women in this cohort to socioeconomic circumstances in childhood, but only examine the bivariate association between CSES and later malaise.

For both men and women, the associations between the CAs and mental health in adulthood mostly show negligible attenuation when adult SES is added, further supporting the hypothesis of childhood as a critical or sensitive period shaping individuals' mental health in later life.

With regard to the question of exposure and vulnerability as potential explanations for the gender difference in psychological distress in adulthood, the findings indicated that neither could account for a substantial portion of the gender difference. Rather, the generally greater number of symptoms of depression and anxiety experienced by women was left unexplained by the observed variables.

A strength of these data is the wide range of CAs measured. For instance, being a victim of bullying is one of the most consistently important CAs across the whole sample. This echoes recent work demonstrating strong effects of bullying in this sample across multiple outcomes (Takizawa et al., 2014), and another recent study suggesting that bullying may be especially important even relative to other major childhood adversities – though it is compared with parental maltreatment *reported by parents* and thus probably measured with considerable error (Lereya et al., 2015). This is an important new direction given that bullying has gone unmeasured in many surveys of childhood adversity and its relationship with adult mental health (Schilling et al., 2008), including such major undertakings as the National Comorbidity Study Replication (Green et al., 2010) and the WHO World Mental Health Survey Initiative (Kessler et al., 2010); it has also been

widely neglected even in work that uses the NCDS (Clark et al., 2010; Hatch et al., 2010; Power et al., 2002).

Some childhood adversities showed unexpectedly weak associations with later mental health. While it is advantageous to have prospective measures, what precisely is being captured is in several cases not entirely clear, making measurement error inevitable and targets for intervention and future study vague. For instance, the indicators of family criminality are rather broad and oblique, and offer little insight into what exposure this might entail for the child. This is the case even for certain indicators which did exhibit associations with the outcome, such as the health visitor's report of 'domestic tension', which seems highly subjective, and could be proxying for some combination of household chaos, the interpersonal conflict styles of parents, acute financial difficulty, and domestic violence. 'Family mental illness' leaves unclear who is suffering and what they are suffering from. Further, separation from one's biological mother or father are not a priori stressful given a capable adoptive, grand-, step-, or foster parent; a better alternative might have been to base the measure on the answer category *no mother figure / no father figure*. These likely sources of measurement error in the CAs also warrant a cautious interpretation of the result that they do not fully account for the association between CSES and later mental health.

Other retrospective measures available in the data may appear to offer a valuable source of more specific information about childhood exposures. These include items asking cohort members about substance abuse, problem drinking, and mental health issues for each parent separately, as well as questions on conflict and tension, and explicitly asking about neglect. However, there are problems with these retrospective items. Where prospective and retrospective items nominally measure the same CA, correlations between the two are rather low (e.g. 0.14 for household conflict and tension, 0.05 and 0.04 for neglect). Several retrospective items reflect household or parental characteristics rather than being specific to the child (e.g. *Did your mother have trouble with drinking?*). Hence their prevalence is not expected to vary with child characteristics such as gender. Retrospective items measuring family mental illness, domestic tension and parental substance abuse are examples of such items. However positive answers are substantially more frequent among women in the sample, raising concerns about gender bias in retrospective reporting.

These problems likely extend, to some extent, to the included measures of sexual and physical abuse. Indeed only a tiny proportion of men reported sexual abuse, despite the self-completion format of the questionnaire module. Further, such retrospective reports may be influenced by current psychological distress. It may partly ameliorate this issue that the retrospective reports are taken from 12 years *after* the outcome measure is recorded. Evidence on retrospective reports of adversities does at least lead Hardt and Rutter to declare that 'major adversities of an easily defined kind' (2004: 260) can validly and reliably be measured in this way. Sexual and physical abuse, as acts that are perpetrated on the child, rather than opaquely observable characteristics of the parent(s) or household in general, seem less susceptible to the sorts of biases that might arise.

Relatedly, a potential concern with respect to the question of the relative importance of CSES and CAs is that some major CA may have gone unmeasured. Schilling demonstrates that the co-occurrence of a high number of CAs is disproportionately associated with experience of the most severe CAs: '[high impact events] tend to occur in the context of high cumulative adversity' (Schilling et al., 2008: 1147). So an effect of some unmeasured adversity is at least likely to be attributed to one or another CA rather than CSES.

Given the focus on the possible effects of early life conditions on outcomes measured in later life, the missing data strategy warrants some discussion. An alternative to standard multiple imputation which I considered is the 'IPW/MI' procedure of Seaman et al., in which inverse probability weighting (IPW) is applied to adjust for attrition, while multiple imputation (MI) is used for item missingness within cases included in the analytical sample (Seaman et al., 2012; Seaman & White, 2013). That is, cases included in the analysis – those observed on the outcome (psychological distress at age 33) – are weighted by the inverse of their probability of being observed on the outcome, which is modelled with predictors of missingness from earlier sweeps. This offers advantages. There is no risk of a poorly-specified imputation model substantially biasing the analysis. The joint distribution of variables where they are observed is not extrapolated to the cases where those variables are unobserved, as in MI; rather, the included cases which are most like the excluded cases (according to the observed predictors of missingness) are more heavily weighted.

On the other hand, there are strong arguments in favour of multiple imputation. First, MI is generally more efficient than any procedure involving IPW. Second, the cases that are excluded under IPW/MI because of missingness on the outcome are mostly non-missing on variables measured between birth and age 16: at age 11, 92% of the eligible sample participated, at age 16, this falls only to 87%. (And to 71% at age 33). So it is not generally the case that those excluded under IPW/MI are missing on most or even many variables. With MI, these cases contribute information about the joint distribution of the variables measured from birth to age 16. Under IPW/MI they are ignored, except that they contribute information on how the included cases are to be weighted. Third, MI does not suffer the problem of missing predictors of missingness, the approach to which outlined in Seaman & White (2013) is, in Seaman's words, 'some ad hoc method' (personal communication), and 'imperfect and we do not recommend it for general use' (Seaman et al., 2012: 135). Fourth, MI allows the use of auxiliary variables, which in this case are likely to be highly informative. For instance there are measures of dimensions of mental health at ages 7, 11, and 16; and at age 23 psychological distress is measured in the same way as at age 33 (i.e. the outcome). The strong continuity of adolescent mental health into adulthood is well-documented (Kim-Cohen et al., 2003).

It is further worth noting that the inclusion of strong auxiliary variables makes estimation including cases with imputed values of the dependent variable acceptable, and arguably preferable (White et al., 2011). The difference between including and excluding cases with an imputed dependent variable from the analysis is not generally large with 20 or more imputed datasets (Johnson & Young, 2011; von Hippel, 2007). Results (not shown) are highly similar if such cases are excluded. For instance the odds ratio of 1.60 for CSES IV/V (versus I/II) for women in model 4 becomes 1.61 when excluding these cases, with the same size standard error.

The childhood environment may exert effects on later mental health well before the age of 33. Poor mental health in early adulthood may in turn influence adult SES at age 33. Such selection effects are not modelled here, but would act to strengthen the association between mental health at age 33 and contemporaneous SES. To the extent that such a process occurs, the coefficients for CSES and the CAs in model 4 will be subject to a downward bias.

Possible sources of measurement error in CSES and the CAs have been mentioned. These might also act to suppress relationships between these variables and the outcome. On the other hand, adult SES is measured with only a single variable, though one with seven categories and which theoretically captures SES in a quite holistic sense. Measurement error in adult SES here might make the childhood environment appear more important than it in fact is. A specific mechanism for measurement error in adult SES arises from the fact that social class is measured using the dominance method. Individuals whose own occupation is subsumed by that of their partner may have quite different work situations (if they work at all) than their measured class implies.

A second possible source of measurement error in adult SES is as follows. While a varying risk of unemployment is inherent in Goldthorpe's differentiation of social classes on the basis of modes of employment relations, it is nevertheless the case that an individual of a given class who is unemployed at a particular time is likely to have worse mental health at that time than one who is not. Indeed, Richards and Paskov (2016) find that controlling for economic status accounts for the social class gradient in mental health in a UK sample. Further, the review of Fryers et al. (2003) finds that social class is less strongly associated with the common mental disorders than other indicators such as unemployment. I therefore test whether the results change markedly when controlling also for economic status at age 33 (not shown). While being unemployed, sick/disabled, or looking after the home/family each show strong associations with psychological distress relative to being employed, adding this variable to model 4 has remarkably little effect on the results. For instance, the 1.60 odds ratio for women's low CSES cited above becomes 1.56 with this extra covariate.

Finally, this chapter is limited in the extent to which it captures poor mental health in adulthood, since it only measures one outcome. To the extent that the childhood environment influences other conditions – both those widely accepted as falling within the bounds of what is defined by 'mental health' (e.g. schizophrenia), and those on the fringes of this contested concept (e.g. antisocial personality disorder) – the effects of the factors examined here on mental health *in toto* are underestimated (Aneshensel, 2002). On the other hand, a score of 7 or more on the malaise inventory is multiply realisable,

reflecting a range of manifestations of psychological distress, and plausibly captures a large portion of poor mental health.

In conclusion, it appears that for women, but not men, the social class one is born into is substantively associated with one's chance of suffering high psychological distress in adulthood, and that this is accounted for neither by a greater risk of suffering severe, noneconomic childhood adversities, nor by a greater risk of reaching a low SES position in adulthood. These results indicate a need to increase our understanding both of gender differences in the causes of psychological distress, and of the subtler ways in which a non-traumatic but disadvantaged childhood may have far-reaching mental health consequences.

2.9 Appendix

Auxiliary variables

Measured at birth:
- mother left school at the end of compulsory education
- mother smoked while pregnant
- teenage mother
- parents not in a stable relationship

Measured at age 7:
- housing tenure
- externalising problems (teacher-rated)
- internalising problems (teacher-rated)
- low reading score
- low maths score
- short stature
- number of family moves
- social class based on father's occupation

Measured at age 11:
- low reading score
- internalising problems (teacher-rated)
- externalising problems (teacher-rated)
- number of family moves
- social class based on father's occupation

Measured at age 16:
- conduct problems (teacher-rated)
- emotional problems (teacher-rated)
- social class based on father's occupation

Measured at age 23:
- malaise inventory (continuous)
- economic status
- social class based on own occupation
- marital status

Measured at age 42:
- economic status
- social class based on own occupation
- marital status

The malaise inventory

‘These questions are concerned with how you are feeling generally. Please answer them by ticking either the ‘Yes’ or ‘No’ box for each one. It is important that you try to answer ALL the questions.

1. Do you often have back-ache?
2. Do you feel tired most of the time?
3. Do you often feel miserable or depressed?
4. Do you often have bad head-aches?
5. Do you often get worried about things?
6. Do you usually have great difficulty in falling or staying asleep?
7. Do you usually wake unnecessarily early in the morning?
8. Do you wear yourself out worrying about your health?
9. Do you often get into a violent rage?
10. Do people often annoy and irritate you?
11. Have you at times had a twitching of the face, head or shoulders?
12. Do you often suddenly become scared for no good reason?
13. Are you scared to be alone when there are not friends near you?
14. Are you easily upset or irritated?
15. Are you frightened of going out alone or of meeting people?
16. Are you constantly keyed up and jittery?
17. Do you suffer from indigestion?
18. Do you suffer from an upset stomach?
19. Is your appetite poor?
20. Does every little thing get on your nerves and wear you out?
21. Does your heart often race like mad?
22. Do you often have bad pains in you [sic] eyes?
23. Are you troubled with rheumatism or fibrosis?
24. Have you ever had a nervous breakdown?’

Table S1 Linear regression results (outcome: psychological distress as indicated by number of malaise symptoms), men

	(1)	(2)	(3)	(4)
CSES: I/II	*Ref.*		*Ref.*	*Ref.*
CSES: III non-manual	0.21		0.04	-0.01
	(0.14)		(0.14)	(0.14)
CSES: III manual	0.50***		0.29**	0.12
	(0.10)		(0.10)	(0.10)
CSES: IV/V†	0.72***		0.31*	0.03
	(0.13)		(0.13)	(0.13)
Ever in care		0.87***	0.86***	0.76***
		(0.20)	(0.20)	(0.20)
Neglected		0.75***	0.72***	0.53***
		(0.15)	(0.15)	(0.15)
Family criminality		0.47**	0.44**	0.35*
		(0.15)	(0.15)	(0.15)
Separated from biological mother		0.19	0.18	0.18
		(0.16)	(0.16)	(0.16)
Separated from biological father		0.16	0.15	0.14
		(0.11)	(0.11)	(0.10)
Family mental illness		0.73***	0.72***	0.67***
		(0.16)	(0.16)	(0.16)
Bullied at school		0.68***	0.65***	0.58***
		(0.12)	(0.13)	(0.12)
Domestic tension		0.56**	0.55**	0.49**
		(0.18)	(0.18)	(0.18)
Sexually abused		1.96*	1.94*	1.89*
		(0.75)	(0.75)	(0.74)
Physically abused		1.32***	1.31***	1.25***
		(0.22)	(0.22)	(0.21)
Adult SES: GSC I				*Ref.*
Adult SES: GSC II				0.09
				(0.10)
Adult SES: GSC III				0.47***
				(0.11)
Adult SES: GSC IV				0.38*
				(0.17)
Adult SES: GSC V				0.44
				(0.23)
Adult SES: GSC VI				0.56***
				(0.13)
Adult SES: GSC VII†				1.30***
				(0.12)

Note: standard errors in parentheses.
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$
† model 4 coefficient significantly differs by sex at 0.05 level.

Table S2 Linear regression results (outcome: psychological distress as indicated by number of malaise symptoms), women

	(1)	(2)	(3)	(4)
CSES: I/II	*Ref.*		*Ref.*	*Ref.*
CSES: III non-manual	0.55***		0.41**	0.34*
	(0.16)		(0.15)	(0.15)
CSES: III manual	0.81***		0.62***	0.39***
	(0.11)		(0.11)	(0.11)
CSES: IV/V†	1.31***		0.91***	0.60***
	(0.13)		(0.13)	(0.13)
Ever in care		0.41	0.35	0.30
		(0.23)	(0.23)	(0.22)
Neglected		0.89***	0.78***	0.53*
		(0.22)	(0.22)	(0.22)
Family criminality		0.41	0.34	0.20
		(0.21)	(0.21)	(0.22)
Separated from biological mother		0.30	0.28	0.26
		(0.19)	(0.19)	(0.18)
Separated from biological father		0.15	0.11	0.08
		(0.10)	(0.10)	(0.10)
Family mental illness		0.50**	0.50**	0.43*
		(0.18)	(0.18)	(0.18)
Bullied at school		0.96***	0.89***	0.82***
		(0.17)	(0.17)	(0.17)
Domestic tension		0.74**	0.71**	0.66**
		(0.24)	(0.24)	(0.24)
Sexually abused		1.43***	1.39***	1.31***
		(0.34)	(0.34)	(0.33)
Physically abused		1.35***	1.31***	1.23***
		(0.19)	(0.19)	(0.18)
Adult SES: GSC I				*Ref.*
Adult SES: GSC II				0.21
				(0.12)
Adult SES: GSC III				0.52***
				(0.11)
Adult SES: GSC IV				0.47
				(0.29)
Adult SES: GSC V				0.56*
				(0.27)
Adult SES: GSC VI				0.81***
				(0.19)
Adult SES: GSC VII†				1.83***
				(0.15)

Note: standard errors in parentheses.
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$
† model 4 coefficient significantly differs by sex at 0.05 level.

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

3.1 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 et al., 1999; Power et al., 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 et al., 2013: 219). Another is that 'men and women with pre-existing illness drift down the social scale' (Power et al., 2002: 1989). Prominent recent work has highlighted the impact of health selection according to childhood mental health, reporting 'large effects on [inter alia] … intergenerational and within-generation social mobility' for the NCDS cohort in the UK (Goodman et al., 2011: 6032), and a 'total lifetime economic cost for all those affected of 2.1 trillion dollars' in the US (Smith & Smith, 2010: 110).

To what extent is health selection driven by prior social causation? This chapter 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. The first – (a) in Figure 1 – is that childhood factors may confound the relationship between adolescent mental health and

SES in young adulthood. Despite 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 (Lundberg, 1991; Wilkinson, 1986). The second – (b) in Figure 1 – 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*. This chapter evaluates the joint contribution of these processes, but does not disentangle the two.

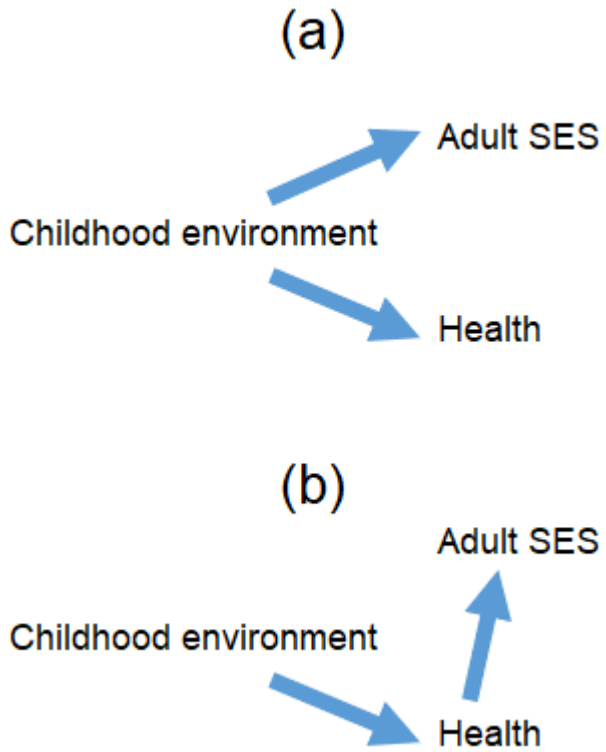


Figure 1 (a) Confounding / 'indirect health selection' (b) Mediated social causation

Health selection may occur both between and within generations. This chapter 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 et al., 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 et al., 2003).

In sum, this chapter 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?

3.2 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 (Cai & Kalb, 2006; Haan & Myck, 2009; Kröger et al., 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 and colleagues 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).

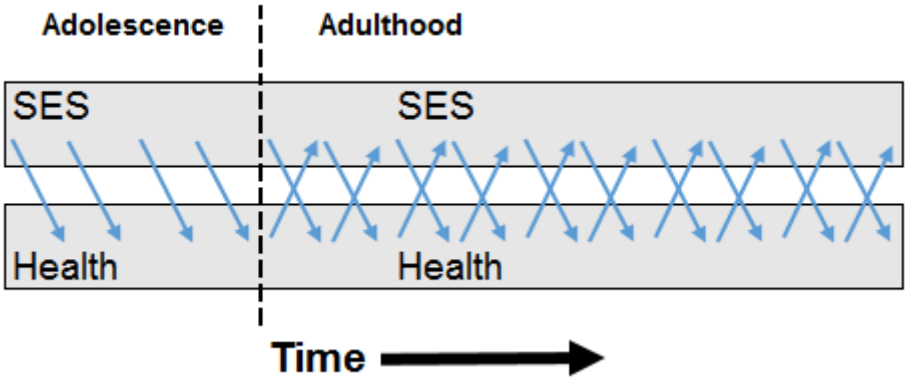


Figure 2 Conceptual model of life course causal relations between SES and health

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 one's SES for the first time. While it may affect intermediate status attainment outcomes such as educational achievement in school (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.

3.3 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 et al., 2011; Bolger et al., 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 et al., 1994; Mills-Koonce 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 (Goodman et al., 2011; Kawakami et al., 2012; Schoon et al., 2003) and with educational attainment (Breslau et al., 2008; Kessler et al., 1995; McLeod et al., 2012; McLeod & Fettes, 2007), a key mediator in the status attainment process.

There is less consensus on the relative importance of different dimensions of adolescent mental health for socioeconomic outcomes. 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 (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 chapter. 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 (McLeod & Kaiser, 2004: 640; Stansfeld et al., 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 and colleagues 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.
> (Schoon et al., 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 versus 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 and 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 and 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 chapter 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 chapter 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).

3.4 Data & measures

3.4.1 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, 10,986 cohort members were interviewed (70.6% of the target sample). These participants are considered to be generally representative of the original cohort (Ferri, 1993).

3.4.2 Dependent variable: intergenerational social class mobility

Intergenerational social class mobility is used to indicate socioeconomic attainment. Change in the British class structure, in particular the growth of managerial and professional occupations, afforded this cohort a high degree of opportunity for upward intergenerational mobility, despite their entry into the labour market coinciding with a recession (Bukodi & Goldthorpe, 2011, 2019).

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; managerial and technical), III non-manual (skilled non-manual), III manual (skilled manual), and IV/V (semi-skilled manual; unskilled manual). The two binary outcomes of upward (versus not upward) and downward (versus not downward) intergenerational social class mobility are determined according to this four-category system.

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

Slightly modified versions of the Rutter behaviour scales are available in the data (Rutter et al., 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 (Goodman et al., 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*). These 13 items are given in full in the Appendix to this chapter. To account for measurement error, a latent variable modelling approach is taken, and a confirmatory factor analysis conducted using the lavaan package in R (Rosseel, 2012) (see Figure S1 in the Appendix). For identification, the variances of the latent variables are 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% CI 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.

3.4.4 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 Appendix.

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

Childhood adversities (CAs) are highly stressful events or conditions which are beyond the child's control (Clark et al., 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. The indicators used are the same as in Chapter 2.

3.5 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 (see Figure 3), whilst adjusting for the rescaling of the coefficients due to the need to fix the residual variance of the latent outcome (Karlson et al., 2012; Kohler & Karlson, 2010). In the reduced model, the three 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 mediated social causation (as defined above: 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.

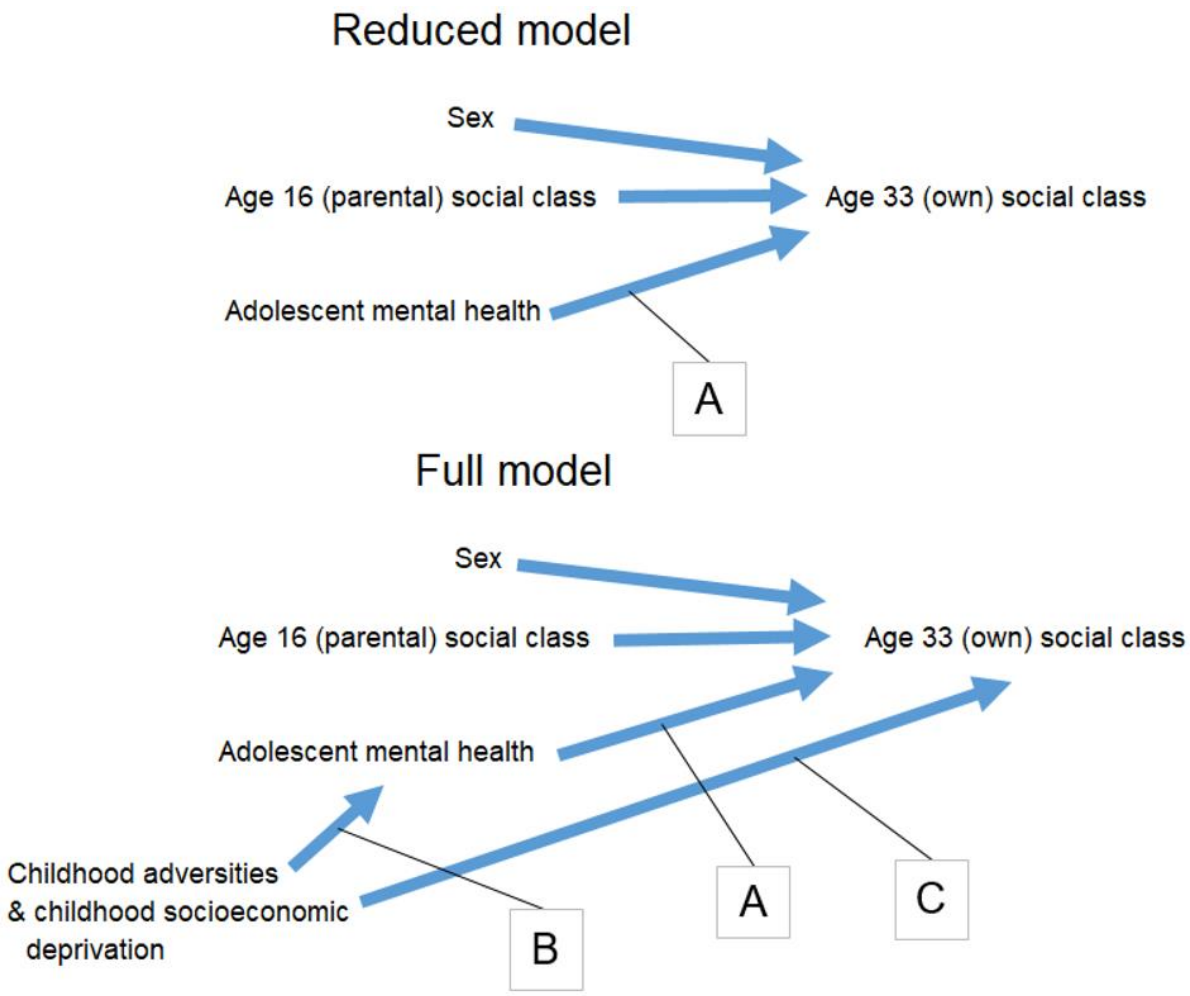


Figure 3 Reduced and full analytical models

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 destination class 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* command in Stata (Royston & White, 2011). 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^{th} 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.

3.6 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 25th, 50th, and 75th 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, even given that the comparison is between the occupations of fathers of cohort members at age 16, and respondents' own occupations at age 33, there is substantial growth in the professional, managerial, and technical 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 Table S1 in the Appendix. The large amount of observed data in non-complete cases supports the use of multiple imputation.

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

	Destination social class (age 33)					
Origin social class (age 16)	I/II	IIINM	IIIM	IV/V	Total	%
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		

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 nonsignificant.

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 nonsignificant 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 models for emotional symptoms or hyperactivity.

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

		(a) Outcome: upward mobility								(b) Outcome: downward mobility							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct	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
problems	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	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
symptoms	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		

Sample size (number potentially upwardly/downwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 13184-13301 (b) 13343-13469.
Not shown: concomitant variables (full & reduced models): female, origin class; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Results of the robustness checks specified above (Tables S2-S7 in the Appendix) 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 nonsignificant (OR 1.21, 95% CI 0.99-1.48). The substantive conclusions appear robust across all four specifications.

3.7 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 chapter 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 observed 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 environmental 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 et al., 2018: 251). 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 et al., 2006). Tables S8 and S9 in the Appendix 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 chapter 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 Tables S10 and S11) showed highly similar results except that, even in the reduced model, the effect of conduct problems on upward mobility was nonsignificant (OR 0.83, 95% CI 0.66-1.04), while emotional symptoms showed no effect on mobility (upward mobility: OR 0.92, 95% CI 0.81-1.05; downward mobility: OR 1.14, 95% CI 1.00-1.30). 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 are very similar, and there is an identical pattern of which effects on mobility attenuate 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 et al., 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 both environmental and genetic pathways, as discussed above.

Third, interpretation of the AMH factor scores is slightly complicated by the fact that the standard deviations 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: 0.84, emotional symptoms: 0.62, hyperactivity: 0.87), 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: -0.02, hyperactivity: -0.05) 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.

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 nonsignificant.

Table S14 details to what extent each of the childhood environment controls accounts for the attenuation of the conduct problems coefficients in the main analysis. 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.

3.8 Appendix

Items from the Rutter behaviour scales

Wording from the parent questionnaire. Identical or negligibly different in the teacher questionnaire unless indicated.

Conduct problems dimension

- Does he/she ever steal things? [Teacher questionnaire: Has stolen things on one or more occasions in the past 12 months]
- Often destroys own or others' property
- Is often disobedient
- Often tells lies
- Bullies other children
- Frequently fights or is extremely quarrelsome with other children
- Irritable. Is quick to 'fly off the handle' [Teacher questionnaire: Irritable, touchy, is quick to 'fly off the handle']

Emotional symptoms dimension

- Often worried, worries about many things
- Tends to be fearful or afraid of new things or new situations
- Tends to do things on own – rather solitary [Teacher questionnaire: Tends to be on own – rather solitary]

Hyperactivity dimension

- Squirmy, fidgety child
- Very restless. Has difficulty staying seated for long
- Cannot settle to anything for more than a few moments

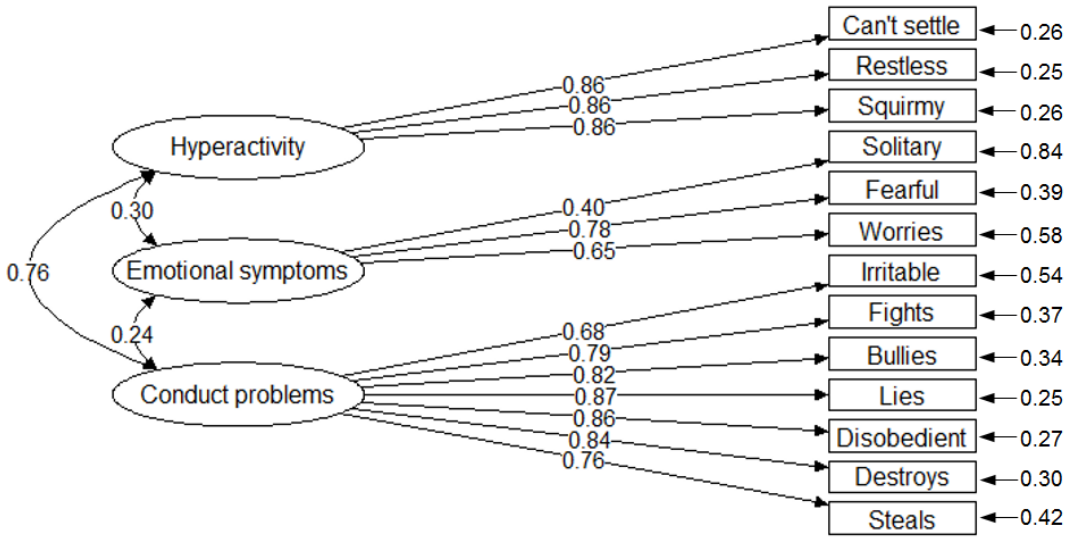


Figure S1 Confirmatory factor analysis of the Rutter behaviour scales

Childhood socioeconomic deprivation indicators

22 dichotomous indicators of material hardship, from across 4 sweeps, are summed to give a childhood socioeconomic deprivation (CSD) score. These are:

From birth: parents not in a stable relationship, teenage mother, low parental social class (RGSC IV/V, or 'no male head of household'), overcrowding (>1.5 persons per room).

From age 7: low parental social class, overcrowding, housing tenure (not owner occupier), unemployment, financial problems (in this sweep, the presence of unemployment and financial problems was determined by a health visitor without directly questioning the family, but who had access to local authority records).

From age 11: low parental social class, overcrowding, housing tenure, unemployment (claiming unemployment benefit), financial problems (according to parent), free school meals, child shares bedroom.

From age 16: overcrowding, housing tenure, unemployment (claiming unemployment benefit), financial problems (according to parent), free school meals, child shares bedroom.

Table S1 Descriptive statistics

	Complete cases (*N*=3321)				Observed data					Multiply imputed data		
Variable	**Mean**	**SD**	**Min**	**Max**	**Mean**	**SD**	**Min**	**Max**	**N**	**Mean**	**95% CI**	
Upward mobility	0.57	0.49	0	1	0.56	0.50	0	1	5543	0.54	0.53	0.55
Downward mobility	0.30	0.46	0	1	0.31	0.46	0	1	6056	0.32	0.31	0.33
Origin class												
I/II	0.29	0.45	0	1	0.25	0.43	0	1	11170	0.24	0.24	0.25
IIINM	0.11	0.31	0	1	0.11	0.31	0	1	11170	0.11	0.10	0.11
IIIM	0.42	0.49	0	1	0.42	0.49	0	1	11170	0.41	0.40	0.42
IV/V	0.18	0.39	0	1	0.22	0.42	0	1	11170	0.23	0.23	0.24
Destination class												
I/II	0.38	0.48	0	1	0.36	0.48	0	1	10582	0.34	0.33	0.35
IIINM	0.24	0.43	0	1	0.24	0.42	0	1	10582	0.23	0.22	0.23
IIIM	0.20	0.40	0	1	0.20	0.40	0	1	10582	0.21	0.21	0.22
IV/V	0.18	0.39	0	1	0.20	0.40	0	1	10582	0.22	0.21	0.23
Female	0.51	0.50	0	1	0.48	0.50	0	1	18554	0.49	0.48	0.49
Adolescent mental health												
Conduct problems	0.02	0.70	-0.75	4.14	0.13	0.76	-0.75	4.25	9313	0.16	0.14	0.17
Emotional symptoms	0.01	0.71	-0.96	3.32	0.06	0.73	-0.96	3.32	9313	0.07	0.05	0.08
Hyperactivity	0.04	0.70	-0.73	3.32	0.13	0.75	-0.73	3.76	9313	0.16	0.15	0.17
Childhood socioeconomic deprivation	3.90	3.37	0	21	4.32	3.64	0	21	6841	4.76	4.70	4.82
Birth: parents not in stable relationsh	0.02	0.14	0	1	0.04	0.19	0	1	17405	0.04	0.04	0.04
Birth: teenage mother	0.04	0.20	0	1	0.06	0.23	0	1	17401	0.06	0.06	0.06
Birth: low social class	0.20	0.40	0	1	0.26	0.44	0	1	17415	0.26	0.26	0.27
Birth: overcrowding	0.11	0.31	0	1	0.14	0.35	0	1	16920	0.15	0.14	0.15
Age 7: low social class	0.23	0.42	0	1	0.31	0.46	0	1	15425	0.31	0.30	0.32
Age 7: overcrowding	0.12	0.32	0	1	0.15	0.35	0	1	13952	0.15	0.15	0.16
Age 7: housing tenure	0.54	0.50	0	1	0.58	0.49	0	1	14550	0.58	0.57	0.59
Age 7: unemployment	0.02	0.14	0	1	0.04	0.19	0	1	13586	0.04	0.04	0.04
Age 7: financial problems	0.05	0.22	0	1	0.08	0.28	0	1	13063	0.09	0.09	0.10
Age 11: low social class	0.22	0.41	0	1	0.34	0.47	0	1	15335	0.34	0.33	0.35
Age 11: overcrowding	0.10	0.30	0	1	0.12	0.33	0	1	13792	0.12	0.12	0.13
Age 11: housing tenure	0.51	0.50	0	1	0.54	0.50	0	1	13799	0.55	0.54	0.55
Age 11: unemployment	0.06	0.23	0	1	0.08	0.27	0	1	13533	0.08	0.07	0.08
Age 11: financial problems	0.08	0.27	0	1	0.11	0.31	0	1	13603	0.11	0.11	0.12
Age 11: free school meals	0.08	0.27	0	1	0.12	0.33	0	1	15055	0.12	0.11	0.12
Age 11: shares bedroom	0.54	0.50	0	1	0.56	0.50	0	1	13585	0.56	0.56	0.57
Age 16: overcrowding	0.03	0.18	0	1	0.05	0.23	0	1	11514	0.05	0.05	0.06
Age 16: housing tenure	0.45	0.50	0	1	0.50	0.50	0	1	11653	0.50	0.49	0.51
Age 16: unemployment	0.03	0.18	0	1	0.05	0.21	0	1	11599	0.04	0.04	0.05
Age 16: financial problems	0.06	0.24	0	1	0.10	0.31	0	1	11518	0.11	0.10	0.11
Age 16: free school meals	0.05	0.23	0	1	0.10	0.30	0	1	11508	0.10	0.10	0.11
Age 16: shares bedroom	0.36	0.48	0	1	0.39	0.49	0	1	11629	0.39	0.38	0.40
Childhood adversities												
Taken into care	0.02	0.15	0	1	0.05	0.22	0	1	11065	0.05	0.04	0.05
Birth - age 7	0.01	0.10	0	1	0.02	0.15	0	1	14667	0.02	0.02	0.03
Age 7 - 11	0.01	0.09	0	1	0.02	0.12	0	1	13280	0.02	0.01	0.02
Age 11 - 16	0.01	0.10	0	1	0.03	0.16	0	1	11142	0.03	0.03	0.03
Neglected appearance	0.04	0.20	0	1	0.07	0.25	0	1	14123	0.07	0.06	0.07
Age 7	0.02	0.15	0	1	0.04	0.20	0	1	15425	0.04	0.04	0.04
Age 11	0.02	0.15	0	1	0.04	0.19	0	1	15335	0.04	0.03	0.04
Family criminality	0.04	0.20	0	1	0.06	0.24	0	1	10732	0.06	0.06	0.07
Age 7	0.01	0.09	0	1	0.02	0.14	0	1	13032	0.02	0.02	0.03
Age 11	0.01	0.10	0	1	0.02	0.14	0	1	15335	0.02	0.02	0.02
Age 16	0.03	0.17	0	1	0.03	0.18	0	1	14653	0.03	0.03	0.04
Separation from biological mother	0.04	0.19	0	1	0.05	0.21	0	1	9246	0.06	0.06	0.07
Age 7	0.01	0.12	0	1	0.03	0.17	0	1	14703	0.03	0.03	0.03
Age 11	0.02	0.14	0	1	0.04	0.19	0	1	13858	0.04	0.04	0.04
Age 16	0.04	0.19	0	1	0.05	0.22	0	1	11690	0.06	0.05	0.06
Separation from biological father	0.08	0.26	0	1	0.13	0.34	0	1	9240	0.15	0.14	0.16
Age 7	0.03	0.18	0	1	0.07	0.26	0	1	14701	0.08	0.07	0.08
Age 11	0.05	0.22	0	1	0.10	0.30	0	1	13858	0.10	0.10	0.11
Age 16	0.07	0.26	0	1	0.13	0.34	0	1	11687	0.14	0.14	0.15
Bullied at school	0.06	0.25	0	1	0.08	0.28	0	1	11872	0.09	0.08	0.09
Age 7	0.04	0.21	0	1	0.05	0.23	0	1	14383	0.05	0.05	0.06
Age 11	0.03	0.17	0	1	0.04	0.20	0	1	13434	0.04	0.04	0.04
Domestic tension (age 7)	0.03	0.18	0	1	0.06	0.24	0	1	12630	0.07	0.06	0.07

Note: upward mobility is logically missing for those with origin class I/II, so too downward mobility for origin class IV/V.

Table S2 Robustness check a) - AMH variables use teacher ratings only: Upward mobility

		(a) Men and women								(b) Men only								(c) Women only							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	0.80	0.66	0.97	0.023		-0.050	-0.092	-0.007	0.76	0.59	0.99	0.043		-0.057	-0.113	-0.002	0.84	0.63	1.12	0.223		-0.038	-0.099	0.024
	Full	0.88	0.72	1.06	0.175		-0.029	-0.072	0.013	0.83	0.65	1.08	0.158		-0.039	-0.093	0.015	0.93	0.70	1.23	0.608		-0.016	-0.077	0.045
	Difference	0.91	0.87	0.95	0.000	41.3	-0.021			0.92	0.86	0.98	0.008	32.2	-0.018			0.90	0.84	0.97	0.005	58.5	-0.022		
Emotional symptoms	Reduced	0.82	0.75	0.90	0.000		-0.043	-0.063	-0.024	0.75	0.66	0.86	0.000		-0.060	-0.088	-0.032	0.88	0.77	1.01	0.061		-0.027	-0.056	0.001
	Full	0.86	0.78	0.93	0.001		-0.034	-0.054	-0.015	0.79	0.69	0.90	0.001		-0.051	-0.079	-0.023	0.92	0.80	1.05	0.189		-0.019	-0.047	0.009
	Difference	0.96	0.93	1.00	0.034	20.0	-0.009			0.96	0.92	1.01	0.112	14.0	-0.008			0.96	0.90	1.02	0.211	31.0	-0.008		
Hyperactivity	Reduced	0.70	0.57	0.85	0.001		-0.080	-0.125	-0.035	0.73	0.56	0.96	0.023		-0.066	-0.122	-0.010	0.65	0.47	0.89	0.007		-0.094	-0.161	-0.027
	Full	0.72	0.58	0.87	0.001		-0.074	-0.118	-0.030	0.75	0.58	0.97	0.029		-0.061	-0.116	-0.006	0.67	0.49	0.92	0.014		-0.085	-0.152	-0.018
	Difference	0.97	0.93	1.01	0.174	7.6	-0.006			0.98	0.93	1.03	0.458	6.7	-0.004			0.96	0.89	1.02	0.210	9.9	-0.009		

Sample size (number potentially upwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 13168-13340 (b) 6785-6888 (c) 6354-6454.
Not shown: concomitant variables (full & reduced models): female (a), origin class; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Table S3 Robustness check a) - AMH variables use teacher ratings only: Downward mobility

		(a) Men and women								(b) Men only								(c) Women only							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	1.21	0.99	1.48	0.057		0.038	-0.001	0.076	1.26	0.98	1.63	0.074		0.042	-0.004	0.089	1.15	0.86	1.55	0.342		0.030	-0.032	0.091
	Full	1.09	0.89	1.34	0.380		0.018	-0.022	0.057	1.14	0.88	1.48	0.304		0.024	-0.022	0.071	1.03	0.77	1.38	0.844		0.006	-0.055	0.067
	Difference	1.11	1.06	1.16	0.000	53.4	0.020			1.10	1.04	1.17	0.002	42.2	0.018			1.12	1.04	1.21	0.005	79.5	0.024		
Emotional symptoms	Reduced	1.24	1.13	1.35	0.000		0.041	0.024	0.059	1.36	1.20	1.55	0.000		0.056	0.034	0.078	1.14	1.00	1.30	0.050		0.027	0.000	0.055
	Full	1.19	1.10	1.30	0.000		0.035	0.018	0.052	1.32	1.17	1.49	0.000		0.050	0.029	0.072	1.10	0.97	1.26	0.146		0.020	-0.007	0.047
	Difference	1.03	1.00	1.08	0.086	16.1	0.007			1.03	0.98	1.08	0.220	10.1	0.006			1.04	0.97	1.11	0.291	26.9	0.007		
Hyperactivity	Reduced	1.43	1.16	1.76	0.001		0.070	0.029	0.110	1.35	1.03	1.77	0.031		0.054	0.006	0.102	1.56	1.09	2.23	0.015		0.092	0.019	0.165
	Full	1.40	1.14	1.73	0.002		0.066	0.025	0.107	1.33	1.02	1.75	0.038		0.052	0.003	0.100	1.52	1.08	2.15	0.018		0.087	0.015	0.158
	Difference	1.02	0.98	1.06	0.421	4.9	0.003			1.01	0.96	1.06	0.688	3.6	0.002			1.03	0.95	1.11	0.531	5.6	0.005		

Sample size (number potentially downwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 13325-13467 (b) 6838-6923 (c) 6471-6561.
Not shown: concomitant variables (full & reduced models): female (a), origin class; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Table S4 Robustness check b) - AMH variables are sum score of items: Upward mobility

		(a) Men and women								(b) Men only								(c) Women only							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	0.92	0.90	0.95	0.000		-0.017	-0.023	-0.012	0.92	0.90	0.95	0.000		-0.017	-0.023	-0.010	0.91	0.87	0.95	0.000		-0.020	-0.029	-0.010
	Full	0.95	0.93	0.98	0.000		-0.011	-0.016	-0.005	0.95	0.92	0.98	0.001		-0.011	-0.017	-0.004	0.95	0.91	0.99	0.011		-0.012	-0.021	-0.003
	Difference	0.97	0.96	0.98	0.000	38.5	-0.007			0.97	0.96	0.98	0.000	34.6	-0.006			0.96	0.95	0.98	0.000	40.2	-0.008		
Emotional symptoms	Reduced	0.96	0.93	0.99	0.008		-0.009	-0.016	-0.003	0.94	0.90	0.99	0.011		-0.013	-0.022	-0.003	0.97	0.93	1.01	0.152		-0.006	-0.015	0.002
	Full	0.97	0.94	1.00	0.026		-0.008	-0.015	-0.001	0.95	0.90	0.99	0.022		-0.012	-0.021	-0.002	0.98	0.94	1.02	0.309		-0.005	-0.013	0.004
	Difference	0.99	0.99	1.00	0.067	16.0	-0.001			1.00	0.98	1.01	0.365	8.3	-0.001			0.99	0.98	1.00	0.176	27.5	-0.002		
Hyperactivity	Reduced	0.87	0.83	0.90	0.000		-0.032	-0.040	-0.023	0.86	0.82	0.91	0.000		-0.031	-0.041	-0.021	0.87	0.82	0.93	0.000		-0.029	-0.042	-0.017
	Full	0.87	0.84	0.91	0.000		-0.030	-0.039	-0.021	0.87	0.83	0.91	0.000		-0.030	-0.040	-0.020	0.88	0.83	0.93	0.000		-0.027	-0.040	-0.015
	Difference	0.99	0.99	1.00	0.107	4.2	-0.001			1.00	0.99	1.01	0.387	2.8	-0.001			0.99	0.98	1.00	0.126	6.6	-0.002		

Sample size (number potentially upwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 13160-13325 (b) 6765-6892 (c) 6345-6451.
Not shown: concomitant variables (full & reduced models): female (a), origin class; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Table S5 Robustness check b) - AMH variables are sum score of items: Downward mobility

		(a) Men and women								(b) Men only								(c) Women only							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	1.07	1.05	1.10	0.000		0.014	0.009	0.018	1.06	1.03	1.10	0.000		0.011	0.005	0.017	1.09	1.04	1.13	0.000		0.017	0.008	0.025
	Full	1.04	1.02	1.06	0.000		0.008	0.003	0.012	1.04	1.00	1.07	0.026		0.006	0.001	0.012	1.05	1.01	1.09	0.024		0.009	0.001	0.017
	Difference	1.03	1.02	1.04	0.000	43.5	0.006			1.03	1.02	1.04	0.000	42.5	0.005			1.04	1.02	1.05	0.000	44.9	0.008		
Emotional symptoms	Reduced	1.06	1.03	1.09	0.000		0.011	0.005	0.016	1.07	1.03	1.12	0.002		0.013	0.005	0.021	1.04	0.99	1.09	0.108		0.008	-0.002	0.017
	Full	1.05	1.02	1.08	0.001		0.009	0.004	0.015	1.07	1.02	1.12	0.003		0.012	0.004	0.020	1.03	0.98	1.08	0.194		0.006	-0.003	0.016
	Difference	1.01	1.00	1.01	0.103	11.4	0.001			1.00	0.99	1.01	0.389	6.1	0.001			1.01	0.99	1.02	0.260	18.8	0.001		
Hyperactivity	Reduced	1.14	1.10	1.19	0.000		0.026	0.018	0.033	1.15	1.09	1.22	0.000		0.025	0.016	0.035	1.14	1.07	1.20	0.000		0.026	0.015	0.038
	Full	1.13	1.09	1.18	0.000		0.024	0.017	0.031	1.15	1.09	1.21	0.000		0.025	0.015	0.034	1.13	1.07	1.19	0.000		0.024	0.013	0.035
	Difference	1.01	1.00	1.01	0.057	5.4	0.001			1.00	1.00	1.01	0.271	3.4	0.001			1.01	1.00	1.02	0.172	7.1	0.002		

Sample size (number potentially downwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 13342-13488 (b) 6858-6949 (c) 6455-6569.
Not shown: concomitant variables (full & reduced models): female (a), origin class; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Table S6 Robustness check c) - AMH variables are binary, 90th percentile cutoff: Upward mobility

		(a) Men and women								(b) Men only								(c) Women only							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	0.52	0.43	0.63	0.000		-0.146	-0.188	-0.104	0.49	0.38	0.62	0.000		-0.155	-0.207	-0.103	0.54	0.39	0.73	0.000		-0.133	-0.200	-0.067
	Full	0.67	0.55	0.82	0.000		-0.088	-0.133	-0.043	0.63	0.49	0.81	0.000		-0.099	-0.152	-0.046	0.70	0.50	0.96	0.028		-0.078	-0.148	-0.009
	Difference	0.77	0.73	0.82	0.000	39.4	-0.057			0.77	0.70	0.84	0.000	36.4	-0.057			0.77	0.70	0.85	0.000	41.4	-0.055		
Emotional symptoms	Reduced	0.67	0.54	0.82	0.000		-0.090	-0.137	-0.044	0.65	0.49	0.86	0.003		-0.094	-0.155	-0.032	0.66	0.51	0.86	0.003		-0.088	-0.144	-0.033
	Full	0.70	0.57	0.87	0.002		-0.078	-0.126	-0.030	0.66	0.50	0.89	0.007		-0.088	-0.150	-0.025	0.72	0.55	0.94	0.016		-0.072	-0.129	-0.014
	Difference	0.95	0.90	1.00	0.032	13.6	-0.012			0.97	0.90	1.04	0.436	6.5	-0.006			0.92	0.85	1.00	0.053	19.1	-0.017		
Hyperactivity	Reduced	0.50	0.41	0.61	0.000		-0.154	-0.197	-0.112	0.53	0.40	0.71	0.000		-0.137	-0.199	-0.075	0.44	0.33	0.59	0.000		-0.176	-0.239	-0.113
	Full	0.53	0.43	0.64	0.000		-0.142	-0.185	-0.099	0.56	0.42	0.74	0.000		-0.126	-0.189	-0.064	0.48	0.36	0.64	0.000		-0.160	-0.222	-0.098
	Difference	0.95	0.90	0.99	0.022	8.1	-0.013			0.95	0.89	1.02	0.148	7.7	-0.011			0.93	0.86	1.01	0.075	9.0	-0.016		

Sample size (number potentially upwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 13169-13290 (b) 6807-6872 (c) 6335-6436.
Not shown: concomitant variables (full & reduced models): female (a), origin class; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Table S7 Robustness check c) - AMH variables are binary, 90th percentile cutoff: Downward mobility

		(a) Men and women								(b) Men only								(c) Women only							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	1.78	1.45	2.19	0.000		0.113	0.073	0.153	1.79	1.38	2.32	0.000		0.106	0.058	0.153	1.80	1.25	2.59	0.002		0.121	0.046	0.196
	Full	1.38	1.10	1.72	0.005		0.063	0.019	0.106	1.39	1.06	1.82	0.019		0.060	0.010	0.109	1.39	0.94	2.06	0.099		0.068	-0.013	0.149
	Difference	1.29	1.20	1.39	0.000	44.4	0.050			1.29	1.18	1.41	0.000	43.6	0.046			1.30	1.15	1.46	0.000	44.1	0.053		
Emotional symptoms	Reduced	1.60	1.31	1.95	0.000		0.091	0.053	0.130	1.66	1.30	2.12	0.000		0.092	0.048	0.136	1.54	1.18	2.02	0.002		0.089	0.035	0.143
	Full	1.50	1.23	1.83	0.000		0.079	0.040	0.118	1.59	1.23	2.05	0.000		0.084	0.038	0.130	1.42	1.09	1.86	0.011		0.072	0.018	0.127
	Difference	1.07	1.01	1.13	0.022	13.7	0.013			1.05	0.97	1.12	0.217	8.8	0.008			1.09	0.99	1.19	0.089	19.0	0.017		
Hyperactivity	Reduced	1.97	1.57	2.46	0.000		0.132	0.089	0.175	1.76	1.31	2.36	0.000		0.103	0.050	0.155	2.46	1.72	3.51	0.000		0.185	0.113	0.257
	Full	1.84	1.47	2.31	0.000		0.120	0.076	0.163	1.68	1.25	2.26	0.001		0.094	0.041	0.147	2.24	1.57	3.20	0.000		0.166	0.094	0.238
	Difference	1.07	1.01	1.13	0.026	9.4	0.012			1.05	0.98	1.13	0.169	8.6	0.009			1.10	1.00	1.21	0.061	10.3	0.019		

Sample size (number potentially downwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 13349-13514 (b) 6843-6948 (c) 6472-6572.
Not shown: concomitant variables (full & reduced models): female (a), origin class; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Table S8 Main results (Table 3, panel (a)) disaggregated by class of origin: Upward mobility

		(a) Origin class: III non-manual								(b) Origin class: III manual								(c) Origin class: IV/V							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	0.69	0.47	1.02	0.062		-0.079	-0.163	0.004	0.67	0.56	0.80	0.000		-0.085	-0.123	-0.048	0.83	0.64	1.07	0.150		-0.039	-0.092	0.015
	Full	0.78	0.53	1.14	0.192		-0.055	-0.137	0.028	0.79	0.66	0.94	0.009		-0.052	-0.090	-0.013	0.97	0.75	1.25	0.797		-0.007	-0.060	0.046
	Difference	0.89	0.82	0.98	0.014	31.3	-0.025			0.85	0.81	0.90	0.000	39.6	-0.034			0.86	0.81	0.91	0.000	82.4	-0.032		
Emotional symptoms	Reduced	0.94	0.77	1.15	0.538		-0.014	-0.058	0.030	0.92	0.83	1.02	0.117		-0.017	-0.039	0.004	0.90	0.78	1.05	0.178		-0.021	-0.051	0.010
	Full	0.94	0.77	1.16	0.568		-0.013	-0.057	0.032	0.94	0.84	1.04	0.224		-0.014	-0.036	0.009	0.92	0.79	1.07	0.255		-0.018	-0.049	0.013
	Difference	1.00	0.92	1.07	0.905	7.2	-0.001			0.98	0.95	1.02	0.387	20.3	-0.003			0.99	0.94	1.03	0.560	13.0	-0.003		
Hyperactivity	Reduced	0.74	0.50	1.08	0.118		-0.066	-0.150	0.017	0.68	0.56	0.82	0.000		-0.083	-0.124	-0.042	0.68	0.51	0.91	0.011		-0.079	-0.139	-0.020
	Full	0.73	0.50	1.06	0.095		-0.069	-0.149	0.012	0.66	0.54	0.81	0.000		-0.088	-0.130	-0.046	0.67	0.50	0.90	0.008		-0.083	-0.142	-0.023
	Difference	1.01	0.94	1.09	0.790	-3.4	0.002			1.02	0.98	1.07	0.238	-6.2	0.005			1.02	0.97	1.07	0.468	-4.5	0.004		

Sample size (number potentially upwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 1867-1958 (b) 7105-7328 (c) 4039-4165.
Not shown: concomitant variable (full & reduced models): female; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Table S9 Main results (Table 3, panel (b)) disaggregated by class of origin: Downward mobility

		(a) Origin class: I/II								(b) Origin class: III non-manual								(c) Origin class: III manual							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	1.39	1.10	1.75	0.006		0.075	0.023	0.127	1.37	0.94	1.98	0.100		0.060	-0.012	0.132	1.22	1.00	1.48	0.054		0.033	0.000	0.067
	Full	1.23	0.98	1.56	0.077		0.048	-0.005	0.100	1.20	0.84	1.73	0.311		0.036	-0.034	0.106	1.04	0.84	1.29	0.698		0.007	-0.029	0.043
	Difference	1.13	1.06	1.20	0.000	36.3	0.027			1.13	1.03	1.24	0.008	40.4	0.024			1.17	1.11	1.23	0.000	78.6	0.026		
Emotional symptoms	Reduced	1.10	0.97	1.25	0.143		0.022	-0.007	0.051	1.07	0.89	1.30	0.467		0.014	-0.024	0.051	1.12	1.00	1.26	0.053		0.019	0.000	0.039
	Full	1.09	0.96	1.23	0.201		0.019	-0.010	0.047	1.09	0.89	1.33	0.417		0.016	-0.023	0.056	1.10	0.98	1.24	0.101		0.016	-0.003	0.036
	Difference	1.01	0.96	1.07	0.587	14.5	0.003			0.99	0.91	1.07	0.760	-17.8	-0.002			1.02	0.98	1.05	0.379	14.0	0.003		
Hyperactivity	Reduced	1.32	1.01	1.72	0.045		0.062	0.001	0.123	1.57	1.08	2.28	0.019		0.087	0.016	0.158	1.45	1.19	1.77	0.000		0.063	0.030	0.097
	Full	1.32	1.01	1.72	0.046		0.062	0.001	0.123	1.58	1.10	2.27	0.015		0.088	0.019	0.157	1.49	1.21	1.83	0.000		0.068	0.033	0.102
	Difference	1.00	0.95	1.05	0.980	0.2	0.000			1.00	0.92	1.08	0.908	-1.1	-0.001			0.98	0.94	1.01	0.207	-6.7	-0.004		

Sample size (number potentially downwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 4207-4324 (b) 1867-1958 (c) 7105-7328.
Not shown: concomitant variable (full & reduced models): female; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Table S10 Complete-cases analysis: Upward mobility

		(a) Men and women								(b) Men only								(c) Women only							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	0.83	0.66	1.04	0.100		-0.040	-0.088	0.008	0.95	0.70	1.29	0.748		-0.011	-0.075	0.054	0.64	0.45	0.91	0.013		-0.089	-0.160	-0.019
	Full	0.96	0.76	1.21	0.743		-0.008	-0.057	0.041	1.11	0.81	1.51	0.526		0.021	-0.044	0.086	0.76	0.53	1.09	0.133		-0.056	-0.128	0.017
	Difference	0.86	0.80	0.93	0.000	79.7	-0.032			0.86	0.77	0.96	0.006	301.2	-0.032			0.85	0.75	0.96	0.009	37.9	-0.034		
Emotional symptoms	Reduced	0.92	0.81	1.05	0.239		-0.017	-0.045	0.011	0.82	0.67	1.00	0.045		-0.042	-0.083	-0.001	0.97	0.80	1.16	0.710		-0.007	-0.045	0.031
	Full	0.91	0.80	1.05	0.189		-0.019	-0.047	0.009	0.79	0.65	0.96	0.020		-0.050	-0.091	-0.008	0.98	0.81	1.18	0.794		-0.005	-0.043	0.033
	Difference	1.01	0.95	1.08	0.767	-12.4	0.002			1.04	0.94	1.14	0.445	-18.3	0.008			0.99	0.89	1.10	0.845	29.2	-0.002		
Hyperactivity	Reduced	0.56	0.44	0.71	0.000		-0.124	-0.173	-0.075	0.53	0.39	0.73	0.000		-0.133	-0.197	-0.068	0.64	0.44	0.93	0.021		-0.089	-0.163	-0.014
	Full	0.53	0.42	0.67	0.000		-0.134	-0.183	-0.085	0.51	0.37	0.70	0.000		-0.142	-0.207	-0.077	0.61	0.42	0.88	0.009		-0.101	-0.176	-0.026
	Difference	1.05	0.98	1.12	0.169	-8.0	0.010			1.04	0.95	1.15	0.403	-6.5	0.009			1.06	0.95	1.19	0.282	-13.7	0.012		

N: (a) 2352 (b) 1176 (c) 1176.

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

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

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

Table S11 Complete-cases analysis: Downward mobility

		(a) Men and women								(b) Men only								(c) Women only							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	1.43	1.13	1.80	0.003		0.065	0.022	0.108	1.29	0.93	1.79	0.130		0.042	-0.012	0.097	1.63	1.15	2.30	0.006		0.094	0.028	0.160
	Full	1.17	0.92	1.48	0.205		0.028	-0.015	0.072	1.08	0.78	1.51	0.641		0.013	-0.042	0.069	1.28	0.90	1.82	0.167		0.048	-0.020	0.116
	Difference	1.22	1.12	1.33	0.000	56.7	0.037			1.19	1.07	1.33	0.002	68.8	0.029			1.27	1.11	1.46	0.001	49.2	0.046		
Emotional symptoms	Reduced	1.14	1.00	1.30	0.050		0.024	0.000	0.048	1.28	1.05	1.56	0.016		0.041	0.008	0.074	1.04	0.87	1.24	0.682		0.007	-0.028	0.042
	Full	1.15	1.01	1.31	0.041		0.026	0.001	0.050	1.30	1.06	1.59	0.011		0.044	0.010	0.078	1.03	0.86	1.24	0.713		0.007	-0.029	0.042
	Difference	0.99	0.92	1.07	0.839	-5.7	-0.001			0.98	0.89	1.08	0.707	-7.6	-0.003			1.00	0.89	1.13	0.956	9.0	0.001		
Hyperactivity	Reduced	1.42	1.12	1.80	0.004		0.064	0.021	0.108	1.59	1.15	2.20	0.005		0.078	0.024	0.131	1.26	0.87	1.81	0.216		0.045	-0.026	0.115
	Full	1.52	1.19	1.93	0.001		0.076	0.033	0.120	1.66	1.20	2.30	0.002		0.085	0.031	0.139	1.40	0.97	2.02	0.074		0.065	-0.006	0.136
	Difference	0.94	0.87	1.01	0.074	-18.9	-0.012			0.96	0.87	1.05	0.361	-9.6	-0.007			0.90	0.80	1.02	0.089	-45.4	-0.020		

N: (a) 2719 (b) 1340 (c) 1379.

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

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

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

Table S12 Main results disaggregated by sex: Upward mobility

		(a) Men and women								(b) Men only								(c) Women only							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	0.74	0.65	0.85	0.000		-0.066	-0.096	-0.036	0.73	0.62	0.85	0.000		-0.066	-0.100	-0.033	0.72	0.58	0.90	0.005		-0.070	-0.117	-0.023
	Full	0.85	0.74	0.98	0.028		-0.035	-0.066	-0.004	0.84	0.71	0.99	0.038		-0.037	-0.071	-0.002	0.84	0.67	1.05	0.128		-0.037	-0.086	0.011
	Difference	0.87	0.84	0.90	0.000	47.2	-0.031			0.87	0.83	0.91	0.000	44.8	-0.030			0.86	0.82	0.90	0.000	46.4	-0.032		
Emotional symptoms	Reduced	0.93	0.87	1.00	0.047		-0.016	-0.032	0.000	0.87	0.78	0.98	0.017		-0.028	-0.052	-0.005	0.97	0.87	1.08	0.547		-0.007	-0.030	0.016
	Full	0.94	0.87	1.01	0.094		-0.014	-0.030	0.002	0.89	0.79	0.99	0.030		-0.026	-0.049	-0.003	0.97	0.87	1.09	0.644		-0.005	-0.029	0.018
	Difference	0.99	0.97	1.01	0.388	14.7	-0.002			0.99	0.95	1.02	0.438	9.8	-0.003			0.99	0.95	1.03	0.709	22.8	-0.002		
Hyperactivity	Reduced	0.68	0.59	0.79	0.000		-0.085	-0.117	-0.053	0.69	0.57	0.83	0.000		-0.079	-0.117	-0.041	0.68	0.54	0.87	0.002		-0.081	-0.131	-0.031
	Full	0.67	0.57	0.78	0.000		-0.089	-0.121	-0.056	0.67	0.56	0.81	0.000		-0.084	-0.123	-0.045	0.67	0.53	0.86	0.002		-0.084	-0.135	-0.034
	Difference	1.02	0.99	1.05	0.129	-5.0	0.004			1.02	0.99	1.06	0.185	-6.1	0.005			1.02	0.97	1.06	0.493	-4.1	0.003		

Sample size (number potentially upwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 13184-13301 (b) 6797-6893 (c) 6357-6454.
Not shown: concomitant variables (full & reduced models): female (a), origin class; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Table S13 Main results disaggregated by sex: Downward mobility

		(a) Men and women								(b) Men only								(c) Women only							
		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI		OR	95% CI		*p*	Δ (%)	APE	95% CI	
Conduct problems	Reduced	1.30	1.14	1.49	0.000		0.051	0.025	0.077	1.30	1.08	1.56	0.005		0.047	0.014	0.079	1.30	1.06	1.58	0.010		0.053	0.013	0.093
	Full	1.13	0.98	1.30	0.081		0.024	-0.003	0.051	1.13	0.94	1.37	0.196		0.022	-0.012	0.057	1.12	0.91	1.36	0.283		0.022	-0.019	0.063
	Difference	1.15	1.11	1.19	0.000	53.4	0.027			1.15	1.09	1.20	0.000	52.2	0.024			1.16	1.10	1.23	0.000	57.9	0.031		
Emotional symptoms	Reduced	1.11	1.02	1.20	0.014		0.020	0.004	0.036	1.16	1.03	1.30	0.012		0.027	0.006	0.048	1.06	0.95	1.18	0.270		0.012	-0.010	0.034
	Full	1.10	1.01	1.19	0.028		0.018	0.002	0.034	1.15	1.02	1.29	0.023		0.025	0.004	0.046	1.05	0.95	1.17	0.338		0.011	-0.011	0.033
	Difference	1.01	0.98	1.04	0.454	9.7	0.002			1.01	0.98	1.05	0.479	8.2	0.002			1.01	0.96	1.05	0.749	11.7	0.001		
Hyperactivity	Reduced	1.39	1.20	1.62	0.000		0.064	0.036	0.093	1.41	1.13	1.75	0.003		0.061	0.022	0.100	1.42	1.14	1.77	0.002		0.072	0.027	0.117
	Full	1.42	1.22	1.65	0.000		0.068	0.039	0.097	1.44	1.16	1.79	0.002		0.065	0.026	0.104	1.44	1.15	1.80	0.002		0.075	0.029	0.121
	Difference	0.98	0.96	1.01	0.204	-5.3	-0.003			0.98	0.94	1.01	0.185	-6.8	-0.004			0.99	0.94	1.03	0.572	-3.8	-0.003		

Sample size (number potentially downwardly mobile) varies due to multiple imputation. Minimum and maximum N: (a) 13343-13469 (b) 6865-6936 (c) 6475-6558.
Not shown: concomitant variables (full & reduced models): female (a), origin class; Z variables (full models only): childhood socioeconomic deprivation, childhood adversities.
APE = average partial effect. 'Difference' OR is the ratio of the ORs in the reduced and full models.
Δ (%) is the percentage reduction in the logit coefficient between the reduced and full model attributable to confounding, net of rescaling (Karlson et al. 2012: 294, equation 12c).

Table S14 Attenuation of conduct problems coefficients decomposed into portions attributable to the various childhood environment controls

	UP				DOWN			
	P_Diff		P_Reduced		P_Diff		P_Reduced	
z variable	Mean	SD	Mean	SD	Mean	SD	Mean	SD
CSD-crowding	17.27	3.16	7.98	2.16	15.81	2.19	8.38	2.19
CSD-low income	31.58	4.10	14.64	3.90	24.77	4.12	12.97	2.96
CSD-unemp.	1.21	2.60	0.60	1.36	1.00	2.57	0.54	1.50
CSD-low father SC	12.17	2.42	5.66	1.88	13.11	2.85	6.95	2.20
CSD-rented housing	24.14	2.23	11.14	2.46	28.68	2.74	15.10	3.23
CSD-birth context	1.04	1.42	0.51	0.67	1.11	1.29	0.54	0.70
Taken into care	0.27	1.82	0.19	0.92	1.10	2.10	0.57	1.10
Sep. from bio. M	1.43	1.10	0.58	0.50	2.46	1.32	1.29	0.66
Sep. from bio. F	-2.79	1.61	-1.27	0.77	-0.21	1.79	-0.07	0.95
Family criminality	6.17	2.89	2.81	1.32	5.61	2.56	2.87	1.24
Neglect	11.90	1.65	5.47	1.20	8.25	1.72	4.33	1.15
Tension	-5.73	2.76	-2.57	1.18	-3.16	2.96	-1.62	1.50
Bullied	1.36	0.79	0.60	0.31	1.47	0.72	0.78	0.43

Note: P_Diff: percentage of the total reduction of the AMH coefficient contributed by the risk factor variable (sums to 100). P_Reduced: percentage the risk factor variable reduces the AMH coefficient by. UP: upward mobility analysis. DOWN: downward mobility analysis. The CSD variables (see **Childhood socioeconomic deprivation indicators** above) are grouped into: CSD-crowding (measures of crowding & whether child shares a bedroom), CSD-low income (measures of financial problems & receipt of free school meals), CSD-unemp. (unemployment), CSD-low father SC (low social class of father), CSD-rented housing (non-owner-occupier housing tenure), and CSD-birth context (whether mother had the child as a teenager, whether parents were not in a stable relationship at the child's birth). Each CSD measure is the simple sum of the included binary indicators.

4 The *when* and the *how* of the emergence of social inequality in mental health: social causation and health selection through employment transitions

4.1 Introduction

The association between income (and other indicators of socioeconomic status) and mental health problems is a topic of enduring importance for social scientists and public health researchers. In this chapter I draw on several strands of the health inequalities literature to attempt to answer the following question: when and how does the association between income and mental health problems arise? The mental health problems examined here are symptoms of depression and anxiety. In past research, hypotheses relating to the *how* – social causation, health selection, or indirect health selection – have received considerably more attention than the *when* (Kröger et al., 2015). This chapter aims to attend to both elements of the question, hypothesising that different mechanisms for the generation of mental health inequality may take precedence at different life course stages.

In attending to the *how*, much health inequalities research has tested whether mechanisms of social causation and health selection *are evident* in a given sample. Blane et al. (1999) and Chandola et al. (2003) are among those to have drawn the distinction between such tests and – further – examining the contribution of these mechanisms to explaining gradients in health. That is, social position may affect subsequent health, but this in itself does not imply anything about the importance of social causation in the generation of health inequalities.

The two studies cited above apply this insight with respect to health inequality between social classes and employment grades respectively, but exclude the non-employed. Attending only to the continuously employed may however miss a substantial part of the true extent of health inequality across the adult population (Dahl, 1993; van de Mheen et al., 1999). Indeed some evidence indicates that health (both physical and mental) is related more strongly to transitions in and out of employment than to moves between occupational or class categories (Elstad, 2004; Ki et al., 2011; Richards & Paskov, 2016).

Methodologically advanced recent work has reported negative health effects of unemployment and job loss (Krug & Eberl, 2018; Steele et al., 2013), adding support to the social causation hypothesis. However this work in turn does not examine the contribution of such employment transitions to explaining social gradients in health. Addressing this limitation may be particularly important given that in the UK context, '[b]etween 1973 and 2009, the relation between good health and securing and sustaining employment has strengthened for both men and women' (Minton et al., 2012: 1).

It is not straightforward to incorporate the non-employed in a model of the generation of health inequality, since the measure of socioeconomic status used is often based on an individual's current employment, and simply ranking the unemployed or non-employed as the lowest grade or social class has notable limitations. In this chapter I include employment transitions in a model of mental health inequality in which the extent of that inequality is measured by the contemporaneous association between household income and mental health. Existing research suggests that mental health problems may be more robustly related to income than social class (Fryers et al., 2003; Richards & Paskov, 2016).

Associations between mental health, SES, and transitions in and out of employment are likely to vary across the life course, making certain life stages and turning points more plausible than others as sites for the generation of mental health inequalities. In particular, I make the case that the transition to adulthood is a period during which the association between mental health and SES is especially likely to strengthen due to both health selection and social causation, while the latter may also play an important role in mid-adulthood. I conceptualise the transition to adulthood as spanning exit from compulsory education to economic independence from one's parents. Life course scholars argue that this transition has expanded from being concentrated in the early 20s, to spanning the ages from 18 to the early- or even mid-30s (Cook & Furstenberg, 2002; Furstenberg et al., 2004; Swartz et al., 2011).

Consideration of the three hypotheses generally applied to the explanation of health inequalities should lead scholars to view the *when* as inextricably linked to the *how*. One potential answer to the *how* is the indirect health selection hypothesis – the idea that the health-SES relation is confounded rather than arising from the causal effects of one upon

the other. This hypothesis has drawn support, as Hoffmann et al. (2018: 30) observe, from 'several studies that begin observing the relationship between SES and health in mid-life, find little or no mutual effects, and conclude that health inequalities can be explained by indirect selection.' Such studies are agnostic as to whether finding no evidence of social causation or health selection within a given age range means that omitted (and perhaps time-invariant) variables determine health and SES through the life course, or – a very different conclusion – whether in fact social causation and health selection *do* generate this inequality, but do so at earlier ages, prior to the observation window, beyond which it merely persists as cohorts age.

Attention to timing, then, is crucial both because different causal mechanisms may take priority at different ages, and because ignoring timing can lead to confusion around the interpretation of tests of the different mechanisms. Policy implications follow from each of these points. Measures to counteract or ameliorate the impact of SES on health or vice versa will be mistargeted if focused on age groups in which those mechanisms do not in fact operate. Moreover, perceptions of the fairness of health inequalities could be coloured by studies which attend only to restricted age ranges, find no evidence of either causal mechanism, and conclude that the SES-health relationship is confounded by fixed traits such as personality or cognitive ability (Gottfredson, 2004; Link et al., 2008), or by adolescent lifestyle (Glendinning et al., 1995).

This chapter uses eight waves of nationally representative panel data from the UK Household Longitudinal Study. First, I describe cross-sectional inequality in mental health by age, revealing at which stages of the life course inequalities are more or less apparent, and over which periods they increase, remain stable, and decline. Adding economic status to this static model accounts for a large part of the contemporaneous income-mental health association and motivates a focus on the role of employment transitions. In the next step I present a series of longitudinal structural equation models. These models test for the presence of social causation, health selection, and indirect health selection; and they measure the extent to which these mechanisms account for observed increases in mental health inequality. Finally, results are compared for different age groups, to examine the timing of the different mechanisms generating mental health inequalities. Before coming to the empirical part of the chapter, I further discuss relevant literature to contextualise what follows.

4.2 Background and hypotheses

Hypotheses regarding the question of *how* social inequalities in mental health arise are the familiar social causation (SES affects mental health), health selection (mental health affects SES), and indirect health selection (confounding; no causal effects between the two). A focus of this chapter is the *when*: if social causation and health selection occur, does the strength of each of these effects – and their contribution to generating health inequality – vary by age, and if so how? I consider first some evidence on when the social gradient in mental health forms; this work directs our attention to the period of transition to adulthood and the processes that occur therein.

4.2.1 Generation of the mental health gradient

One particularly relevant cluster of evidence describes the (re-)emergence of inequalities in mental health from late adolescence to early adulthood. Both cross-sectional (Vallejo-Torres et al., 2014) and longitudinal (Sweeting et al., 2016) evidence from the UK indicate an absence of mental health inequality in late adolescence (ages 16-19 for Vallejo-Torres et al., and age 18 for Sweeting et al.). In each of these studies, mental health inequality has emerged by the next age group or measurement point respectively (ages 20-24 and age 24). Similarly, Wickrama et al. (2009), using US data, find equalisation of depressive symptoms according to parental education from early (ages 13/14) to late (19/20) adolescence, but re-emergence of inequalities by age 23.

If the research mentioned above is indicative of a lower bound for the life course stage at which the social gradient in mental health is generated, a second relevant body of work suggests an upper bound. Though highly informative contributions, these however effectively ignore the transition to adulthood as a site for the generation of (mental) health inequalities. Foverskov and Holm (2016) aim to test the hypotheses of social causation, health selection, and indirect selection using the British Household Panel Survey. They restrict their analyses to men and women aged between 30 and 60, and model health as a single latent factor including a measure of psychological wellbeing. In a sensitivity analysis the GHQ-12 measure of depressive symptoms is used as the measure of health,

with the same results. While cross-sectional inequalities are evident, they find no evidence of prior health influencing later SES or vice versa, and so conclude that '[t]he well-known cross-sectional correlations between health and SEP in adulthood seem not to be driven by a causal relationship, but instead by dynamics and influences in place before the respondents turn 30 years old that affect both their health and SEP onwards' (Foverskov & Holm, 2016: 180-181). Richards and Paskov (2016) do not explicitly test these causal hypotheses, but rather explore mechanisms which might account for the social class gradient in mental health, limiting their analyses to those aged 25-65. Their fixed-effects regressions show that changes in social class are unrelated to changes in GHQ score. They therefore reach a similar conclusion, suggesting the importance of unobserved heterogeneity due to factors acting earlier in life which influence both the psychological wellbeing and social class of individuals in adulthood. These studies both, then, point towards younger ages for the actual generation of mental health inequalities.

These two strands of literature taken together suggest that social causation and/or health selection occur across the transition to adulthood, generating mental health inequalities which persist thereafter. However, there is further complexity to consider. In the case of Foverskov and Holm (2016), the authors test for effects at various lag lengths of household income on mental health and vice versa. Economic status is not included in the model.[3] This appears to reflect a tacit assumption that economic status only affects mental health through income. On the contrary, a large literature attests psychosocial and social pathways too: the loss of purpose, social networks, status, time structure, and sense of control (Brand, 2015; Krug & Eberl, 2018; Newman, 1988). A more plausible causal model includes economic status as a direct and synchronous determinant of both income and mental health; indeed this is a central assumption of the current chapter.

In the case of Richards and Paskov (2016), economic status also looms large. A second major finding from this paper is that, conditional on economic status, there is no social class gradient in mental health. The implication is that lower social class is associated with poorer mental health precisely because it entails a greater risk of unemployment.

[3] Foverskov and Holm (2016) use the Cambridge Social Interaction and Stratification Scale as an alternative measure of SES, but not, as they indeed admit, in a manner that takes into account employment transitions: non-employed individuals are given a value based on their 'last know [sic] position' (2016: 181).

This is wholly in line with the theoretical underpinnings of the NS-SEC measure used, under which lower social class position is defined by the greater instability of the type of employment contract that defines it (Erikson & Goldthorpe, 1992).

The finding of no social causation and no health selection among individuals in mid-adulthood thus neglects the possibility that those processes do occur within this age range, and do so through employment transitions: job loss may represent social causation if it leads to worse mental health, and health selection if it is precipitated by poor mental health.

4.2.2 Health selection and social causation over the life course

Clark and Oswald (1994) report a stronger association between unemployment and depressive symptoms among those aged 30-49 than older or younger. In general however, previous work on the links between labour market status and mental health appears to have paid little attention to variation by age, tending either to aggregate the whole working-age population or examine specific age groups. At what life course stages are employment transitions – and other pathways of social causation and health selection – likely to have the greatest impact? Considerations deriving from life course theory highlight the transition to adulthood as a period when health selection is especially likely to play out.

Past research views the transition to adulthood as comprised of a set of discrete events, such as leaving formal education, commencing employment, leaving the parental household, first union, and birth of first child (Billari, 2001; Buchmann & Kriesi, 2011). This chapter conceptualises the transition to adulthood primarily as a period during which an individual's SES shifts from that of their parents, to their own. This shift may be viewed as discrete, perhaps coinciding with leaving the parental household. However, and as I argue further below, it is better viewed as continuous, with parents tending to provide financial and other forms of material support through the transition from dependence to independence.

This shift from parental to own SES entails the *formation* of an individual's own SES. The distal causes of success here – for instance genetic endowment, socialisation,

parental time investments, and educational resources – may extend back throughout the earlier stages of life. However the proximal causes of one's early own SES, such as educational and early-career decision-making, performance in school and university examinations, and job applications, are contingent events occurring around the ages 16-25. By contingent I mean to emphasise that although individuals may be endowed with or gradually develop traits and abilities which predispose them to a high early own SES, nevertheless the translation of this latent predisposition into actual, realised SES depends on events and processes which are vulnerable to disruption by negative shocks to mental health. One's nascent own SES could for instance be impacted by an episode of poor mental health coinciding with examinations, or disrupting the job search process. The cognitive characteristics of common mental health problems may influence decision-making relevant to SES formation. Relatively pessimistic thinking among the depressed is one such characteristic (Alloy et al., 1990; Wisco, 2009), implying that individuals would make less risky – or ambitious – educational and career decisions.

There is ample evidence for health selection at this life course stage. US research indicates that adolescent depressive symptoms may disrupt the status attainment process (Kessler et al., 1995; McLeod & Fettes, 2007), for instance through an association with risky behaviours such as committing crimes and excessive drinking (Wickrama & Wickrama, 2010). Studies of the 1979 US National Longitudinal Survey of Youth report associations between higher initial levels of depressive symptoms (at ages 27-35) and higher odds of both remaining (Prause & Dooley, 2001) and becoming (Dooley et al., 2000) unemployed two years later. Samples from the lower end of the transition to adulthood age range report similar conclusions: Swedish 16-year-olds with above-average levels of depressive symptoms were at greater risk both of any and of long-term unemployment over the following five years (Hammarström & Janlert, 1997); low self-esteem among high school students in the US was associated with spending a greater proportion of the following seven years unemployed (Dooley & Prause, 1997). The most robust evidence for mental health influencing employment status among young adults is the sibling fixed-effects models reported by Egan et al. (2016): individuals with depressive symptom scores at least one standard deviation above the mean at age 16-20 were substantially more likely to be unemployed or out of the labour force 11 years later, and experienced substantially more time unemployed over the follow-up period.

Due then to its contingent and provisional nature, SES in the transition to adulthood may be particularly vulnerable to health selection effects. At later stages of working-age life, episodes of poor mental health may adversely affect SES, but individuals are more likely to have accrued resources with which to buffer this impact, such as financial savings, the support of a long-term partner, or tenure and firm-specific knowledge as barriers to job loss.

Social causation may also be especially strong during the transition to adulthood. This life course stage generally involves a range of stressful role transitions, discontinuities, and new responsibilities, as well as the competitive pressures involved in entering the labour market and finding a long-term partner. Early employment careers are marked by volatility and uncertainty. Such stressors will result in social causation to the extent that their prevalence and impact are unevenly distributed by SES. Reviewing intergenerational transfers during the transition to adulthood, Waithaka notes that '[b]eyond assistance with the big ticket items, family wealth through *inter vivo* [sic] transfers also plays an instrumental role in smoothening the everyday experiences of the young adult transition process' (2014: 480). Various dimensions of parental SES are positively associated with financial assistance to young adult children (Hardie & Seltzer, 2016; Schoeni & Ross, 2005), in both the US and the UK (Henretta et al., 2002). Parents also provide other forms of assistance such as social connections and advice on finances and the labour market, in a similarly stratified manner (Waithaka, 2014).

On the other hand, social causation – and particularly the impact of job loss and unemployment – may be particularly strong through mid-adulthood. This is a period of increasing responsibility, not only in the workplace and for children, but also for elderly parents (Aneshensel et al., 1995; Lachman et al., 2015). While job loss may be less likely in this period, it may have a greater impact on mental health for being relatively unexpected and arriving when many have reached their peak in seniority and earnings (Lachman, 2004). Moreover, the accumulation through adulthood of social and financial resources mentioned earlier is socially stratified; individuals living in economic precariousness will be especially vulnerable to the effects of job loss.

Finally, in contrast to the above, the large literature documenting the importance of childhood factors for mental health in adulthood (see Chapter 2) suggests that causal

relations between mental health and SES might not be observed in the transition to adulthood, nor at any point in working-age life. Rather, this would suggest that a conclusion similar to that of Foverskov and Holm (2016) and Richards and Paskov (2016) will be found, supporting the indirect health selection hypothesis and highlighting the importance of even earlier ages in determining individuals' SES and mental health trajectories through the life course.

4.2.3 Unobserved heterogeneity

I consider one final topic before presenting the empirical part of the chapter: unobserved heterogeneity and its relevance for the social causation and health selection hypotheses.

Recent research has increasingly used cross-lagged panel models to test these two hypotheses. Foverskov and Holm's (2016) study is one example, in which the authors apply the Anderson-Hsiao fixed-effects estimator (Anderson & Hsiao, 1982). As these authors note, in contrast to their own analysis the majority of similar previous studies harness both between- and within-individual variation rather than excising unobserved heterogeneity by focusing on within variation (Aittomäki et al., 2012; Chandola et al., 2003; Elovainio et al., 2011; Mulatu & Schooler, 2002; Steele et al., 2013; Warren, 2009). Moreover, these studies all find support for social causation, and nonsignificant or relatively weak health selection effects. For instance, Chandola et al. (2003) report consistent associations of prior employment grade with subsequent mental health in the Whitehall II study. They only find the opposite when modelling SES as financial deprivation, and that only for men, with effects only 25-40% the size of the equivalent social causation estimates. Warren (2009) explicitly addresses the threat of unobserved heterogeneity by including measures of child health and SES, but these observed measures are unlikely to capture the full range of possibly relevant time-invariant individual factors which might jointly influence SES and health across the life course. Steele et al. (2013) explore the (social causation) effects of employment transitions through simultaneous equations for the set of transitions and GHQ. The authors adjust for selection through terms linking prior GHQ both with GHQ at t and with the $t–1$ to t employment transitions; they also allow nonzero correlations between individual-level random effects for each outcome, thus allowing 'for the possibility that changes in mental health and employment transitions are jointly determined' by time-invariant

unobservables (2013: 706). This is a relatively strong design but nevertheless requires that the random effects assumption be made with respect to the other controls in the model: age, marital status, care responsibilities, and occupation class.

The one analysis taking a similar approach to Foverskov and Holm (2016) and utilising only within-individual variation in a cross-lagged model, and which like Chandola et al. (2003) uses the Whitehall II study, in fact finds support for health selection and not for social causation (Case & Paxson, 2011). The lag structure proposed in Case and Paxson's model is highly questionable, implying that a health shock between measurement points 1 and 2 causes a decline in SES *between* measurement points 3 and 4. It is hard to see why negative effects might not show up at measurement point 2, in which case they are missed by this model, or at measurement point 3, in which case they lower the baseline from which deviations at point 4 are measured. Moreover, the Whitehall II study represents a selected group, as in fact Case and Paxson (2011) demonstrate by comparison with the nationally representative NCDS and BCS. Nevertheless, it is suggestive that conclusions differ depending on whether unobserved heterogeneity is accounted for.

Foverskov and Holm (2016) thus both identify and add to an important discrepancy in the literature. The current evidence, despite its limitations, suggests a major role for unobserved heterogeneity in biasing tests of the health selection and social causation hypotheses. One of the contributions of this chapter is to strengthen the evidence with a method robust to unobserved heterogeneity – by including individual fixed effects.

4.3 Data

This chapter uses eight waves of the nationally representative UK Household Longitudinal Study (UKHLS), also known as Understanding Society. The first wave was collected between 2009 and 2011, and the eighth between 2016 and 2018, with individuals surveyed at approximately annual intervals. From wave 2 onwards, Understanding Society has absorbed the British Household Panel Survey (BHPS), yielding a total combined sample of 39,942 households. Of these, 30,428 (76.2%)

responded partially or fully at wave 2, with 50,389 (59.4%) of 84,891 adults (those aged 16 or older) giving a full interview.

Understanding Society has a complex survey design, which is accounted for by including design weights and using the *svy* suite of commands in Stata 14.

4.3.1 Missing data strategy

Understanding Society provides a range of weights to account for sampling design as well as non-response and attrition. The latter is accomplished through modelling based on a wide range of information collected at various levels (including region, neighbourhood, household, and individual) and from various sources including, if applicable, previous waves of the survey. Extensive detail is provided in the Understanding Society *User Guide* (Knies, 2018).

For the cross-sectional description of mental health inequality by age and sex, I pool observations from all waves, using the cross-sectional individual-level self-completion weight. These results can be thought of as pooling eight samples which are each representative of the UK at the time that that wave's data was collected. 'Self-completion' refers to the fact that these weights adjust for the differential probability of individuals to complete the self-completion portion of the individual interview, which contains the questionnaire modules on mental health.

In the longitudinal structural equation models, I combine the use of longitudinal individual-level self-completion weights with full-information maximum likelihood estimation (FIML). This is in order to strike a balance between robust adjustment for selective attrition on the one hand, and maximising use of the available data on the other. The longitudinal weights provided by Understanding Society are constructed such that an individual who does not respond at a given wave receives a zero weight for that and all subsequent waves. Using the longitudinal weights from wave 8 thus drastically reduces sample size, discarding information even from individuals who have only missed one wave (except insofar as they inform the weighting of the minority of individuals who are successfully interviewed at all waves). FIML utilises all available data, including from non-complete cases, but does so under a missing-at-random assumption. I therefore

use the longitudinal weights from wave 3 and thus include all individuals who respond in waves 1-3 (or 2-3 in the case of the BHPS sample), even those with missing data from later waves. These weights adjust for survey design and initial non-response, and for predictors of attrition over three waves. I therefore make the assumption that the predictors of attrition across three waves are similar to the predictors of attrition across eight waves. The longitudinal sample is representative of the UK population at wave 2, since this is the wave at which this combined sample (UKHLS and BHPS) enters the study.

4.4 Measures

4.4.1 Mental health problems

The General Health Questionnaire 12-item version (GHQ or GHQ-12) is used to measure mental health. The GHQ is a widely-used measure of the symptoms of common mental health problems including depression, anxiety, and associated somatic complaints (Goldberg et al., 1997; Jackson, 2007). Each of the 12 items is scored from 0 to 3; for example: 'Have you recently lost much sleep over worry?' (0 = *not at all*, 1 = *no more than usual*, 2 = *rather more than usual*, 3 = *much more than usual*). This 'Likert' scoring method gives a total score ranging from 0 to 36, which I then standardise (within sex and wave). Items are listed in full in the Appendix to this chapter.

4.4.2 Household income

The raw variable taken from the data is monthly household income, net of taxes on earnings.[4] This measure sums the net incomes of all household members, including those who declined an interview or were interviewed by proxy. For individuals with missing data on any component of income, Understanding Society imputes values based on a wide range of personal and contextual information. Extensive detail is given in the *User Guide* (Knies, 2018). In the following order, values are equivalised, logged, top- and bottom-coded at the 99th and 1st percentiles, and within-wave standardised.

[4] As also reported by Hounkpatin et al. (2015) from their examination of two other sources of data, *personal* income correlated no more closely with depressive symptoms than did household income.

Equivalisation is applied using the modified OECD equivalence scale supplied with the data. This adjusts for household size and composition. The log transform is applied to reflect expected diminishing returns of income on mental health; in other words, linearity in the relationship is not expected to hold at extreme values (this was confirmed by a binned scatterplot – see Supplementary Figure S1 in the Appendix to this chapter). Top- and bottom-coding is used for the same reason, as well as to reduce the influence of outliers. The within-wave standardisation has two purposes: to ease interpretation and to adjust for inflation and shifts in the income distribution between waves.

Understanding Society income data has been validated against the Households Below Average Income series, the data source for official UK statistics on the income distribution, which is based on a high-quality specialist income survey, the Family Resources Survey (Fisher et al., 2019).

4.4.3 Employment transitions

Self-reported economic status is categorised as either *employed* or *not employed*. On this basis, four binary variables are constructed indicating each of the possible (non-)transitions in employment status across adjacent waves. I refer to these as *EN* (i.e. employed at *t–1*, not employed at *t*), *NE*, *EE*, and *NN*. Because the models place high demands on the data, in the analysis I aggregate *NE* and *EE* for simplicity, leaving **E* as the reference category – those currently in employment, regardless of their *t–1* status. The groups represented by *NE* and *EE* report similar GHQ scores (Thomas et al., 2005).

4.5 Analytical approach

I take a structural equation modelling (SEM) approach throughout the chapter, and consistently index the (unexplained) social gradient in mental health by the residual correlation between GHQ and household income z-scores; that is, the synchronous correlation between GHQ and household income that is not accounted for by any of the other processes specified in the model. In the figures below, the idiosyncratic error terms for income and GHQ are denoted by ε and υ respectively.

The first stage of the analysis is descriptive. To describe cross-sectional inequality in mental health at each age, I pool all available observations and, for each age from 16 to 70, report the income-mental health correlation (*a* in Figure 1a). This is done separately for men and women, as for all analyses in this chapter. Next, I explore to what extent any evident gradient remains, conditional on economic status. A dichotomous variable indicating whether or not the individual is employed is added to the model. This variable is included as a contemporaneous influence on both income and mental health (Figure 1b), and I again report *a*. Results from these preliminary steps (presented below) confirm two expectations from the literature reviewed above: that the gradient in mental health is negligible in late adolescence and grows over the following decades; and second, economic status explains a large portion of this cross-sectional inequality and should be central to a proposed causal model.

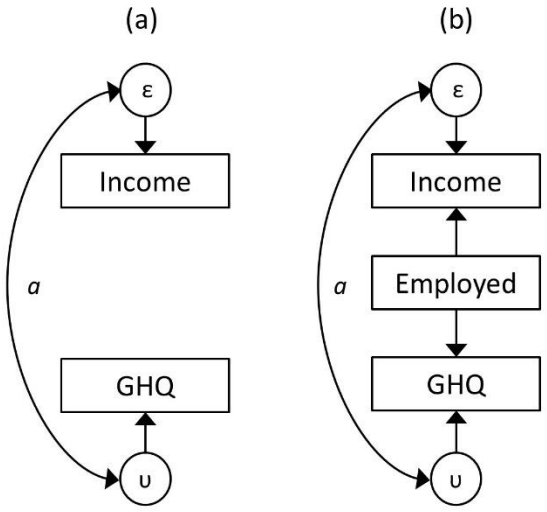


Figure 1 Cross-sectional structural equation models describing the social gradient in mental health, unconditionally (a) and conditional on economic status (b)

In the second stage of the analysis I introduce a sequence of six longitudinal models, utilising the eight available observations for each individual. In each case, the parameters of interest are constrained to be the same across waves. Age differences in the results are thus inferred from differences in results between older and younger individuals, not from differences within the same individual over time. This is discussed below.

Figure 2 shows the full model, Model 6. Model 1 includes path *a* only, reproducing the initial descriptive model shown in Figure 1a. Model 2 adds the autoregressive parameters *b* and *c*; this introduces the first-wave measures of income and GHQ, which are allowed to correlate (path *d*). With this addition, the residual correlations *a* are now conditional on income and GHQ at *t–1* and thus capture the year-to-year change in mental health inequality.

Models 3, 4, and 5 each separately add paths to this baseline model (2). The paths added represent the three hypotheses of social causation, health selection, and indirect health selection respectively. In Model 6 these terms are all included simultaneously.

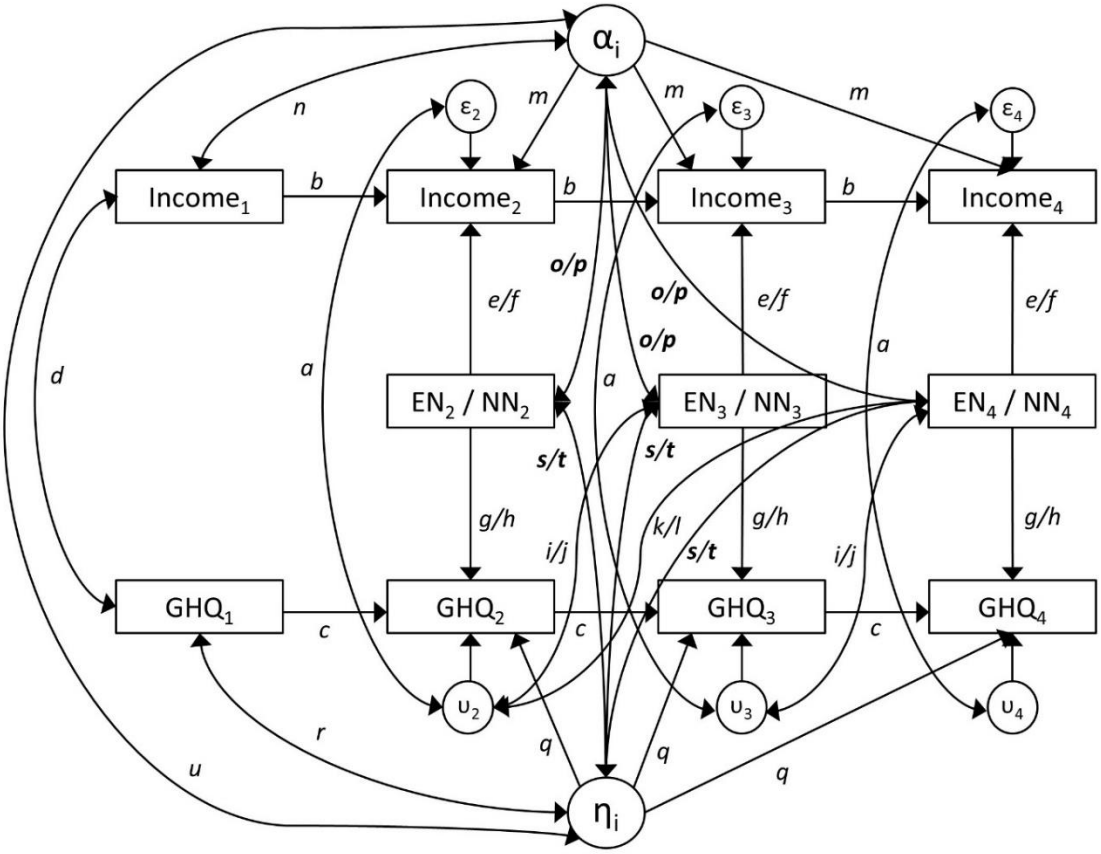


Figure 2 Longitudinal structural equation model (Model 6)
Note: Only four (of eight) waves shown. Employment transition indicators *EN* and *NN* combined for clarity; in the actual models they are separate (hence the two paths represented in one line by *e/f* etc.). Correlations between the two within and across time, and within each over time, are estimated but not shown here. Error variances and all paths except *o*, *p*, *s*, and *t* are constrained to be equal over time.

Model 3 adds the *t–1* to *t* employment transition indicators as causes of both income (paths *e* and *f*) and mental health at *t*. The paths *g* and *h* from employment transitions to mental health represent the mechanism of social causation. For example, EN_t -> GHQ_t represents the effect of moving out of employment between *t–1* and *t* on mental health at *t*. Estimating the effect of employment transitions rather than the (lagged or contemporaneous) effect of economic status has some advantages for causal inference. Recent work has highlighted that 'for continuously varying states … relying on the temporal ordering of the data can be much worse than useless' for determining causal relationships (Leszczensky & Wolbring, 2019; Vaisey & Miles, 2017: 63), requiring a condition which in many contexts it is impossible to know if one has met: that 'the lags between [observations] match the real-world causal lags in the processes under study' (Vaisey & Miles, 2017: 64). In addition, EN_t, for instance, captures both the effect of an event with a clear temporal position – between *t–1* and *t* – and the likely synchronous influence of economic status on both income and mental health. Life *events* are prominent in models of mental health problems as potential triggers of onset (Brown & Harris, 1978; Goldberg & Huxley, 1992). By contrast, modelling just the effect of contemporaneous economic status (conditional on lagged economic status) would give an estimate that aggregates two substantively distinct groups: those who leave employment, and those who remain non-employed.

Leaving employment or remaining non-employed may of course be caused by a deterioration in mental health. Model 4 tests the health selection hypothesis. It includes employment transitions as determinants of income as in Model 3, but not of mental health (paths *g* and *h* are constrained to zero). Health selection is modelled by allowing covariances (*i*, *j*, *k*, and *l*) between the error terms for GHQ at *t–1* and *t–2* and the employment transition indicators at *t*. (Covariances representing selection at all greater lags are also estimated but not reported). This parametrisation enables estimation of Model 6 (see below), but a more straightforward version – in which there are directed paths from GHQ at *t–1* and *t–2* to each of the employment transition indicators at *t* – gives almost identical results (Model 4a, reported in the Appendix).

As a test of the indirect health selection hypothesis, according to which stable, unobserved differences between individuals generate an association between income and mental health, while the two do not causally affect one another, Model 5 adds individual

fixed effects to Model 2 (α_i for income and η_i for GHQ). Under OLS, doing so introduces bias: whether the fixed effects are removed by demeaning or first-differencing, a correlation arises between the transformed lagged dependent variable and the transformed error term (Angrist & Pischke, 2009: 245; Halaby, 2004: 539; Nickell, 1981). However, recent work has demonstrated the feasibility of maximum-likelihood estimation of models including both fixed effects and lagged dependent variables (Allison et al., 2017; Moral-Benito, 2013). This is accomplished within a SEM framework by modelling the fixed effects as latent variables allowed to correlate freely with the time-varying covariates (Bollen & Brand, 2010; Mundlak, 1978; Teachman et al., 2001). The time-invariant effects of α_i and η_i are represented by *m* and *q*; *n*, *r*, and the vectors ***o***, ***p***, ***s***, and ***t*** allow unrestricted correlations with the time-varying covariates. The two fixed effects are not assumed to be uncorrelated (path *u*).

Finally, Model 6 includes all these paths, as represented in Figure 2. Given prior theory and evidence in favour of all three (not mutually exclusive) hypotheses, this model is the most plausible from which to draw estimates of the magnitude of each parameter. Further, it explores the extent to which growth in mental health inequality can be accounted for by the combination of the three mechanisms.

Conceptually, both employment transitions and mental health are dependent variables in this full model (under the health selection and social causation hypotheses respectively). For simplicity I only refer to the *EN* transition:

$$GHQ_t = cGHQ_{t-1} + gEN_t + \eta_i + \upsilon_{it} \quad (1)$$

$$EN_t = \beta_1 GHQ_{t-1} + \beta_2 GHQ_{t-2} + \nu_{it} \quad (2)$$

However, modelling these simultaneously as causal paths violates the assumption of strict exogeneity – that the disturbance terms are mean independent of all past, current, and future values of the independent variables (Halaby, 2004). For instance, under this system of equations, GHQ_{t-1} in (2) is determined by EN_{t-1} and therefore may correlate with its idiosyncratic component ν_{it-1}. Drawing on work developing a cross-lagged panel model with fixed effects estimated within a SEM framework (Allison et al., 2017; Williams et

al., 2018), I instead allow for health selection in the full model by eliminating (2) and instead including the covariances *i*, *j*, *k*, and *l*. This represents an assumption that employment transitions are not strictly exogenous but rather sequentially exogenous or predetermined: 'independent of all future values of [the time-varying error] but may be correlated with past values of [it]' (Allison et al., 2017: 3); that is, prior shocks to mental health may affect subsequent employment transitions. In a more general context, independent simulation results indicate that this approach deals well with reverse causality under a range of scenarios (Leszczensky & Wolbring, 2019).

In interpreting paths *g* and *h* in Model 6 from (*t–1* to *t*) employment transitions to GHQ (at *t*) as effects of the former on the latter, I make the assumption that the employment transition is not caused by a change in GHQ at some point *between t–1* and *t*. In support of this is the intuition that health selection effects are likely to be 'slow' relative to social causation effects. Whereas mood is susceptible to immediate influence, and income – though not determined wholly by employment – declines mechanically as a function of leaving employment, it may take some time for deteriorating mental health to reach the point where an individual opts or is forced to leave employment; this is likely to be a complex and relatively gradual social process.

Model 7, shown in the Appendix, presents an alternative specification for comparison: it is the same as Model 6 but without the lagged dependent variables (i.e. paths *b* and *c* are constrained to zero). One might prefer this specification under the assumption that income and mental health at *t–1* are not causally related to themselves at *t*, and that persistence in these two variables owes solely to time-invariant individual characteristics captured by the fixed effects. On this account, income and mental health are 'created anew' with each wave (Finkel, 1995: 8). The interpretation of individual parameters in the presence of fixed effects is also simplified by exclusion of the LDV terms. The choice between Models 6 and 7 is discussed further below.

The third stage of the analysis is to run the six models described above, now stratified by sex and age group, and examine differences in the estimated parameters. Defining age groups is not straightforward in a longitudinal context: doing so by age-at-observation splits individuals between age groups. For example, to define an age group as all person-years between the ages of 30 and 40 would be to discard half the information from an

individual followed from age 37 to age 44. To preserve the 'length' of the panel, I instead define age groups with reference to an individual's age in the middle of the observation period: that is (arbitrarily), their age at wave 4. To illustrate, the age group 30 to 40 now no longer 'cuts in half' the individual who is followed from age 37 to age 44, but includes them, using all eight observations. The label '30 to 40' is, correspondingly, not strictly accurate or entirely intuitive, and includes some observations of individuals when they are aged below 30 or over 40; however it maximises the use of information and most person-years do fall within the nominal age range. The age groups I use are 16-32, 33-49, and 50-66.

4.6 Results

4.6.1 Cross-sectional associations

Cross-sectional inequality in mental health at each age is shown in Figure 3. The pattern is generally similar among men (panel (a)) and women (panel (b)). The association between household income and GHQ score is close to zero at the youngest ages, gradually increasing until it becomes statistically significant for those aged 25, among both men and women. With a few exceptions, significant inequalities are evident at all ages past this. The strength of the association appears to increase steadily from its tentative emergence in the early 20s, up to the early 30s. From this age until the mid-50s there is volatility, especially among men, but in general the coefficients remain stable around –0.15 to –0.20. From the mid-50s to age 70 the association declines steadily, becoming nonsignificant among women and nearly so among men. Even at their largest, around –0.22, these correlations represent only modest associations. These descriptive findings offer cautious support for the idea that health inequalities are generated in the transition to adulthood.

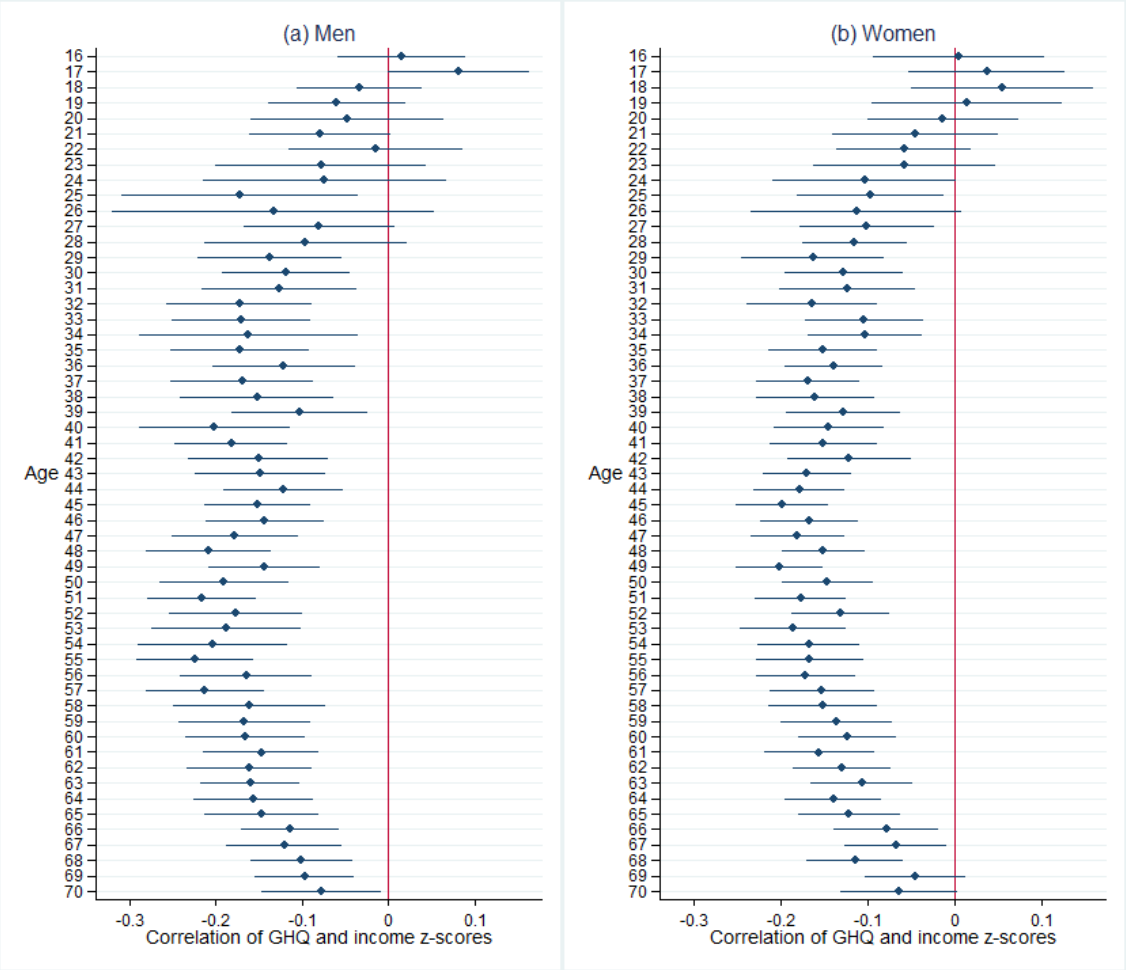


Figure 3 Mental health inequality by age and sex
Note: the correlations shown correspond to path *a* in Figure 1a. Higher GHQ score indicates worse mental health.

Figure 4 shows the same correlations, but with a control for economic status (employed versus not employed). Conditional on economic status the associations are substantially reduced, falling mostly in the range –0.05 to –0.10 and often not statistically significant among men (for whom the estimates are less precise due to a lower response rate). The association between income and depressive symptoms is largely accounted for by whether or not individuals are in work.

A simple explanation for the pattern observed in Figure 3 could be that the association varies as a function of the proportion of individuals at a given age who live with their parents. An individual's depressive symptoms are likely to be more closely related to their household income, the greater the extent to which that individual determines their household income – for instance by being a member of a smaller household. Where

individuals live with their parents, the latter are likely to primarily determine household income, and the two variables may be relatively 'insulated' from one another. Figure S2 in the Appendix to this chapter replicates Figure 3, separately for men and women who do and do not live with their parents. The association is weaker when individuals live with their parents, but at age 22 (and up to 24 for men), there is no evidence for the inequality having emerged even among individuals who no longer live with their parents.

4.6.2 Longitudinal SEMs

Before contrasting results between men and women of different ages, I first present longitudinal results for the aggregated sample, shown in Table 1. *a* from Model 1 represents the synchronous association between GHQ score and household income before any other elements are added to the model. At –0.139, this is in line with the correlations in Figure 3, though the latter highlights the heterogeneity concealed by aggregating the sample.

Model 2 adds the autoregressive paths *b* and *c*. These show large coefficients (0.717 and 0.551 respectively), reflecting a high degree of persistence in both variables and household income in particular. Though over half of the synchronous mental health inequality (path *a*) is accounted for by the association between these variables in the previous year, what remains is substantial (–0.060) and represents the year-to-year growth in the social gradient. This does not accumulate arithmetically because the autoregressive coefficients are less than 1; that is, individuals' GHQ and income tend to regress to the mean, attenuating the association between the two and offsetting part of the year-to-year growth.

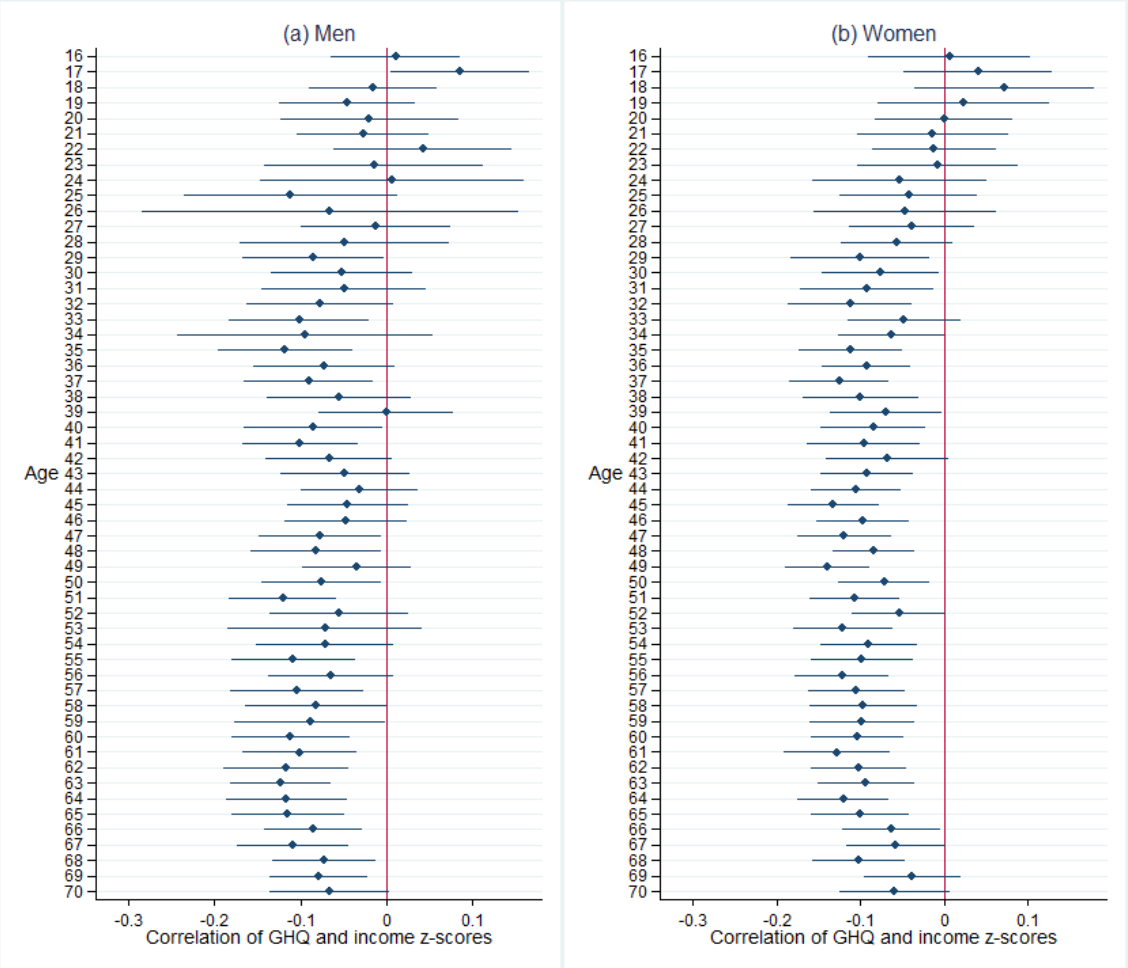


Figure 4 Mental health inequality conditional on economic status, by age and sex
Note: the correlations shown correspond to path *a* in Figure 1b. Higher GHQ score indicates worse mental health.

Model 3 introduces employment transitions. It is the first to achieve acceptable model fit by any measure, if only for RMSEA (0.070) (Little, 2013). As expected, moving from employed to non-employed is negatively associated with household income (path *e*; –0.639), and those non-employed in consecutive waves have lower incomes than those employed at *t* (path *f*; –0.233). These two indicators are also associated with worse mental health relative to individuals employed at *t* (paths *g* and *h*; 0.154, 0.161). Since GHQ is standardised and the population standard deviation is estimated at close to 5.5, each of these transitions is associated on average with close to one additional point on the 0-36 GHQ scale.

Model 4 adds covariance paths representing health selection to the baseline model. The unexplained portion of GHQ at both *t–2* and *t–1* covaries significantly with subsequent

moves out of employment (paths *i* and *k*). In the alternative specification (Model 4a, see Table S1 in the Appendix) some health selection is also evident, but in this case the association is between prior GHQ and remaining out of employment. Paths *a-f* are negligibly different in Model 4a compared to Model 4, while the latter shows clearly better fit.

Moving from Model 2 to Models 3 and 4, path *a* is reduced from –0.060 to –0.047 in both cases. Initially, this would appear to suggest that mechanisms of social causation and health selection each account for about a fifth of the year-to-year growth in inequality when looking at the aggregated sample. However, this reduction in path *a* in fact results simply from the addition of paths *e* and *f* (see Model 2a in Table S1); that is, from introducing employment transitions as determinants of income only (and unrelated to mental health). Paths *e* and *f* and the consequences of their addition cannot be 'claimed' by any of the hypotheses being tested; this addition can only be interpreted as a refinement in the measurement of SES. This leads to the surprising result that social causation and health selection as operationalised here, while both evident, do not themselves appear to account for any of the year-to-year growth in the social gradient.

Table 1 Longitudinal SEM path estimates and model fit statistics, aggregated sample

	Model	1	2	3	4	5	6
a	ε_t <-> υ_t	-0.139 ***	-0.060 ***	-0.047 ***	-0.047 ***	-0.038 ***	-0.029 ***
b	$Income_{t-1}$ -> $income_t$		0.717 ***	0.694 ***	0.694 ***	0.325 ***	0.303 ***
c	GHQ_{t-1} -> GHQ_t		0.551 ***	0.543 ***	0.552 ***	0.160 ***	0.160 ***
d	$Income_1$ <-> GHQ_1		-0.147 ***	-0.144 ***	-0.147 ***	-0.004	-0.030 ***
e	EN_t -> $income_t$			-0.639 ***	-0.634 ***		-0.636 ***
f	NN_t -> $income_t$			-0.233 ***	-0.227 ***		-0.399 ***
g	EN_t -> GHQ_t			0.154 ***			0.202 ***
h	NN_t -> GHQ_t			0.161 ***			0.126 ***
i	υ_{t-1} <-> EN_t				0.026 ***		0.039 ***
j	υ_{t-1} <-> NN_t				0.000		-0.004
k	υ_{t-2} <-> EN_t				0.024 ***		0.030 ***
l	υ_{t-2} <-> NN_t				0.013 ***		0.010
	Fixed effects	No	No	No	No	Yes	Yes
	RMSEA	0.274	0.108	0.070	0.072	0.035	0.030
	AIC	484512	319345	269260	308645	364836	257240
	CFI	0.016	0.835	0.821	0.820	0.984	0.971
	TLI	0.122	0.847	0.838	0.832	0.984	0.970
	SRMR	0.370	0.179	0.106	0.106	0.068	0.066

Note: *a*, *b*, *c* etc. refer to the paths shown in Figure 2. Paths with double-headed arrows represent covariances, reported here as correlations. N(individuals) = 17,843. Fit statistics from models without adjustment for survey design or attrition (unavailable otherwise).
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$

Model 5 adds individual fixed effects to the equations for income and mental health, accounting for the effects of any time-invariant unobserved characteristics of individuals, on the assumption that those effects do not vary over time. 51% of individuals in the sample are continuously employed across the eight waves and thus show no variation in the employment transition variables.[5] 26% of individuals experience at least one transition out of employment (*EN*), while 39% have at least one observation at which they are out of employment at both *t–1* and *t* (*NN*). Unobserved characteristics of the sort highlighted by the indirect health selection hypothesis appear to play an important role. The residual association *a* is substantially reduced compared with Model 2, at –0.038. As one might expect, the fixed effects also account for a large portion of the persistence in income and mental health, attenuating paths *b* and *c*.

[5] N does not change with the introduction of fixed effects because estimation is not achieved by differencing out the fixed effects or time-demeaning the data. Rather, fixed effects estimation is achieved by estimating (rather than constraining to zero as in a random effects model) the correlations between the latent variables (α_i and η_i) which represent the fixed effects, and the time-varying covariates (Allison et al., 2017; Bollen & Brand, 2010; Teachman et al., 2001).

Model 6 allows all the paths previously described. The unexplained synchronous association between income and depressive symptoms, *a*, is reduced to –0.029 (as above, the further reduction is most likely due to the inclusion of paths *e* and *f*), but not however completely accounted for. Estimates for the paths corresponding to social causation and health selection are generally similar to those from earlier models. *g*, the effect of moving out of employment on GHQ, is around a third larger (0.202), and the selection effects associated with this transition are also larger than previously. Since the coefficient for *NN* is somewhat reduced (0.126), the two transition variables here clearly differ in their effects on depressive symptoms. By most measures, Model 6 shows the best (and good) fit (RMSEA 0.030, CFI 0.971, TLI 0.970). While Model 7 (see Table S1) has a lower SRMR value, 0.066 is acceptable (Hu & Bentler, 1999). Model 7 itself, in which the lagged dependent variable is removed from each equation, gives very similar results and does not alter the conclusions.

The residual correlation between income and depressive symptoms net of the values of those variables from a year earlier (and the other influences in the model) is thus substantially smaller a) when income is made conditional on employment transition status, and b) when only within-individuals variation is considered. The former does not discriminate between the hypothesised mechanisms. There is minimal support for social causation and health selection in explaining growth in the association between income and mental health problems. However there is support for the indirect health selection hypothesis, which highlights between-individual differences from early life as important determinants of unequal SES and mental health trajectories.

4.6.3 Variation by age group

I now examine how these results vary when looking at men and women of different ages. I first present the disaggregated results from Model 6 (Table 2) and then examine change in path *a* across models for the different groups (Table 3).

Table 2 Longitudinal SEM path estimates for Model 6, by sex and age group

		Men			Women		
		16-32	33-49	50-66	16-32	33-49	50-66
a	ε_t <-> υ_t	-0.022	-0.031 **	-0.026 **	-0.011	-0.051 ***	-0.018 *
b	$Income_{t-1}$ -> $income_t$	0.356 ***	0.251 ***	0.301 ***	0.340 ***	0.267 ***	0.311 ***
c	GHQ_{t-1} -> GHQ_t	0.135 ***	0.138 ***	0.168 ***	0.173 ***	0.145 ***	0.168 ***
d	$Income_1$ <-> GHQ_1	-0.005	-0.098 ***	-0.026	-0.046	-0.030 *	-0.017
e	EN_t -> $income_t$	-0.687 ***	-1.120 ***	-0.830 ***	-0.371 ***	-0.453 ***	-0.580 ***
f	NN_t -> $income_t$	-0.458 ***	-0.597 ***	-0.473 ***	-0.353 ***	-0.357 ***	-0.324 ***
g	EN_t -> GHQ_t	0.241 ***	0.513 ***	0.261 ***	0.070	0.250 ***	0.122
h	NN_t -> GHQ_t	0.125 **	0.473 ***	0.172 **	0.043	0.200 ***	0.115 **
i	υ_{t-1} <-> EN_t	0.023	0.057 ***	0.074 ***	0.001	0.030 **	0.037 ***
j	υ_{t-1} <-> NN_t	-0.020 *	0.008	-0.009 *	-0.006	0.005	0.004
k	υ_{t-2} <-> EN_t	0.001	0.040 **	0.055 ***	0.014	0.018	-0.022
l	υ_{t-2} <-> NN_t	-0.021	0.032	0.020	-0.009	0.014	0.011
	N(individuals)	1461	2851	3143	2144	4114	4130

Note: see note to Table 1.
*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$

What do the disaggregated Model 6 results tell us about the strength of social causation and health selection across different life course stages for men and women? Moving out of employment has a greater impact on household income (*e*) among men and particularly those in the 33-49 group (–1.120). The impact on GHQ (*g*) is also largest for men (0.513) and women (0.250) in this age group, and generally larger for men than women across all age groups. Path *h* too is greatest for the 33-49 age group among both men (0.473) and women (0.200). These results confirm the prediction that social causation would be especially strong in mid-adulthood. By contrast, there is little evidence for health selection in the transition to adulthood: selection out of employment (*i* and *k*) is evident only among the older two age groups, while the significant correlation *j* for younger men is not in the expected direction.

How does the addition of processes of social causation, selection, and the introduction of fixed effects differentially affect the parameter *a* for the year-to-year growth of inequality in depressive symptoms? Table 3 presents estimates of *a* for each model by sex and age group.

Table 3 Path *a* across Models 1-6, by sex and age group

	Men			Women		
	16-32	33-49	50-66	16-32	33-49	50-66
Model 1	-0.092 ***	-0.149 ***	-0.171 ***	-0.093 ***	-0.161 ***	-0.129 ***
Model 2	-0.052 ***	-0.070 ***	-0.066 ***	-0.038 ***	-0.085 ***	-0.037 ***
Model 3	-0.031 *	-0.034 **	-0.061 ***	-0.021 *	-0.065 ***	-0.035 ***
Model 4	-0.029 *	-0.035 **	-0.050 ***	-0.019 *	-0.065 ***	-0.034 ***
Model 5	-0.043 **	-0.057 ***	-0.041 ***	-0.015	-0.060 ***	-0.019 *
Model 6	-0.022	-0.031 **	-0.026 **	-0.011	-0.051 ***	-0.018 *

*** $p < 0.001$, ** $p < 0.01$, * $p < 0.05$

The results for Model 1 again reflect the findings presented in Figure 3: the cross-sectional association between household income and depressive symptoms is larger in the older age groups, and similar for men and women of the same ages. As described above, Model 2 adds the autoregressive parameter and thus changes the interpretation of *a*. This produces somewhat surprising results for the 50-66 age group: *a*, though reduced to a greater degree than among the other age groups, remains substantial in magnitude and negative, suggesting that the gradient continues to grow across these ages when in fact it attenuates.

a is generally substantially smaller in Models 3 and 4 compared with Model 2. As above, this reduction is mainly due to the addition of paths *e* and *f*, from employment transitions to income (see Model 2a in Table S2). Model 5, which includes fixed effects for income and mental health, shows a pattern of reduction in *a* compared with Model 2 which varies across groups. The reduction is most pronounced among the 50-66 age group and among women in the 16-32 age group.

In the full model (6), *a* is generally reduced by more than 50% compared with Model 2. An exception to the pattern is women aged 33-49, for whom *a* remains relatively large in magnitude (–0.051 in Model 6, –0.085 in Model 2). This aside, differences between groups in Model 6 are rather small. Model 7 (see Table S2) gives similar results. What remains of *a* in Model 6 represents the synchronous residual association of income and GHQ, conditional on the previous year's values and individuals' means of those variables; what might account for this is discussed below.

4.7 Discussion

The aim of this paper was to use nationally representative longitudinal UK data to explore when and how the social gradient in mental health is generated. The gradient is operationalised here as the association between an individual's household income and depressive symptoms. Previous work has generally focused on testing for the *presence* of two mechanisms which each contribute to the formation of this inequality – social causation and health selection. The contribution of this chapter is to explore the extent to which each matters *for the generation of this inequality*, and at what stages of the adult life course they may do so. Further, in light of past research, this chapter uses a method that adjusts for unobserved heterogeneity – enabling direct testing of the indirect health selection hypothesis, and focuses on employment transitions due to their close links with health.

In line with past research on social inequality in mental health, the descriptive results showed the absence of an association between household income and depressive symptoms at age 16. Looking at person-year observations at increasing ages, the gradient becomes clearly evident by the mid-20s, and continues to grow into the mid-30s, stays relatively stable, before declining approximately through the mid-50s to 70. The same picture emerges for men and women.

Some previous work had found the association between income and depressive symptoms to be substantially rather small (and/or statistically nonsignificant) conditional on economic status (Hounkpatin et al., 2015; Oswald & Powdthavee, 2008). Adding an indicator for being employed to the initial cross-sectional model attenuated the association to a substantial degree. Accordingly, the longitudinal analysis focused on employment transitions as drivers of the social gradient in mental health.

A series of longitudinal SEMs was fit to test the extent to which the social causation, health selection, and indirect health selection mechanisms explain growth in mental health inequality – that is, the synchronous association between income and mental health conditional on the values of those variables in the previous year. Social causation was operationalised as the impact of leaving employment or remaining non-employed over the course of a year, health selection as the impact of shocks to mental health on

employment transitions over the following year(s), and indirect health selection as stable unobserved differences between individuals, modelled with individual fixed effects.

Theory and past research suggested that social causation would be especially important across both the transition to adulthood and mid-adulthood, and individuals would be particularly susceptible to health selection during the transition to adulthood. With regard to whether these mechanisms were evident, and the magnitude of effects of employment transitions on mental health and vice versa, the full model (including individual fixed effects) yielded support only for the prediction that social causation would be particularly strong in mid-adulthood. For individuals in the age range 33-49, being non-employed or, particularly, leaving employment, had a relatively large impact both on income and mental health. For men in this age group, leaving employment was associated with a sharp increase in symptoms of depression and anxiety relative to the same individual when in employment. The coefficient 0.513 is equivalent to approximately three points on the 0-36 GHQ scale. With regard to health selection, there were modest associations between negative shocks to mental health and leaving employment over the following year, but these were only evident among the mid-aged and older groups in the sample. An important caveat is that conclusions about differences in the strength of the different processes across life course stages rely on an assumption of no cohort or period effects.

With regard to whether and for which groups the three mechanisms explain growth in the association between income and mental health, there was surprisingly minimal support for either social causation or health selection, for men or women in any of the age groups. Apparent reductions upon introducing these mechanisms into the model in fact owed to the inclusion of paths from employment transitions to income. On the other hand, indirect health selection explained a modest portion of this growth – just over a third across the whole sample.

These results hint at the importance of the relatively neglected indirect health selection hypothesis. A role for stable unobserved differences between individuals observed in adulthood points to the complex range of genetic and early environmental factors which influence individuals' socioeconomic and wellbeing trajectories. However, the finding that neither social causation nor health selection contribute to generation of the social gradient in mental health through adulthood is paradoxical. There is strong prior evidence

that employment transitions both affect and are affected by mental health, and these associations were clearly evident here too. Given this, one would expect these mechanisms to act to increase the cross-sectional association between income and mental health. It may be that these processes are more complex than the data and methods applied here can capture.

A full model including all three hypothesised pathways did not completely account for the observed increases in mental health inequality. This residual association could arise from a combination of some kind of time-varying confounding (for instance, changes in marital status (Sbarra et al., 2014)), a causal effect of income on depressive symptoms (Hounkpatin et al., 2015) and, less plausibly, a relatively 'fast' causal effect of depressive symptoms on income.

The hypothesis that social inequality in mental health would increase over the course of the transition to adulthood was derived in part from earlier work which had reported 'equalisation' in adolescence. The equalisation hypothesis has been the subject of methodological debate. The issues raised are worth briefly reviewing for their possible relevance to the finding reported here – that indeed there is relative equality in mental health across household income levels in late adolescence. Criticism of the evidence for the equalisation hypothesis has centred on using occupation-based measures of SES to assess health inequalities in adolescence (Judge & Benzeval, 1993; Macintyre & West, 1991). One approach has been to use geographical deprivation indices; this has yielded further evidence of equalisation with respect to mortality and self-esteem, the latter outcome being the closest conceptually to mental health which has been examined in this way (Dibben & Popham, 2013; Fagg et al., 2013; Green, 2013). Others have continued to focus on the family but argued for household income as preferable to occupation-based measures. 'Important tasks, therefore', write Emerson et al., 'are to examine the negative patterning of health … using measures of [SES] which are inclusive and equivalent … Household income provides such a measure' (2006: 354). These authors also note that evidence for the equalisation hypothesis 'appears strongest … for some rather than all dimensions of young people's health, including … self-reported symptoms of psychological distress' (*ibid*.). It is perfectly plausible that the claim of equalisation varies in strength across health outcomes; this is not grounds for a wholesale rejection.

Due to potential objections to the inclusion of lagged dependent variables in the equations for income and depressive symptoms, I presented (in the Appendix) Model 7 in which these paths are removed. While Finkel (1995) generally advocates the LDV model for the analysis of change using panel data, Allison (1990) argues that in most situations the first-difference estimator is preferable.[6] The two exceptions are in the case of a causal effect of Y_{t-1} on Y_t, and 'when the *transient* (period-specific) components of [Y_{t-1}] … are correlated with X' (emphasis in original) (Allison, 1990: 109), X being a treatment that occurs between waves. Finkel (1995) further argues that even in the absence of a causal effect of Y_{t-1} on Y_t, an LDV is preferable in the presence of regression to the mean.

Even from the position of Allison (1990), there are grounds for including LDVs for income and depressive symptoms. For the former, causal mechanisms are plausible though likely to play a relatively weak role in determining future income. Greater income may allow individuals to make investments which generate higher future income; employees may accept jobs only on the condition that firms match or exceed their existing salary, regardless of other considerations; or employers may view past salary as the best available signal of an employee's value. Indeed income has been used as an illustrative case both of past values being likely to influence future values (Bollen & Brand, 2010; Finkel, 1995) and of the opposite (Allison, 1990). The other justifications for an LDV are more relevant for depressive symptoms. Correlations between the transient components of depressive symptoms (i.e. mental health shocks) and the 'treatment' here, employment transitions, are precisely what has been found, though not in every case. Regression to the mean is also particularly likely with respect to depressive symptoms. High GHQ score is a validated predictor of case-level depression (Lundin et al., 2016), which tends to present episodically. Recent psychiatric epidemiological research has argued for a distinction between the more common episodic or non-chronic depression, and chronic depression, with the latter representing about 20-30% of cases (Köhler et al., 2015; Murphy & Byrne, 2012). Even chronic depression, however, 'typically exhibit[s] a waxing and waning course' (Klein, 2008: 553). In sum, one can make a reasonable case for both Models 6 and 7; as it turns out, they yield the same substantive conclusions.

[6] Though cf. Allison et al. (2017) which develops a cross-lagged panel model with fixed effects as an alternative to econometric first-differencing-based methods, and which outperforms them in simulations (Leszczensky & Wolbring, 2019). Such cross-lagged models include lagged dependent variables.

One limitation of this paper is that the relative strength of social causation and health selection are not *directly* compared in terms of their magnitude, as for instance in the work of Chandola et al. (2003). This reflects the operationalisation of the hypotheses through employment transitions, which are necessarily modelled as binary variables. Nevertheless, the paradoxical result notwithstanding, the approach does allow a comparison of the two hypotheses with respect to their contribution to explaining growth in the association between income and mental health.

A second, more major limitation is the lack of specificity in the category 'not employed', and consequently in the meaning of the employment transition variables. Further analysis shows that for men aged 16-49, *EN* represents a transition into unemployment in most cases, while for women in these age groups the most common non-employment state is maternity leave or looking after the home or family; among 50-66 year-olds of both sexes retirement is the most common transition represented by *EN*. The approach taken here is the consequence of a trade-off: specifying more categories leads to an exponentially greater number of transition indicators. Some convergence problems occurred even when including the *NE* transition to differentiate between individuals employed at both *t–1* and *t*, and those employed at *t* but not *t–1*. The evidence of Steele et al. (2013) does at least indicate that for men (women were not analysed), effects on GHQ are in the same direction when contrasting transitions out of employment and into unemployment (2.22) or into economic inactivity (0.59), and of very similar magnitude when looking at individuals who are unemployed (0.40) or economically inactive (0.45) in successive waves. Further evidence from the BHPS indicates that transitioning into retirement is associated with a (nonsignificant) increased risk of 'caseness' under a dichotomised operationalisation of the GHQ (OR 1.23 for men, 1.41 for women), while transitioning from employment into long term illness (ORs 3.16, 2.09), maternity leave (1.52, women only), and family care (1.56, women only) were all significantly associated with a greater risk of high GHQ score (Thomas et al., 2005). This suggests that leaving employment is experienced as a *loss of benefits* (Anaf et al., 2013), as well as an introduction to the challenges particular to the new economic status.

The measure also obscures variation within the category 'employed' – but recall Richards and Paskov's (2016) finding of no social class gradient in mental health conditional on economic status. While gaining poor quality employment has been linked to worse

physical health outcomes even than remaining unemployed, this conclusion did not hold for a measure of mental health (Chandola & Zhang, 2018). Further, a recent analysis of UKHLS data finds that 'even a small number of working hours (between one and [eight hours] a week) generates significant mental health and well-being benefits for previously unemployed or economically inactive individuals' (Kamerāde et al., 2019: 1). On the positive side, this relatively low-resolution measurement of economic status and transitions therein allows for a methodological approach that is robust to unobserved heterogeneity, and to reverse causality acting over the course of a year or more.

A final caveat relates specifically to the fixed-effects aspect of the analytical approach used here. Fixed effects control for unobserved characteristics which do not vary *across the period over which individuals are observed*. Eight years is a considerable length of time to follow an individual, but it should be borne in mind that processes of social causation and health selection that occur prior to survey entry may affect individuals in ways which determine their subsequent mean levels of income and depressive symptoms; an obvious candidate for such a process is the proposed 'scarring effect' of unemployment on both future earnings and mental health (Arulampalam et al., 2001; Daly & Delaney, 2013). Inconveniently, this of course applies with more force the older the individuals in question.

The findings reported here reaffirm the importance of employment for depressive symptoms and suggest a partial redistribution of attention towards the relatively neglected – and not much elaborated – mechanism of indirect health selection. Past work has highlighted factors measured early in life which make large contributions to explaining social inequality in mental health in adulthood. For instance, teacher-rated ability across five domains at age 7 explained as much of the social gradient in mental health at age 33 as did job insecurity at age 33 (Power et al., 2002). Rather than consign early life influences and individual differences to 'unobserved heterogeneity', a priority for future work on social inequality in health should be to more clearly delineate what sorts of relatively fixed characteristics of individuals might matter, and through what sorts of pathways. At the same time, another clear conclusion of this chapter is that individuals leaving work – for whatever reason – are vulnerable to deterioration in their mental health and should be supported where possible, and in particular to find new employment if desired.

4.8 Appendix

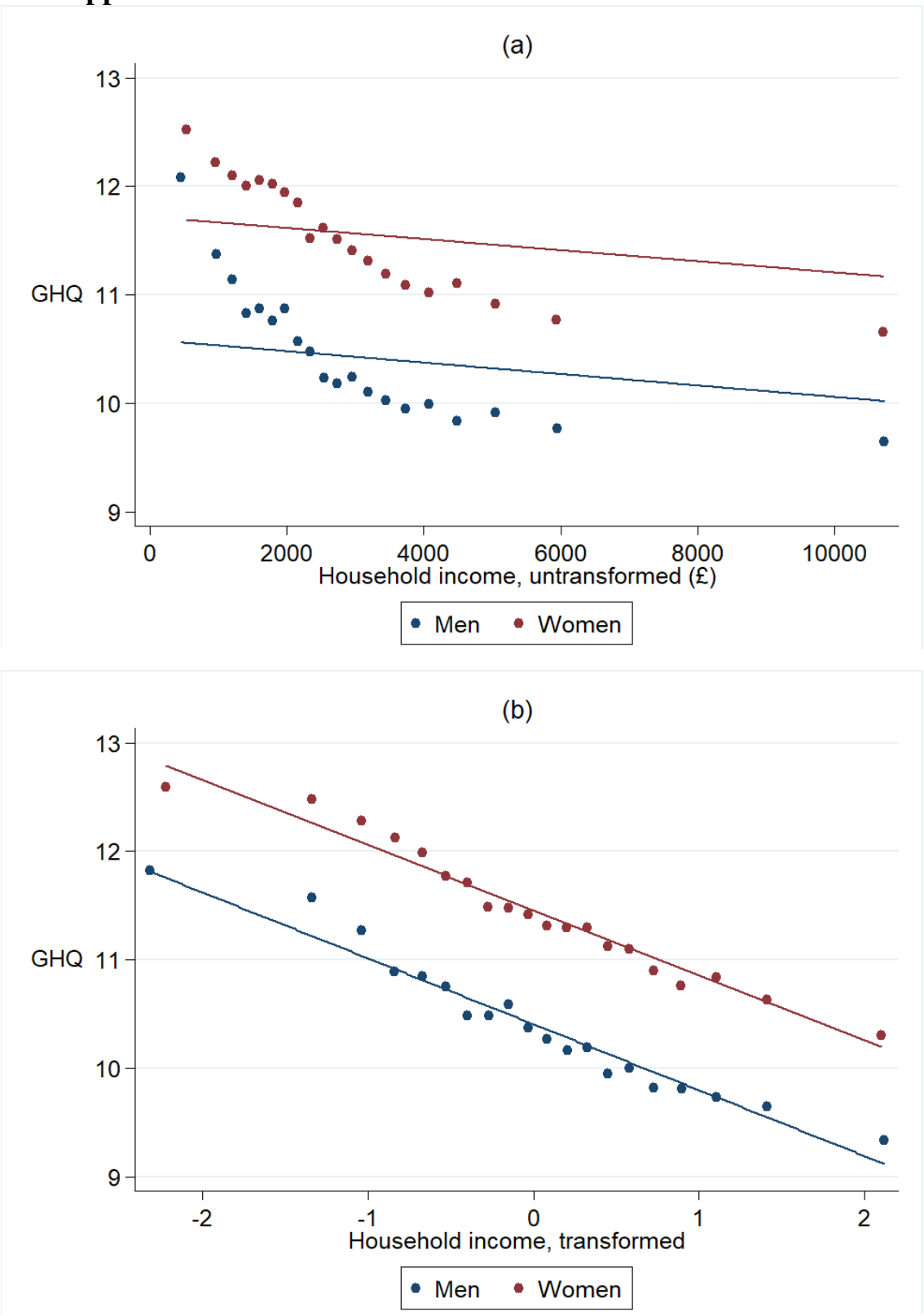


Figure S1 Binned scatterplots showing the association between GHQ score and monthly household income in its raw form (a) and transformed (b)

Note: see *Household income* above for details. Higher GHQ score indicates worse mental health.

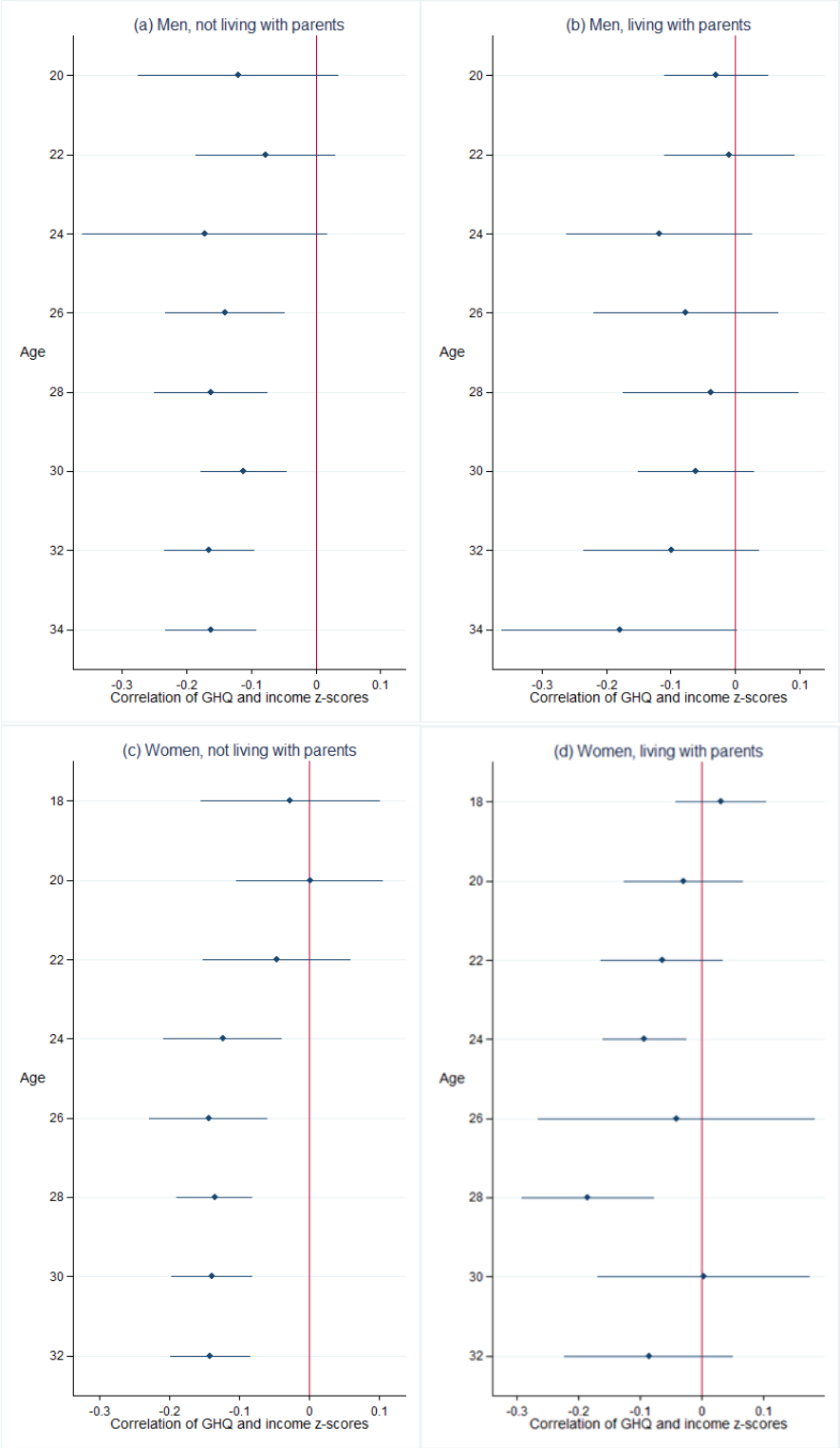


Figure S2 Mental health inequality by sex, age, and whether 'living at home with parent(s)'

Note: the correlations shown correspond to path *a* in Figure 1a. Higher GHQ score indicates worse mental health. Men aged 16-19 and women aged 16-17 omitted because very few not living with parents; women aged 33-34 omitted because very few living with parents.

General Health Questionnaire (GHQ-12) items and response categories

‘The next questions are about how you have been feeling recently.

Have you recently…

- been able to concentrate on whatever you're doing?[a]
- lost much sleep over worry?[b]
- felt that you were playing a useful part in things?[a]
- felt capable of making decisions about things?[a]
- felt constantly under strain?[b]
- felt you couldn't overcome your difficulties?[b]
- been able to enjoy your normal day-to-day activities?[a]
- been able to face up to problems?[a]
- been feeling unhappy or depressed?[b]
- been losing confidence in yourself?[b]
- been thinking of yourself as a worthless person?[b]
- been feeling reasonably happy, all things considered?[a]’

[a] Response categories: *More* [or *better*] *than usual* / *Same as usual* / *Less so than usual* / *Much less than usual*

[b] Response categories: *Not at all* / *No more than usual* / *Rather more than usual* / *Much more than usual*

Note: from a self-completion questionnaire module.

Table S1 Longitudinal SEM path estimates and model fit statistics, aggregated sample; including supplementary models 2a, 4a, and 7

	Model	1	2	2a	3	4	5	6	7
a	ε_t <-> υ_t	-0.139 ***	-0.060 ***	-0.047 ***	-0.047 ***	-0.047 ***	-0.038 ***	-0.029 ***	-0.030 ***
b	$Income_{t-1}$ -> $income_t$		0.717 ***	0.694 ***	0.694 ***	0.694 ***	0.325 ***	0.303 ***	
c	GHQ_{t-1} -> GHQ_t		0.551 ***	0.552 ***	0.543 ***	0.552 ***	0.160 ***	0.160 ***	
d	$Income_1$ <-> GHQ_1		-0.147 ***	-0.147 ***	-0.144 ***	-0.147 ***	-0.004	-0.030 ***	
e	EN_t -> $income_t$			-0.634 ***	-0.639 ***	-0.634 ***		-0.636 ***	-0.595 ***
f	NN_t -> $income_t$			-0.227 ***	-0.233 ***	-0.227 ***		-0.399 ***	-0.507 ***
g	EN_t -> GHQ_t				0.154 ***			0.202 ***	0.188 ***
h	NN_t -> GHQ_t				0.161 ***			0.126 ***	0.109 ***
i	υ_{t-1} <-> EN_t					0.026 ***		0.039 ***	0.033 ***
j	υ_{t-1} <-> NN_t					0.000		-0.004	-0.007 **
k	υ_{t-2} <-> EN_t					0.024 ***		0.030 ***	0.020 ***
l	υ_{t-2} <-> NN_t					0.013 ***		0.010	0.005
	Fixed effects	No	No	No	No	No	Yes	Yes	Yes
	RMSEA	0.274	0.108	0.085	0.070	0.072	0.035	0.030	0.046
	AIC	484512	319345	316139	269260	308645	364836	257240	243582
	CFI	0.016	0.835	0.746	0.821	0.820	0.984	0.971	0.938
	TLI	0.122	0.847	0.771	0.838	0.832	0.984	0.970	0.929
	SRMR	0.370	0.179	0.129	0.106	0.106	0.068	0.066	0.018

		4a
a	ε_t <-> υ_t	-0.047 ***
b	$Income_{t-1}$ -> $income_t$	0.696 ***
c	GHQ_{t-1} -> GHQ_t	0.552 ***
d	$Income_1$ <-> GHQ_1	-0.153 ***
e	EN_t -> $income_t$	-0.629 ***
f	NN_t -> $income_t$	-0.219 ***
	GHQ_{t-1} -> EN_t	0.002 *
	GHQ_{t-1} -> NN_t	0.040 ***
	GHQ_{t-2} -> EN_t	0.000
	GHQ_{t-2} -> NN_t	0.034 ***
	Fixed effects	No
	RMSEA	0.187
	AIC	447382
	CFI	0.349
	TLI	0.354
	SRMR	0.211

Note: see note to Table 1.

Table S2 Path *a* across all models, by sex and age group

	Men			Women		
	16-32	33-49	50-66	16-32	33-49	50-66
Model 1	-0.092 ***	-0.149 ***	-0.171 ***	-0.093 ***	-0.161 ***	-0.129 ***
Model 2	-0.052 ***	-0.070 ***	-0.066 ***	-0.038 ***	-0.085 ***	-0.037 ***
Model 2a	-0.032 *	-0.035 **	-0.057 ***	-0.021 *	-0.065 ***	-0.035 ***
Model 3	-0.031 *	-0.034 **	-0.061 ***	-0.021 *	-0.065 ***	-0.035 ***
Model 4	-0.029 *	-0.035 **	-0.050 ***	-0.019 *	-0.065 ***	-0.034 ***
Model 4a	-0.032 *	-0.036 **	-0.056 ***	-0.021 *	-0.066 ***	-0.035 ***
Model 5	-0.043 **	-0.057 ***	-0.041 ***	-0.015	-0.060 ***	-0.019 *
Model 6	-0.022	-0.031 **	-0.026 **	-0.011	-0.051 ***	-0.018 *
Model 7	-0.027	-0.034 **	-0.025 **	-0.013	-0.049 ***	-0.014

Note: compare Table 3.

5 Conclusion

This thesis has presented three sets of analyses into separate but related questions in the sociology of mental health. In this section I briefly recapitulate the conclusions of each of the empirical chapters, place them in context, and explore questions that have been raised.

The existing literature on the associations between the childhood environment and later life mental health is a large one. Nevertheless, there is much we do not know. Chapter 2 highlighted the enduring importance of social background for adult *women's* risk of mental health problems manifested as symptoms of depression and anxiety, in contrast to men. While gender differences in the risk of different sorts of mental health problems are widely-attested, this particular risk factor may be rather context-specific. The role of women in society has changed substantially, and what holds for the 1958 cohort may mean little for younger generations. Nevertheless, the question is raised of why the generalised disadvantage of low CSES should appear to matter more for women than men.

Evidence discussed in Chapter 1 on the gender disparity in depression highlighted the importance of what psychologists refer to as cognitive vulnerabilities to depression (such as a less instrumental approach to problem-solving, and external locus of control); no doubt these characteristics are shaped by context-specific and socially stratified socialisation patterns. The socialisation of women born in the cohort under study may have exhibited an especially important divergence along class lines. This period saw rapid advances in and changes of opinion about the position of women in society. The 'fundamental causes' theory of health inequalities posits that knowledge sits alongside money, prestige, power, and beneficial social connections as one of a set of flexible resources which high-SES individuals harness in ever-changing ways to maintain a health advantage (Phelan et al., 2010). If high-SES parents, aware of trends in gender equality, disproportionately socialised their daughters with full educational and labour force participation in mind, this may have had the effect of increasing their cognitive resilience, explaining the observed enduring effect of CSES. Such a pattern of diffusion of innovations, with earlier adoption among informed and connected high-SES groups, is

well evidenced in other health-related contexts (Mackenbach, 2012). A more focused test of this idea would be an interesting avenue for future research, and may inform policy in contexts where women are still educationally and economically marginalised.

The finding that the childhood environment has strong associations with mental health in adulthood, whether independent of later life circumstances or mediated through them, has implications for normative evaluations of the 'fairness' of health inequalities (Mackenbach, 2012). For instance, health behaviours such as smoking mediate part of the association between SES and health (Chandola & Jenkinson, 2000). While such behaviours are shaped by differential resources, access to knowledge, and social connections (Link, 2008), nevertheless many will observe that such health-endangering behaviours are an individual's own choice, and its outcomes their responsibility. This argument is less easily made when the factors influencing health and potentially generating inequalities are events or conditions which occur during childhood, and over which the child has no control. The response may be that the child's parents are responsible for the prevention of such adversities. However, such a response would likely be given by far fewer than would hold individual adults primarily responsible for the health outcomes of their own behaviours.

A third finding of Chapter 2 was that the measured variables in the childhood environment could not account for women's worse mental health, either through greater exposure (for instance, to sexual abuse) or greater vulnerability (to the stresses of low childhood socioeconomic status). The gender disparity in symptoms of depression and anxiety clearly deserves further attention. It should not be forgotten, meanwhile, that abuse of alcohol and other psychotropic substances, as well as what are termed 'impulse-control disorders', show a similar gender difference but with greater risk among men; indeed, within the psychiatric paradigm, lifetime prevalence of experiencing a mental disorder *of some kind* is estimated to be equal between men and women (Kessler et al., 2005). Measurement of problematic substance use is, however, far more unreliable than of depression and anxiety symptoms. There is a risk that some manifestations of psychological suffering can be paid less attention because they are less visible, and less easily incorporated into survey research.

Chapter 3 found that, as expected, conduct problems, emotional symptoms, and hyperactivity among adolescents are associated with their chances of achieving upward social mobility and avoiding downward social mobility. Taking the childhood environment into account attenuated the association of conduct problems with mobility by around 50%, indicating a substantial role for confounding and earlier processes of social causation in the generation of this health selection effect. On the other hand, the effects of emotional symptoms and hyperactivity on mobility outcomes were not significantly accounted for by childhood risk factors. Findings of health selection effects should come with a recognition that the mental health problems influencing social position may themselves be partly determined by prior social position. The mental health inequalities literature as a whole suggests a highly complex interplay between these processes, perhaps defying even adequately transparent description given the vast range of factors at play.

Further analyses indicated 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. This further highlights the finding among women in Chapter 2 that generalised disadvantage can matter for later mental health, to an extent comparable to (and independently of) the specific adversities of childhood which are widely recognised as traumatic.

The finding that the *hyperactivity* dimension of adolescent mental health shows associations not just with education (Breslau et al., 2011) and downstream maladaptive behaviour such as criminality (Savolainen et al., 2010), but also with *social mobility*, may be new, or at least extend a rather small body of evidence. Attention to the short- and longer-term socioeconomic consequences of hyperactivity and related constructs is an important area for future research – especially given the only recent recognition in the academic literature that the cognitive and behavioural features of ADHD do not simply disappear in adulthood, as appears to have been widely assumed in the past (Asherson et al., 2016).

In Chapter 4 I began by describing the distribution of mental health inequality by income across a wide age range in the contemporary UK. A clear pattern emerged of inequality emerging and increasing through the 20s and 30s, remaining relatively stable through

mid-adulthood, before decreasing from the mid-50s onwards. The granular and broad description of the profile of mental health inequality by age marks an addition to the descriptive evidence, which has tended to offer coefficients measuring inequality at a handful of more widely-spaced time points (Hoffmann et al., 2018), obscuring year-to-year patterns.

A series of longitudinal SEMs was fit to test the extent to which the social causation, health selection, and indirect health selection mechanisms explain growth in mental health inequality – that is, the synchronous association between income and mental health conditional on the values of those variables in the previous year. Social causation was operationalised as the impact of leaving employment or remaining non-employed over the course of a year, health selection as the impact of shocks to mental health on employment transitions over the following year(s), and indirect health selection as stable unobserved differences between individuals, modelled with individual fixed effects.

Theory and past research suggested that social causation would be especially important across both the transition to adulthood and mid-adulthood, and individuals would be particularly susceptible to health selection during the transition to adulthood. With regard to whether these mechanisms were evident, and the magnitude of effects of employment transitions on mental health and vice versa, the full model (including individual fixed effects) yielded support only for the prediction that social causation would be particularly strong in mid-adulthood. For individuals in the age range 33-49, being non-employed or, particularly, leaving employment, had a relatively large impact both on income and mental health. For men in this age group, leaving employment was associated with a sharp increase in symptoms of depression and anxiety relative to the same individual when in employment. The coefficient 0.513 is equivalent to approximately three points on the 0-36 GHQ scale. With regard to health selection, there were modest associations between negative shocks to mental health and leaving employment over the following year, but these were only evident among the mid-aged and older groups in the sample. These findings echo other work on the mental health implications of employment transitions (Steele et al., 2013).

With regard to whether and for which groups the three mechanisms explain growth in the association between income and mental health, there was surprisingly minimal support

for either social causation or health selection, for men or women in any of the age groups. On the other hand, indirect health selection explained a modest portion of this growth – just over a third across the whole sample.

These results hint at the importance of the relatively neglected indirect health selection hypothesis. A role for stable unobserved differences between individuals observed in adulthood points to the complex range of genetic and early environmental factors which influence individuals' socioeconomic and wellbeing trajectories. However, the finding that neither social causation nor health selection contribute to generation of the social gradient in mental health through adulthood is paradoxical. There is strong prior evidence that employment transitions both affect and are affected by mental health, and these associations were clearly evident here too. Given this, one would expect these mechanisms to act to increase the cross-sectional association between income and mental health. It may be that these processes are more complex than the data and methods applied here can capture.

There are several findings in this thesis of which I am confident. On the other hand, this inconclusiveness with respect to the role of social causation and health selection in *generating* mental health inequality is admittedly frustrating. At the same time it is instructive. Mental health is an extremely complex outcome; idiographic accounts show that individuals' idiosyncratic motivational structures and messy interpersonal relationships are central to generating their feelings of low mood, whether merely transient and bothersome or persistent and gnawing. Income and other dimensions of social stratification can appear quite distant when one reads case vignettes such as those presented by Nesse – for instance of the 'many depressed VIPs, mostly corporate vice presidents and deans' (2019: 183) he has treated, individuals enviably situated on every dimension of social position. There is clear value in nomothetic attempts to relate distal factors such as the structure of society and its implications for the distribution of status, resources, vulnerabilities, and risks, to mental health outcomes. It is difficult to ask for ever more granular measurement in regular, large-scale, longitudinal social surveys. Yet, what has been largely missing, including from this thesis, is micro-situational *links* between the two levels: proximal factors in the causation of mental health problems such as situations subjectively and somewhat reliably experienced as involving humiliation, entrapment, or futile effort. A priority for future research should be refinement of the

theoretical and empirical links between broad sociological ‘background’ variables and the micro-situational determinants of mental health, and particularly an increased attention to the latter among sociologists and social epidemiologists. In the quantitative literature I have read, an explicit theory of what brings about the feelings we call depression and anxiety is almost invariably absent, or its place held by a vague notion of ‘stress’; more progress might come from starting with an evolutionarily-informed model of mood and anxiety (Baron-Cohen, 1997; Brown, 2002; Nesse, 2000, 2019), and working ‘up’ from the micro to the meso and macro to infer how social arrangements compound and ameliorate our suffering, and how policy might orient us towards the latter.

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