HOW DO SOCIOECONOMIC HEALTH INEQUALITIES DEVELOP?

Exploring mechanisms in the development of socioeconomic inequalities in mental health and health behaviours in adolescents and young adults

Heiko Schmengler

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Hoe ontstaan sociaaleconomische gezondheidsverschillen?

Het verkennen van mechanismes in de ontwikkeling van sociaaleconomische ongelijkheden in de geestelijke gezondheid en het gezondheidsgedrag van jongeren en jongvolwassenen

(met een samenvatting in het Nederlands)

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> The enjoyment of the highest attainable standard of health is one of the fundamental rights of every human being **without distinction of race, religion, political belief, economic or social condition.**

Constitution of the World Health Organization (1948)

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CHAPTER 1

GENERAL INTRODUCTION

1. THE INTERACTIONIST MODEL OF DEVELOPMENT AND SOCIOECONOMIC HEALTH INEQUALITIES IN ADOLESCENCE AND YOUNG ADULTHOOD

Despite improvements in overall population health in many affluent countries over the past decennia, socioeconomic health inequalities have remained substantial (Hosseinpoor et al., 2012; Kunst et al., 2005), and in some countries even have grown (Elgar et al., 2015; Mackenbach, 2012; Mackenbach et al., 2003). These inequalities already become visible in childhood, when children growing up in lower socioeconomic status (SES; **Box 1.1**) households have higher risks of problematic health behaviours (Elgar et al., 2015), mental health problems (Reiss, 2013), as well as some somatic health problems (Spencer et al., 2013). These children are also more frequently selected into educational trajectories preparing for lower qualified professions with lower income and less job security (Bodin et al., 2022; de Mooij et al., 2012; van Spijker et al., 2017), affecting both their future socioeconomic prospects and health over the life course.

Box 1.1 Working definition of socioeconomic status (SES)

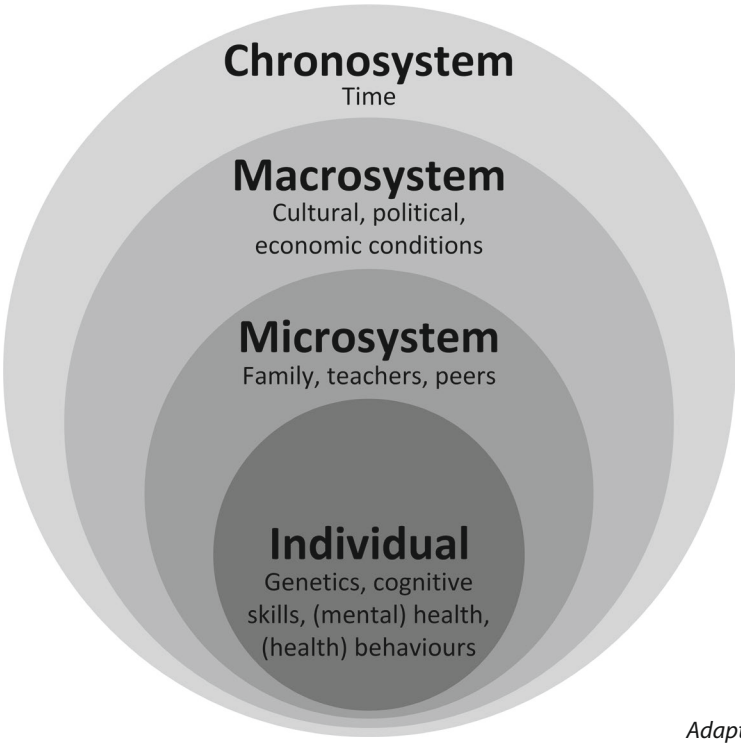
SES is a measure of social stratification, which refers to a person's position in society based on factors such as income, educational attainment, and occupational class. These factors are associated with 'flexible resources', such as knowledge, money, power, prestige, and beneficial social connections, which affect one's capability to avoid disease and minimize consequences once disease occurs (Batty et al., 2006; Clouston & Link, 2021; Link et al., 2008; Mackenbach, 2012). These resources are called 'flexible' because they are highly general and can be deployed to gain health advantages irrespective of the type of health condition, as long as it is preventable or its course can be intervened on (Clouston & Link, 2021).

By means of affecting financial, social, and cultural capital, SES is associated with considerable differences in the social environment in which adolescents grow up, including in the family, the neighbourhood, and at school. Resulting differences in socialization experiences can lead to differences in the social norms, tastes, and habits adolescents develop, including those related to health, which can become internalized into one's social identity as parts of a broader 'habitus' (Bourdieu, 1986; Djojosoeparto et al., 2022).

In addition to parental SES (as assessed by e.g., parents' educational attainment, occupational class, and income), adolescents' educational level constitutes a critical component of their developing SES. Educational level in adolescence is strongly predicted by parental SES (van Spijker et al., 2017), and itself predicts educational attainment in young adulthood, as well as later occupational class and income (Andersen & Van De Werfhorst, 2010; de Mooij et al., 2012).

The interactionist model of development (Conger & Donnellan, 2007) posits that socioeconomic health inequalities emerge in the context of a dynamic interplay between individual differences and the social environments at home, in the neighbourhood, and at school, which vary depending on parental SES and adolescents' – and later young adults' – educational trajectory. These social environments – in bioecological models of development (**Figure 1.1**) also referred to as microsystem – are influenced by the wider political, cultural, and economic conditions in each society (macrosystem). These conditions themselves depend on the historical period in which an adolescent or young adult lives (chronosystem). Elements of the micro-, macro-, and chronosystem interplay with adolescents' and young adults' individual differences in shaping development, including the development of health inequalities (Bronfenbrenner & Morris, 2007; Conger & Donnellan, 2007; Weinberg, 2021). Few attempts have been made to study this complex interplay in the emergence of socioeconomic health inequalities over the course of adolescence and young adulthood. This is a crucial omission because adolescence and young adulthood are critical periods for the development of health behaviours (Moor et al., 2015; Wiium et al., 2015), mental health (Solmi et al., 2022), as well as emerging SES (Breen, 2010).

FIGURE 1.1 Bioecological systems model



Adapted from Weinberg (2021).

2. ADOLESCENCE AND YOUNG ADULTHOOD AS CRITICAL PERIODS FOR THE DEVELOPMENT OF SOCIOECONOMIC HEALTH INEQUALITIES

Adolescence is a turbulent phase of life characterized by rapid physical, cognitive, and socioemotional changes. From adolescence to early adulthood, the brain undergoes substantial development and reorganization, including in areas associated with emotional regulation, decision-making, and social behaviour (Arain et al., 2013; Paus et al., 2008; Steinberg, 2008). Adolescence is also a time when individuals explore and develop their sense of personal identity (de Moor et al., 2022), and when friendships and peer groups become essential sources of support, validation, and socialization (Brown & Larson, 2009). These changes are often accompanied by marked increases in novelty seeking and a propensity towards risky behaviours, leading many adolescents to initiate substance use, including smoking and drinking (Steinberg, 2008). Other health behaviours, such as physical activity and diet, also tend to change during the transition from childhood to adolescence, and unfortunately often not in ways promotive of long-term health. For example, past studies have found declines in average levels of physical activity (Dumith et al., 2011), fruit and vegetable consumption (Albani et al., 2017), as well as the frequency of having breakfast in adolescence (Sincovich et al., 2022), whilst the consumption of some unhealthy products, such as soft drinks, has been found to increase (Inchley et al., 2020). In young adulthood, the transition into adult work and family roles is often accompanied by improvements in some health behaviours, in particular reductions in alcohol use (Green et al., 2017; Staff et al., 2010), be it that adolescents who initiated drinking at a very early age remain at higher risk of alcohol use disorder and other alcohol-related harms over the life course (Grant & Dawson, 1997).

Trajectories of health behaviours in adolescence and young adulthood are not uniform and may depend on a variety of individual and contextual factors (i.e., the micro-, macro-, and chronosystem). For example, adolescents to whom alcohol is readily available at home (Komro et al., 2007), and those part of peer groups characterized by high levels of substance use are more likely to initiate drinking themselves, in order to fit in and avoid social exclusion (Peeters et al., 2021). The impact of such contextual factors can depend on individual factors. For example, adolescents and young adults with relatively high levels of self-regulation are more likely to resist peer pressures and refrain from substance use (Peeters et al., 2017; Piehler et al., 2012). Health behaviours established in adolescence and young adulthood are associated with health behaviours later in life (Friedman et al., 2008; Paavola et al., 2004; Szabó et al., 2021; Wiium et al., 2015). This means that socioeconomic inequalities in health behaviours in adolescents and young adults tend to persist over the life course, hereby contributing to the substantial disparities in morbidity and mortality observed later in life (Katikireddi, Skivington, et al., 2017; Mehta et al., 2015).

Besides for the formation of healthy behaviours, adolescence and young adulthood are critical periods for the development of young people's mental health. According to a recent meta-analysis, 48.4% of mental disorders have their onset before age 18, and 62.5% before age 25, with a peak at age 14.5 years (Solmi et al., 2022). Some mental health problems, such as ADHD symptoms, tend to become salient already in childhood (Polanczyk et al., 2010), and others, such as externalizing problems, often increase strongly in adolescence (Moffitt, 1993; Nivard et al., 2017). While for many adolescents, symptoms persist into young adulthood (de Groot et al., 2022), for some adolescents, mental health problems, particularly externalizing problems and ADHD symptoms, subside to an extent in young adulthood (Moffitt, 1993; Sibley et al., 2021). Changes in the brain, if suboptimal in magnitude and timing (Paus et al., 2008), in combination with increased peer and academic stressors (Cosma et al., 2022; Norrington, 2021), as well as the challenges of identity formation (Klimstra & van Doeselaar, 2017; Potterton et al., 2022), all whilst emotional regulation and coping skills are still developing (Eschenbeck et al., 2018; Paus et al., 2008; Silvers, 2022), may contribute to the increased incidence of mental health problems in adolescence. Mental health problems can have a profound impact on a young person's life, not only by severely affecting their wellbeing and social relationships, but also by interfering with their educational career and impairing their potential to be upward socially mobile, whilst placing them at risk of downward social mobility (Anderson, 2018; Patel et al., 2007; Veldman et al., 2014). Considering that much, if not most, social mobility takes place in adolescence and young adulthood within the educational system (Breen, 2010), these phases of life are hence also critical periods for the formation of young people's SES.

The lack of research on the developmental mechanisms underpinning socioeconomic health inequalities leads to an incomplete understanding of how inequalities develop and why they are so persistent. The main aim of this dissertation is to study these mechanisms, and thus contribute to a more in-depth understanding on the origins of socioeconomic health inequalities, which is urgently needed to develop adequate policies and interventions targeting these inequalities already in adolescence and young adulthood. In particular, this dissertation aims to shed light on two types of processes, which are thought to underlie the emergence of socioeconomic health inequalities over the course of development: social causation and health-related selection (Conger & Donnellan, 2007). Both types of mechanisms are unlikely to act on their own, may interplay over time, may differ in their strength across phases of development, and cannot be understood without taking into account the role of the different elements of the microsystem (e.g., the family, neighbourhood, and school), macrosystem (i.e., the broader societal or national context), and chronosystem (i.e., the specific historical period) in which young people grow up (Bronfenbrenner & Morris, 2007). While some socioeconomic disparities in physical health are already present in adolescents and young adults, inequalities in mental health and

health behaviours typically tend to be larger (Hale et al., 2015; Howe et al., 2013; Mikkonen et al., 2020, 2021), which is why these inequalities are the focus of this dissertation.

3. SOCIAL CAUSATION AND HEALTH-RELATED SELECTION MECHANISMS IN THE DEVELOPMENT OF SOCIOECONOMIC INEQUALITIES IN MENTAL HEALTH AND HEALTH BEHAVIOURS

Two types of mechanisms may contribute to the development of socioeconomic inequalities in adolescents' and young adults' mental health and health behaviours: social causation and health-related selection.

3.1. Social causation mechanisms

Social causation assigns an important role to the social context as predictor of health inequalities. This includes socioeconomic differences in the home and school context, as key components of the microsystem. Differences in the social context across educational trajectories may contribute to educational differences in mental health and health behaviours between these trajectories. At the same time, parental SES may still play an important role in the formation of adolescents' health and health behaviours. Parental SES is also very strongly associated with the type of educational trajectory which adolescents enter (van Spijker et al., 2017), and therefore may be considered a shared predictor of adolescents' and young adults' education, mental health, and health behaviours. Children from lower SES families more frequently grow up in disadvantaged and less secure neighbourhoods, and their parents more often lack the financial means to provide a save and stimulating environment, which is highly important for children's cognitive and emotional development (Ng-Knight & Schoon, 2017; Rosen et al., 2018). This may place these children at higher risk of mental health problems, including both internalizing and externalizing conditions (Amone-P'Olak et al., 2011; Piotrowska et al., 2019; Reiss, 2013; Russell et al., 2016). Health behaviours also frequently vary depending on family SES. For example, it has been found that lower SES parents often show a higher permissiveness towards underage drinking, and correspondingly higher alcohol use has been found in young adolescents growing up in lower SES households (Foxcroft et al., 2022; Pape et al., 2017). Also, lower SES parents tend to have fewer resources to support the education of their children or to vouch for them at school. Accordingly, parental SES is a strong predictor of adolescents' educational trajectory, above and beyond their performance on standardized tests. For example, it has been found that in the Netherlands children of higher SES parents more often receive recommendations for the higher educational tracks than would be expected based on their score on the Cito test, which is a standardized test to measure, among other things, children's skills in mathematics, reading, and writing by

the end of elementary school (Douma et al., 2022; van Spijker et al., 2017). After selection into different educational trajectories, adolescents are exposed to different social norms, stressors, occupational prospects, and future expectations, depending on their educational track, which might contribute to educational inequalities in adolescents' – and later young adults' – mental health and health behaviours (Berten et al., 2012; Elstad, 2010; Peeters et al., 2021; Van Houtte & Stevens, 2008).

3.2. Direct and indirect health-related selection mechanisms

Health-related selection can refer to two types of processes, which are referred to as direct and indirect health-related selection in the public health literature. Direct health-related selection refers to processes by which health problems, including mental health problems, such as attention problems and externalizing behaviours, and some health-risk behaviours (e.g., intensive alcohol use) influence young people's emerging socioeconomic position (Jensen et al., 2023; Mackenbach, 2012; Van Hoof et al., 2018; Veldman et al., 2014). Mental health problems may lead to lower long-term educational attainment, for instance, by means of impairing educational performance (e.g., in case of ADHD symptoms) (Polderman et al., 2010), and leading to decreased educational expectations and less engagement in school (e.g., in case of externalizing behaviours) (McLeod & Fettes, 2007; Mikkonen et al., 2021; Olivier et al., 2020). Similarly, heavy alcohol use has been associated with cognitive impairments in adolescents (Peeters et al., 2014) and hereby may contribute to lower long-term educational outcomes.

Indirect health-related selection refers to the role of individual differences already present in childhood that predict later educational attainment, as well as health or health behaviours, and hereby contribute to associations between educational trajectories, health, and health behaviours as 'third variables' (Mackenbach, 2012). These variables may include genetic dispositions and genetically influenced phenotypic characteristics, such as children's effortful control and general intelligence (Deary et al., 2006; Yamagata et al., 2005), which have been associated with later educational attainment (Brody, 1997; Veronneau et al., 2014), as well as mental health and health behaviours in past studies (Daly & Egan, 2017; Daly et al., 2016; Deary et al., 2021; Kavish et al., 2020; Kubička et al., 2001; Meehan et al., 2013; Moffitt, 1993; Peeters et al., 2017; Wedow et al., 2018). It is, however, important to note that while genetics may play a strong role in the formation of many of the phenotypic characteristics underlying indirect health-related selection, preceding social causation mechanisms may also contribute to the development of these characteristics. For example, past studies have found associations between socioeconomic deprivation in early childhood and lower levels of effortful control and general intelligence (Lawlor et al., 2005; Farah, 2017; Ng-Knight & Schoon, 2017; Sturge-Apple et al., 2017; Zhang et al., 2020).

General intelligence refers to the content- and context-free ability to process information of any type, learn quickly, reason, and solve problems (Gottfredson, 2004). Effortful control

refers to an individual's capacity to self-regulate, inhibit impulses, delay gratification, and shift and sustain attention and effort towards long-term goals, even if tasks are unpleasant and distractions are present (Nigg, 2017; Piehler et al., 2012; Veronneau et al., 2014). At school, general intelligence is a key factor in determining a student's ability to learn and process information, and to apply knowledge in tests (Brody, 1997). Formal education is also very taxing on effortful control, as it requires adolescents and young adults to concentrate for extended periods of time, manage time effectively, and to complete tasks that are often not very interesting to them. Unsurprisingly, both general intelligence and effortful control are amongst the strongest cognitive predictors of educational attainment (Brody, 1997; Veronneau et al., 2014). While general intelligence shows some variability in infancy and childhood, it tends to be relatively stable from early adolescence onwards (Deary et al., 2013; von Stumm & Plomin, 2015). Conversely, after a dip in early adolescence, effortful control tends to increase substantially as adolescents become young adults (Atherton et al., 2020a). In selective educational systems like in the Netherlands, the levels of cognitive skills even before adolescence are particularly critical for long-term educational outcomes, as they play a strong role in determining into which educational track children are selected at an age as early as 11-12 (Schmengler et al., 2022). After this initial selection, educational track membership tends to be relatively stable (Schmengler et al., 2022), suggesting that the potential of cognitive skills to influence long-term educational careers may indeed be strongest during the transition from primary to secondary school.

Good cognitive skills are not only highly beneficial in the context of formal education, but also in many, if not most, other domains of life, including health. Higher intelligence has been associated with better health literacy (Gottfredson, 2004) as well as with better health behaviours, including less tobacco use both in adolescents and adults (Daly & Egan, 2017; Deary et al., 2021; Kubička et al., 2001; Weiser et al., 2010). High effortful control is also associated with good health behaviours, as it allows individuals to desist from behaviours that provide short-term gratification (e.g., substance use), in favour of pursuing long-term health benefits (Piehler et al., 2012). It is thought that an on average relatively weak effortful control in combination with high reward sensitivity explains why many adolescents engage in health-risk behaviours (Peeters et al., 2017; Steinberg, 2008). Despite this, adolescents differ substantially in their trajectories of effortful control (Atherton et al., 2020a), and adolescents with relatively low levels of effortful control compared to their peers may be at particular risk of escalating in risky behaviours, including substance use (Peeters et al., 2017). Both IQ and effortful control could also be considered proxies of suboptimal and optimal general neurodevelopment, and accordingly low scores on both cognitive skills have been related to various developmental problems, including externalizing behaviour and attention problems in adolescence (Atherton et al., 2020b; Deary, 2012; Kavish et al., 2020; Krieger et al., 2019; Moffitt, 1993; Rommelse et al., 2017; Schlotz et al., 2008). The benefits of high effortful control and intelligence may be so general that these cognitive

skills could, similar to the 'flexible resources' associated with SES (**Box 1.1**), be deployed to gain advantages across multiple, if not most, domains of health (cf., Gottfredson, 2004).

3.3. The interplay of social causation and health-related selection over the course of development

It is likely that both social causation and health-related selection mechanisms contribute to health inequalities by dynamically interplaying with each other over the life course, which is why it is important to study both types of mechanisms simultaneously (Hoffmann et al., 2018; Lundberg, 2020). For example, it is possible that someone enters a lower socioeconomic group due to a health problem (i.e., direct health-related selection), and subsequently their health deteriorates further due to contextual risk factors associated with being in that group (i.e., social causation). This is also why some researchers argue that health-related selection mechanisms should primarily be viewed as allocation mechanisms into socioeconomic groups, rather than an alternative explanation to social causation (Mikkonen, 2021).

During adolescence and young adulthood, young people may move out of the SES of their parents and move into their own SES, hereby experiencing the impact of both social causation and health-related selection. The Netherlands has a selective educational system, characterized by an early selection into different classrooms – and hereby different social contexts – based on standardized testing and the primary school's recommendation (van Spijker et al., 2017). Subsequently, a proportion of students is mobile, mostly between adjacent educational tracks. The selection into educational tracks at an age as early as 11–12 years means that Dutch adolescents grow up in distinct educational environments that are characterized by different social norms, future expectations, cognitive resources, and occupational prospects (Berten et al., 2012; Elstad, 2010; Van Houtte & Stevens, 2008) – characteristics that are closely related to conceptualizations of SES in adulthood (Mackenbach, 2012). One could therefore argue that in selective educational systems, such as in the Netherlands, youngsters move into 'their own' SES at a much earlier age than in comprehensive systems, such as in Finland or the USA. Data from Dutch longitudinal adolescent cohorts provide a unique opportunity to investigate both the antecedents and consequences, in terms of mental health and health behaviours, of this differentiation and subsequent intragenerational social mobility in adolescents and young adults.

So far, the roles of social causation and health-related selection in explaining educational differences in mental health problems, as well as substance use, have not been thoroughly investigated in a selective educational system like in the Netherlands. We therefore investigated bidirectional associations (i.e., direct health-related selection and social causation) between alcohol use and educational level (**Chapter 2**), as well as between externalizing behaviour and attention problems and educational level (**Chapter 3**) in Dutch adolescents and young adults from around age 11 to around age 26. Whilst doing so, we

also took into account social causation from parental SES, as well as indirect health-related selection effects related to cognitive skills in childhood. Out of all mental health problems, attention problems (and related constructs, such as ADHD symptoms) and externalizing behaviour have been especially consistently associated with a lower educational level in adolescence (Evensen et al., 2016; Meißner et al., 2022; Van Houtte & Stevens, 2008; Veldman et al., 2014), which is why we decided to focus on these problem behaviours in our studies. Subsequently, in **Chapter 4**, we explored developmental mechanisms linking genetic risk factors for lower educational attainment and smoking, as measured using polygenic scores (PGSs), to educational inequalities in tobacco use in adolescence and young adulthood. Specifically, we evaluated whether genetic variants underlie cognitive skills in childhood (i.e., IQ and effortful control), and whether these skills are then directly associated with both later smoking and educational level, consistent with indirect health-related selection related to cognitive skills. Additionally, we explored whether genetic variants are associated with inequalities in smoking indirectly by predicting placement into educational trajectories, and hereby differences in the social context, which in turn may predict smoking (consistent with social causation explanations).

3.4. Aspects of the family and school context (i.e., the microsystem) as potential mediators or moderators of direct health-related selection processes

When health behaviours or mental health problems adversely influence educational prospects (i.e., direct health-related selection), they always do so within the social context in which they occur. For example, the family and school context, which are key elements of the microsystem in bioecological systems models (Bronfenbrenner & Morris, 2007), can influence how strongly, for instance, mental health problems affect education. Mental health problems may be less consequential for the academic performance of adolescents who are well supported by their teachers (e.g., in the form of accommodations) and come from well-functioning families possibly better equipped to handle their child's symptoms. At the same time, the social environment frequently responds negatively to adolescents' problematic behaviours (Ewe, 2019; Glatz et al., 2011; McQuade, 2020), and this may deprive these adolescents of resources critical for their academic development.

In **Chapter 5** we explored the role of three key aspects of adolescents' immediate social context (microsystem) in associations between ADHD symptoms and lower education: social support by teachers, social support by classmates, and family functioning. So far, little research has focussed on risk and protective factors related to the social environment in the context of ADHD and educational attainment (Dvorsky et al., 2018). This is surprising given that ADHD symptoms have been strongly associated with problems in relationships with parents, teachers, and peers in past research (Ewe, 2019; Glatz et al., 2011; McQuade, 2020), whilst many other studies have highlighted the importance of these relationships

for academic development in general (Lin et al., 2019; Robertson & Reynolds, 2010; Roorda et al., 2017; Tao et al., 2022; Wentzel et al., 2021). We aimed to add to the literature by investigating whether ADHD symptoms contribute to poorer family functioning and less social support by teachers and classmates, and consequently a lower educational level (i.e., mediation). While doing so, we also considered potential interactions between ADHD symptoms and these family and school factors, to assess whether ADHD symptoms are less consequential for the education of adolescents who feel well supported by their teachers and peers, and who come from well-functioning families.

4. THE ROLE OF THE NATIONAL CONTEXT (MACROSYSTEM), AND IN PARTICULAR COUNTRY-LEVEL SOCIAL MOBILITY, AS MODERATOR OF ASSOCIATIONS BETWEEN FAMILY AFFLUENCE AND ADOLESCENT HEALTH BEHAVIOURS

The broader social and cultural context in a country (i.e., the macrosystem) may influence the strength of mechanisms underpinning the development of socioeconomic inequalities in health and health behaviours. One national-level characteristic that is hypothesized to play a role in the persistence of socioeconomic health inequalities, even in states with extensive welfare arrangements, is country-level social mobility (Mackenbach, 2012). If country-level social mobility is high, individuals' own SES is less dependent on their parents' SES and therefore potentially more dependent on personal characteristics, including cognitive skills, such as IQ and effortful control – individual factors that are also associated with health behaviours and a myriad of other health outcomes (in line with indirect health-related selection), as mentioned above (Mackenbach, 2012). During the post-war period, social mobility rose substantially in many high-income countries, facilitated by egalitarian policies (Breen, 2010; Mackenbach, 2012). If individuals with good cognitive skills and other resilient personal characteristics increasingly moved upwards, while those with poorer skills and other less resilient personal characteristics remained or ended up in the lower socioeconomic groups, stronger social mobility on the country-level during the post-war period could have led to an increasing homogenization of the lower-SES groups in terms of relatively poor health and lower cognitive skills (Mackenbach, 2012; Simons et al., 2013). Cognitive skills and many other health-related personal characteristics of parents and children tend to be correlated, which may be due to both genetic and environmental transmission (Kim & Kim, 2019; Scarr et al., 1993; Willems et al., 2019; Willoughby et al., 2021). It is therefore possible that increased socioeconomic inequalities in cognitive skills and other health-related personal characteristics in past generations may have led to widening inequalities in these characteristics by parental SES in the current generation of adolescents. While, to my knowledge, appropriate cross-national data to address this

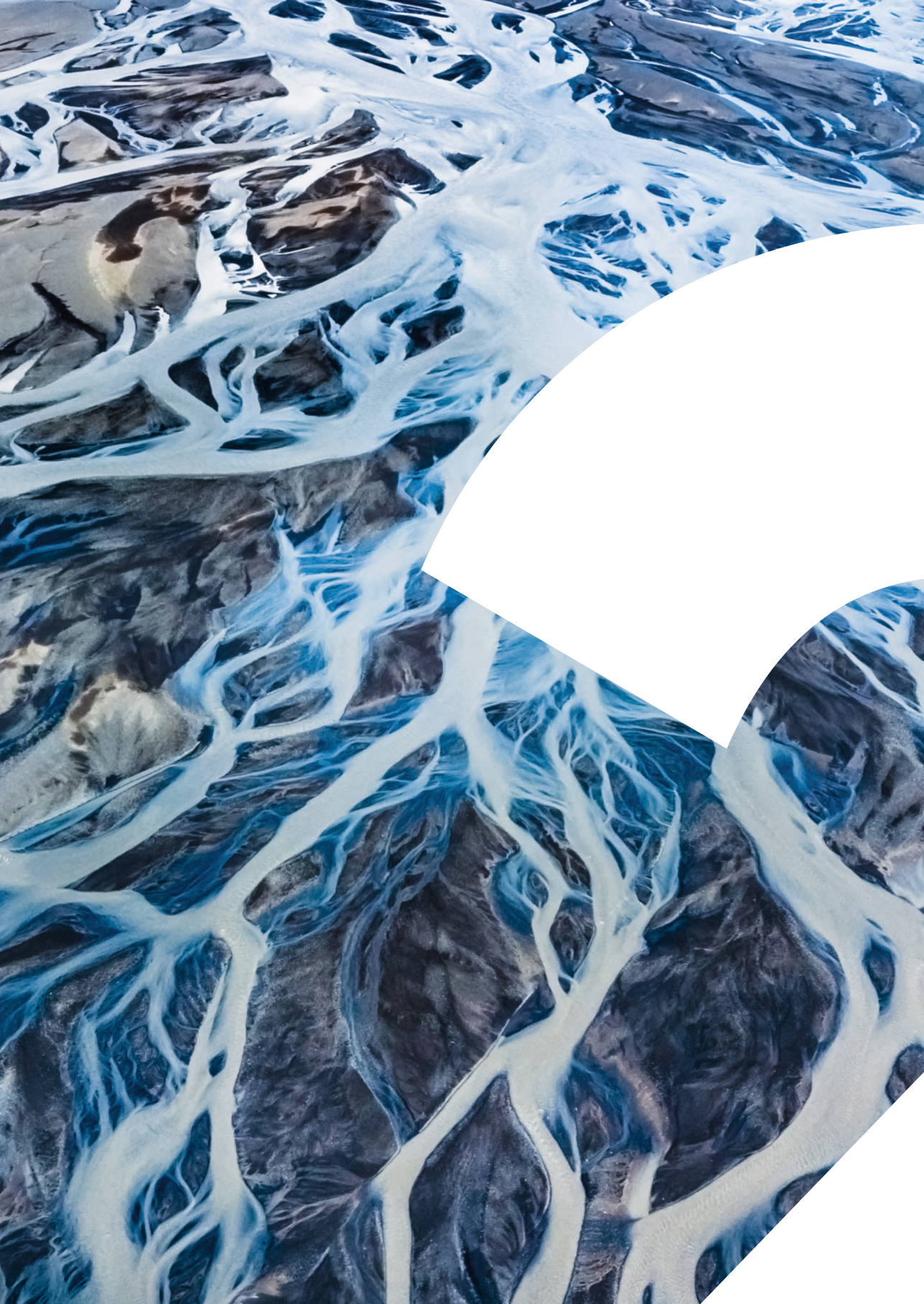
question are not yet available, it is possible to study whether associations between lower parental SES and poorer health behaviours, which are associated with cognitive skills (Beenackers et al., 2017; Daly et al., 2016; Junger & van Kampen, 2010; Kubička et al., 2001), are stronger in countries with higher levels of social mobility during the past generations.

It is important to note that the relationship between country-level characteristics like national-level social mobility and health inequalities is highly complex and may involve a multitude of mechanisms that affect inequalities in opposite directions. For example, it is possible that socioeconomic inequalities in adolescent health behaviours might, in fact, be smaller in countries with higher levels of social mobility due to other characteristics of such countries. Highly socially mobile countries are often characterized by equitable social policies (Breen, 2010; Esping-Andersen, 2015; Pförtner et al., 2019; Rathmann et al., 2015), which may partially offset disadvantages faced by low-SES adolescents, weakening social causation effects emanating from the home environment. Additionally, if adolescents from low-SES families feel that they can benefit from social mobility and reach a higher SES then their parents, they may be more strongly oriented towards the future, which has been associated with better health behaviours in adolescents (Bak & Yi, 2020; Ritterman Weintraub et al., 2015). Thus far, to my knowledge, there have only been studies investigating the potential moderating role of contextual-level social mobility in socioeconomic health inequalities in adults, with conflicting results (Consolazio et al., 2022; Simons et al., 2013; Venkataramani et al., 2020), but research among adolescents is lacking. In **Chapter 6**, we therefore investigated whether social mobility at the country-level during the past generations moderates associations between family affluence and adolescent health behaviours.

5. METHODOLOGICAL APPROACH

This dissertation uses the TRacking Adolescents' Individual Lives Survey (TRAILS), which consists of a population cohort (N = 2,229), and clinical cohort (N = 543) of adolescents followed up from age 11 to 26, to explore mechanisms explaining educational inequalities in mental health and substance use in the Netherlands. The population cohort was recruited from 135 schools in the provinces of Groningen, Friesland, and Drenthe, of which 122 decided to participate (de Winter et al., 2005). The clinical cohort consists of adolescents who had been referred to the Groningen University Child and Adolescent Psychiatric Outpatient Clinic at any point in their life for consultation or treatment (20.8% ≤5 years, 66.1% 6–9 years, 13.1% 10–12 years). A detailed description of TRAILS can be obtained elsewhere (Oldehinkel et al., 2015). **Chapters 2 and 3** use wave 1 to 6 (around age 11 – 26), and **Chapter 5** wave 1 to 4 (around age 11 – 19) of the population-based cohort. **Chapter 4** combines data from the population cohort with the clinical cohort (waves 1 – 6; around

age 11 – 26). **Chapter 6** uses data from 32 countries participating in the 2017/2018 edition of the Health Behaviour in School-aged Children (HBSC) study (N = 185,086), an international cross-sectional survey investigating health behaviours of adolescents aged 10–16 years (mean age 13.50 years) in collaboration with the World Health Organization (WHO), which is carried out every four years in a network of countries in the WHO European Zone and North America. The same standardized protocol is used in all countries, facilitating cross-national comparisons (Inchley et al., 2018). **Chapter 6** additionally comprises country-level data on national-level social mobility, which was computed based on the European Social Survey (ESS) (ESS Data Team, 2021), as well as data on gross national income (GNI) from the World Bank DataBank (World Bank, 2021), and income inequality, as determined by the GINI index for disposable (post-tax, post-transfer) income, from the 9th version of the Standardized World Income Inequality Database (Solt, 2019).



CHAPTER 2

EDUCATIONAL LEVEL AND ALCOHOL USE IN ADOLESCENCE AND EARLY ADULTHOOD – THE ROLE OF SOCIAL CAUSATION AND HEALTH-RELATED SELECTION – THE TRAILS STUDY

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ABSTRACT

Both social causation and health-related selection may influence educational gradients in alcohol use in adolescence and young adulthood. The social causation theory implies that the social environment (e.g., at school) influences adolescents' drinking behaviour. Conversely, the health-related selection hypothesis posits that alcohol use (along other health-related characteristics) predicts lower educational attainment. From past studies it is unclear which of these mechanisms predominates, as drinking may be both a cause and consequence of low educational attainment. Furthermore, educational gradients in alcohol use may reflect the impact of 'third variables' already present in childhood, such as parental socioeconomic status (SES), effortful control, and IQ. We investigated social causation and health-related selection in the development of educational gradients in alcohol use from adolescence to young adulthood in a selective educational system. We used data from a Dutch population-based cohort (TRAILS study; N = 2,229), including measurements of educational level and drinking at ages around 14, 16, 19, 22, and 26 years (waves 2 to 6). First, we evaluated the directionality in longitudinal associations between education and drinking with cross-lagged panel models, with and without adjusting for pre-existing individual differences using fixed effects. Second, we assessed the role of childhood characteristics around age 11 (wave 1), i.e., IQ, effortful control, and parental SES, both as confounders in these associations, and as predictors of educational level and drinking around age 14 (wave 2). In fixed effects models, lower education around age 14 predicted increases in drinking around 16. From age 19 onward, we found a tendency towards opposite associations, with higher education predicting increases in alcohol use. Alcohol use was not associated with subsequent changes in education. Childhood characteristics strongly predicted education around age 14 and, to a lesser extent, early drinking. We mainly found evidence for the social causation theory in early adolescence, when lower education predicted increases in subsequent alcohol use. We found no evidence in support of the health-related selection hypothesis with respect to alcohol use. By determining initial educational level, childhood characteristics also predict subsequent trajectories in alcohol use.

1. INTRODUCTION

Lower socioeconomic status (SES) has been associated with increased alcohol-related morbidity in adulthood (Katikireddi, Whitley, et al., 2017; Mackenbach et al., 2015). To understand the mechanisms by which SES and alcohol-related outcomes become associated, it is important to focus on adolescence and young adulthood, as this is when alcohol use is initiated, and youngsters can affect their own later SES through education (Bosque-Prous et al., 2017). Indeed, many studies have shown that alcohol use is associated with lower adolescent educational attainment in the selective educational systems common in Western Europe. These educational systems are characterized by an early selection into different classrooms – and hereby different social contexts – based on academic aptitude. Subsequently, a proportion of students is mobile mostly between adjacent educational tracks (van Spijker et al., 2017). In a Dutch sample of 12 to 16-year-olds, the prevalence of past month alcohol use was 31.1% in the lower vocational track, whilst it was only 12.2% in the academic track (de Looze et al., 2017). Similar results have been found in other countries with selective educational systems, such as Belgium, Austria, and Germany (Berten et al., 2012; Berten et al., 2013). However, the mechanisms by which educational trajectories and drinking behaviours become associated remain poorly understood (Mackenbach, 2012, 2019).

Two mechanisms may explain educational gradients in alcohol use: social causation and health-related selection (Elstad, 2010; Mackenbach, 2012, 2019). The social causation theory implies that the social environment (e.g., at school) predicts adolescents' drinking behaviour (Mackenbach, 2012). For example, educational tracks may differ in terms of future expectations and alcohol-related norms. Students in the lower tracks may more frequently experience feelings of futility, have poor future prospects and low self-esteem, and hence may turn to risk behaviours as means to gain recognition amongst peers (Berten et al., 2012; Elstad, 2010; Van Houtte & Stevens, 2008). Conversely, the health-related selection hypothesis posits that poor health behaviours, such as an early onset of drinking, predict lower academic achievement and may lead to downward or impair upward social mobility in the educational system (Mackenbach, 2012). For example, early onset heavy alcohol use has been associated with cognitive impairments in adolescents, and may hereby negatively affect performance at school (Nguyen-Louie et al., 2017; Peeters et al., 2014). Importantly, social causation and health-related selection are not mutually exclusive, may reinforce each other over time, but differ in their relative importance in different phases of adolescence.

In addition, associations between educational level and alcohol use may reflect the impact of 'third variables' (i.e., confounders) already present in childhood (Davies et al., 2017; Mackenbach, 2012). On the individual level, differences in psychological dispositions may impact both initial selection into educational tracks and later substance use (Mackenbach, 2012). For example, students with high levels of effortful control are more

likely to succeed in educational settings (Veronneau et al., 2014) and may also be more likely to abstain from substance use (Peeters et al., 2017). Similarly, good cognitive functioning is related to both higher educational attainment and better health behaviours later in life (Brody, 1997; Kubička et al., 2001). On the social (environmental) level, characteristics of the family environment may influence both educational prospects and alcohol use. Adolescents from lower SES families may more frequently be exposed to harmful drinking at home (Mackenbach et al., 2015; Pape et al., 2017), and lower SES families may also have less resources to support the education of their children (van Spijker et al., 2017). These mechanisms may be referred to as 'indirect' social causation or health-related selection, depending on whether emphasis is given to the social-environmental or individual-level factors (Elstad, 2010; Mackenbach, 2012, 2019). Like the 'direct' mechanisms, the 'indirect' mechanisms can work in conjunction. For example, higher SES parents may be able to provide a safer and more stimulating family environment, positively influencing their young child's emotional and cognitive development (Ng-Knight & Schoon, 2017; Rosen et al., 2018). Resulting differences in psychological dispositions can then predict both later educational attainment and health behaviours.

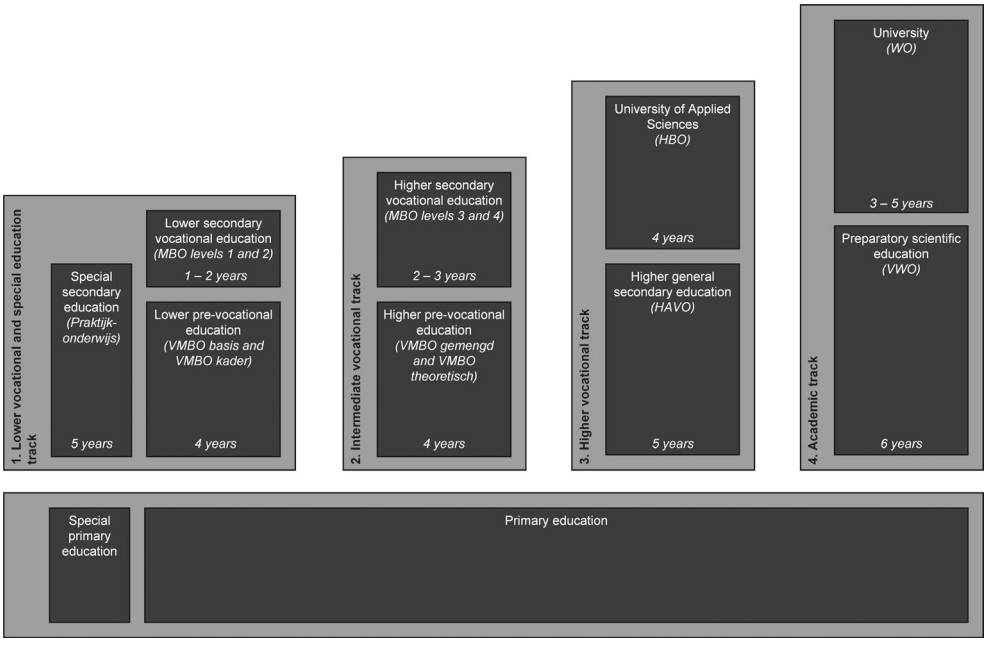
Only few studies have investigated the temporal directions of associations between alcohol use and education throughout adolescence (Crosnoe, 2006; Latvala et al., 2014; Owens et al., 2008). One method to study these is by modelling autoregressive and cross-lagged associations simultaneously in cross-lagged panel models (CLPMs). Overall, results were mixed and do not provide conclusive evidence for the dominance of either the 'direct' social causation or the 'direct' health-related selection hypothesis. In the USA, Crosnoe (2006) mainly found evidence for social causation, as academic failure predicted more subsequent drinking in 16-year-old adolescents, but not vice versa. Conversely, Latvala et al. (2014) mainly found evidence for health-related selection in Finnish early and mid-adolescents. Higher alcohol use around age 12 predicted lower GPA around age 14, and higher alcohol use around age 14 was associated with a lower likelihood of being in education around age 16. Latvala et al. (2014) found no evidence for social causation effects throughout adolescence, except for one significant path from higher education around age 17 to increased alcohol use around age 24. Similar to Latvala and colleagues (2014), Owens et al. (2008) found health-related selection effects, with alcohol use predicting lower GPA one year later from around age 14 through around age 18 in a US sample. Social causation results were less consistent and revealed mixed findings, with higher GPA predicting less alcohol use in younger and more alcohol use in older adolescents.

This heterogeneity in results may partially relate to the fact that CLPMs cannot separate the within and between-person variances of the cross-lagged variables. Therefore, they are unable to rule out the possibility of confounding by unmeasured time-invariant (or trait-like) 'third variables' often already present prior to adolescence (Hamaker et al., 2015). These variables may include relatively stable genetic or temperamental factors, which are both associated with health behaviours and educational outcomes, as mentioned above (Mackenbach, 2012). Unmeasured heterogeneity affecting reciprocal associations can

be addressed with novel statistical methods, which combine CLPMs with fixed effects methods, allowing to assess bidirectional associations between education and alcohol use at the within-person level (Allison et al., 2017).

In addition, all past studies were from comprehensive educational systems and results may not be applicable to the selective educational systems common in Western Europe. In the Netherlands, this selection into one of four tracks (**Figure 2.1**) takes place at age 11-12, based on a large battery of cognitive tests and the advice of the teacher in primary education (van Spijker et al., 2017). The Dutch system may allow assessing social mobility in adolescents at an earlier age than possible in comprehensive educational systems, as social stratification already occurs around the beginning of adolescence.

FIGURE 2.1 The Dutch educational system



In the current study, we aimed to contribute to the literature by addressing the following research question: "To what extent do the social causation and health-related selection hypotheses explain educational differences in alcohol use in adolescents and young adults in a selective educational system?" To answer our research question, we modelled reciprocal relationships (i.e., 'direct' social causation and health-related selection) between alcohol use and educational level throughout adolescence and young adulthood. Furthermore, we aimed to evaluate the role of family SES, as well as childhood effortful control and IQ, which may all act both as determinants of the initial selection into educational tracks and

early alcohol use, and as confounders ('third variables') in the cross-lagged paths. Finally, we addressed potential residual confounding by unmeasured relatively stable 'third variables' in bidirectional associations, using a fixed effects approach.

Based on past findings highlighting the strong role of the social environment in adolescent drinking (Berten et al., 2012; Elstad, 2010; Van Houtte & Stevens, 2008), as well as the deleterious effect of intensive alcohol use on cognitive performance (Nguyen-Louie et al., 2017; Peeters et al., 2014), we expected to find evidence for both mechanisms in CLPMs (i.e., lower education predicting increases in alcohol use and vice versa). We also expected an attenuation in cross-lagged associations after adjusting for childhood characteristics, in line with the 'indirect' social causation and health-related selection hypotheses (Mackenbach, 2012). During the transition from childhood to early adolescence, we expected to find associations between childhood psychological characteristics (i.e., IQ and effortful control) and the educational track in which adolescents were initially placed, in line with the health-related selection hypothesis. Finally, we expected to find associations between higher parental SES and higher educational level, as well as lower alcohol use, in early adolescence, in line with the social causation theory.

2. MATERIALS AND METHODS

2.1. Study population

We used data from the first six waves (T1 – T6) of the TRacking Adolescents' Individual Lives Survey (TRAILS), a population-based prospective cohort study of Dutch adolescents. A detailed description of the cohort can be obtained elsewhere (Oldehinkel et al., 2015). At the beginning of the study, 135 schools in the province of Groningen were invited, of which 122 decided to participate (de Winter et al., 2005). Adolescents were followed between 2000 and 2017 with assessments around age 11, 14, 16, 19, 22, and 26. Ethical approval for TRAILS was obtained from the Dutch national ethics committee Central Committee on Research Involving Human Subjects (#NL38237.042.11). Written informed consent was obtained from both adolescents and their parents prior to inclusion in the study.

2.2. Alcohol use

Alcohol use was assessed contemporaneously from waves 2 through 5 by self-report using a quantity-frequency measure (Sobell & Sobell, 1995). Quantity-frequency measures of alcohol use have shown adequate/good validity and reliability across studies (McKenna et al., 2018). Frequency was assessed by asking adolescents about the number of weekdays (Monday to Thursday) and weekend days (Friday to Sunday) on which alcohol was consumed. Quantity was measured by asking about the average number of alcoholic beverages consumed on a typical week or weekend day (9-point scale ranging from 1='I

never drink on a weekday/ weekend day' to '11 glasses or more'). A quantity-frequency measure was obtained by multiplying the quantity scores for week and weekend days by the corresponding frequency scores, and then summing both scores (Sobell & Sobell, 1995). At wave 6, alcohol use was assessed by self-report using the sum score of the AUDIT-C, which consists of the first three items of the Alcohol Use Disorders Identification Test (AUDIT) (Bush et al., 1998). The AUDIT-C (Cronbach's alpha = 0.66) includes each one item assessing frequency (number of drinking occasions in past 12 months; range: never to ≥ 4 per week), quantity (number of glasses per typical drinking occasion; range: 1 – 2 to ≥ 10), as well as a measure of binge drinking (number of occasions where ≥ 6 glasses of alcohol are consumed; range: never to daily or almost daily). Both measures were z-score transformed before inclusion in our analyses. It was not possible to compute alcohol use scores for a number of participants who had missing information for at least either quantity or frequency items (waves 2 – 5), or at least one item of the AUDIT-C (wave 6): wave 2: N = 190, 8.85%; wave 3: N = 249, 13.70%; wave 4: N = 263, 13.99%; wave 5: N = 315, 17.69%; wave 6: N = 438; 27.10%.

2.3. Adolescents' educational level

The Dutch educational system is characterized by an early (age 11-12) selection into a particular educational track, based on a battery of cognitive tests and the advice of the primary school. In line with this selection, we used a measure of educational level that is consistent throughout all of secondary and tertiary education (**Figure 2.1**). There are four tracks in the Dutch educational system, each consisting of a specific type of secondary school followed by tertiary education at the corresponding level: 1. lower vocational track, 2. intermediate vocational track, 3. higher vocational track, 4. academic track. In addition, there is a special education track, attended by students who are unable to attend regular education. This track was collapsed with the lower vocational track. Educational track membership was assessed from wave 2 to 6 by asking for participants' current enrolment, as well as their highest completed diploma. Participants who finished the final diploma of a given track received the value corresponding to that level for all subsequent waves, unless they continued education at a higher level. Educational level was not assessed at wave 1, since most children were still in elementary school. Our measure of educational level allows us to assign a score that represents an age-appropriate measure of educational attainment as proxy of developing SES.

Missing information on educational track membership from waves 2 through 6 was filled in using retrospective event history calendars conducted at wave 3 and wave 5. Participants who were still in elementary education or in a combined class at wave 2 were assigned according to their elementary school teachers' recommended level. If this information was not available, pupils were classified according to the first track they attended after leaving elementary education or the combined class. It was not possible to classify a number of

participants, who were not in education for a longer period, were not classifiable into an educational track (e.g., because of education abroad), whose educational level was assessed incompletely, who did not respond to questions on education, or who had permanently left the educational system (wave 2: N = 221, 10.29%; wave 3: N = 289, 15.90%; wave 4: N = 373, 19.84%; wave 5: N = 352, 19.76%; wave 6: N = 424, 26.24%). Educational level was set to missing for these participants.

2.4. Characteristics at baseline (wave 1)

Characteristics at baseline hypothesized to be associated with both alcohol use and education were selected based on earlier studies (Bosque-Prous et al., 2017; Davies et al., 2017) and include:

1. *Parents' socioeconomic status (SES)*, constructed as the mean score of five indicators (standardized): maternal and paternal educational attainment, maternal and paternal occupational position (according to the International Standard Classification of Occupations), and family income (Amone-P'Olak et al., 2009).
2. *Childhood psychological characteristics* include effortful control and the Intelligence Quotient (IQ). Effortful control was assessed using the corresponding subscale from the parent-report Early Adolescent Temperament Questionnaire (EATQ-R), which consists of 11 items with 5 response categories (Cronbach's alpha = 0.86; McDonald's omega = 0.87 (Oldehinkel et al., 2004; Shaw, 2021)). Children's IQ was estimated using the Block Design and Vocabulary subtests of the Revised Wechsler Intelligence Scale for Children (WISC-R) (Brunnekreef et al., 2007).
3. Furthermore, we adjusted for demographic characteristics at baseline, that is, area of residence (City of Groningen, Leeuwarden, Assen, other regions), adolescent age, gender, and ethnicity. Children were classified as having non-Dutch ethnicity if at least one of their parents was born outside the Netherlands (Vollebergh et al., 2005).

2.5. Analytic approach

First, we computed descriptive statistics of the study population by cross-tabulating baseline characteristics (mean age 11) with early adolescent educational track membership at wave 2 (mean age 14), as well as alcohol use with concurrent educational level from wave 2 through wave 6 (mean age 26). Second, we computed cross-lagged panel models (CLPMs) between educational level and alcohol use from wave 2 through wave 6, whilst sequentially adjusting for different sets of baseline covariates. Our full covariate-adjusted CLPM included age, gender, area of residence, ethnicity, parental socioeconomic status, IQ, and effortful control at baseline (wave 1) as predictors. The CLPM estimates prospective associations between educational level and changes in subsequent alcohol use, and between alcohol use and changes in subsequent educational level, whilst taking into account temporal stability and reciprocity (**Figures 2.S1 and 2.S2**) (Allison et al., 2017). Third, we conducted

analogous CLPMs with fixed effects. These models only use within-person variance to estimate associations between cross-lagged variables, hereby adjusting for all measured and unmeasured time-invariant characteristics. In line with the one-sided specification by Allison et al. (2017), two separate fixed effects models were fit to assess lagged associations from education to changes in subsequent alcohol use, and from alcohol use to changes in subsequent education (**Figure 2.S3**). The fixed effects term was represented by a latent variable of all measurements of the outcome with each having its factor loading constrained to be 1. This latent variable was allowed to be correlated freely with all time-varying exogenous variables in the model. Reciprocal causation was accommodated by including correlations between the error term of the outcome at each measurement occasion and all future values of the time-varying exposure. Fourth, we evaluated the role of parental SES and adolescents' psychological characteristics at baseline (i.e., IQ, effortful control) in predicting alcohol use and educational level at wave 2 in the full covariate-adjusted CLPM. Finally, we computed intra-class correlations (ICC) for education and alcohol use over time, to assess which proportions of the variance were at the within-person and at the between-person level.

Attrition analyses showed that at wave 2 3.63% (N = 81) of the original participants no longer participated in the study. At wave 3 this was the case for 18.44% (N = 411), at wave 4 for 15.66% (N = 349), at wave 5 for 20.10% (N = 448), and at wave 6 for 27.50 (N = 613) of the original participants. Adolescents with male gender, non-Dutch ethnicity, lower educational level, IQ, and effortful control, as well as those from lower SES households were more likely to drop out of the study (**Table 2.S1**). Higher alcohol use was also related to dropout, but only significantly at wave 3. Similar differences were found when comparing participants with complete information on educational level to those whose educational level was missing or could not be classified (**2.S2 Table**). To deal with missing information, full-information maximum likelihood (FIML) was implemented, allowing to incorporate information from all participants. All variables that were not part of each respective analytic model were included as auxiliary variables to adjust for potential bias due to missing data on these variables (Asparouhov & Muthén, 2008). Model fit in Structural Equation Models (SEM) was assessed using the Comparative Fit Index (CFI), the Tucker Lewis Index (TLI), the Root Mean Square Error of Approximation (RMSEA), and the Standardised Root Mean Residual (SRMR). Following the suggestions by Hu and Bentler (1999), model fit was judged as 'good' if the CFI and TLI were >0.95, the RMSEA was <0.06, and the SRMR was <0.08. Standard errors were estimated using robust maximum likelihood (MLR) to take into account potential normality violations. Analyses were conducted in Mplus 8.6.

2.6. Sensitivity analyses

To determine whether the fact that a different indicator of alcohol use was used at wave 6 (i.e., the AUDIT-C) influenced our results, we conducted a sensitivity analysis in which

the binge drinking item was removed from the AUDIT-C, creating a quantity-frequency measure that is somewhat similar to the one used from wave 2 to 5. Additionally, we assessed whether the ordinal nature of our educational variable affected the linear regression results by executing the bivariate CLPM and the fixed effects models using the Bayes estimator in Mplus, whilst declaring all endogenous measurements of educational level as "categorical". Overall, the results of the sensitivity analyses were similar to our main results.

3. RESULTS

3.1. Descriptive statistics

Table 2.1 shows the characteristics of TRAILS participants around age 11 according to educational level around age 14. Children with less affluent or non-Dutch parents more commonly attended the lower educational tracks. Girls more frequently attended the academic and intermediate vocational tracks than boys. Children in the lower vocational track and the academic track were slightly older at baseline than those in the intermediate and higher vocational tracks. Further, higher IQ and higher effortful control around age 11 predicted higher education around age 14.

Table 2.2 shows educational level around age 14, 16, 19, 22, and 26, and concurrent alcohol use. Around age 14 and 16, we found an educational gradient in alcohol use, with adolescents in the lower tracks consuming more alcohol compared to those in the higher tracks. No general gradient was seen around age 19, and around age 21 young adults in the intermediate vocational track consumed less alcohol than those in the other three tracks. By age 26, the educational gradient in alcohol use was reverse, with young adults in the higher tracks scoring higher on the AUDIT-C.

TABLE 2.1 Characteristics of adolescents participating in the TRAILS study (the Netherlands, 2000–2017, N = 2,229) at wave 1 (2000-2002) and according to educational level at wave 2 (2003-2005)

	All levels		**Lower vocational & special education**		**Intermediate vocational**		**Higher vocational**		**Academic**	
	N = 2,229		**N = 635**		**N = 497**		**N = 383**		**N = 457**	
Male gender, N (%)	1,098	(49.26)	341	(53.70)	217	(43.66)	196	(51.17)	195	(42.67)
District, N (%)										
City of Groningen	794	(35.62)	227	(35.75)	157	(31.59)	128	(33.42)	197	(43.11)
Leeuwarden	596	(26.74)	193	(30.39)	125	(25.15)	84	(21.93)	124	(27.13)
Assen	489	(21.94)	127	(20.00)	98	(19.72)	124	(32.38)	102	(22.32)
Other regions	350	(15.70)	88	(13.86)	117	(23.54)	47	(12.27)	34	(7.44)
Non-Dutch ethnicity, N (%)	301	(13.50)	108	(17.01)	61	(12.27)	39	(10.18)	45	(9.85)
Age, mean (SD)	11.11	(0.56)	11.16	(0.56)	11.07	(0.54)	11.05	(0.56)	11.14	(0.56)
Parental socioeconomic status (SES), mean (SD)	-0.05	(0.80)	-0.53	(0.70)	-0.16	(0.67)	0.21	(0.68)	0.55	(0.70)
Wechsler Intelligence Deviation Quotient (IQ), mean (SD)	97.19	(15.00)	86.05	(12.49)	95.20	(10.98)	102.68	(11.20)	111.14	(11.91)
Effortful control, mean (SD)	3.23	(0.68)	2.92	(0.62)	3.06	(0.63)	3.35	(0.65)	3.65	(0.61)

SD = standard deviation.

3.2. Cross-lagged associations between educational level and alcohol use

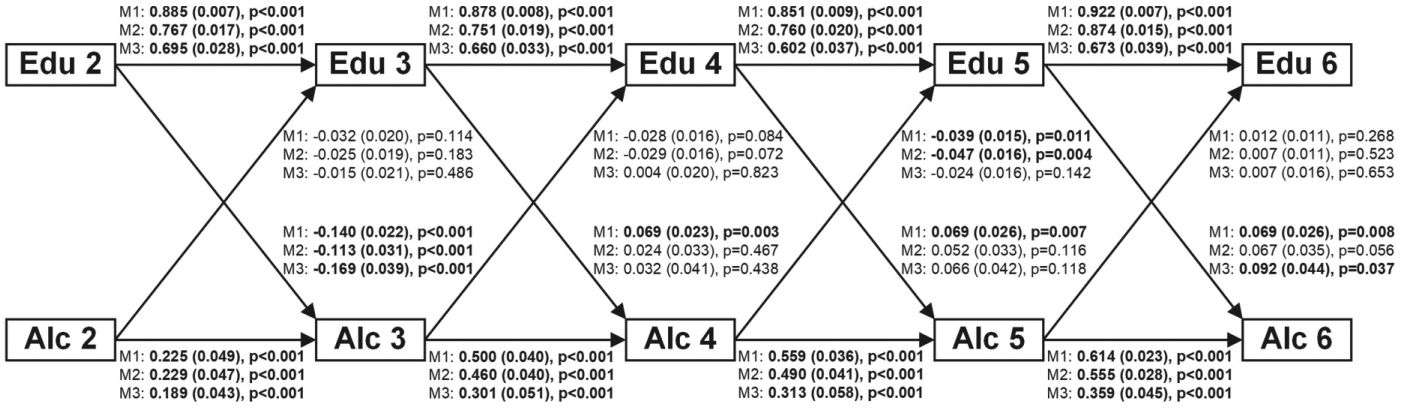
In **Figure 2.2**, we assessed the 'direct' social causation and health-related selection hypotheses by evaluating bidirectional associations between educational level and alcohol use from age 14 to 26, using CLPMs. In bivariate CLPMs (**Figure 2.2**, Model 1), educational level exhibited very high stability (ß > 0.80), while the stability of alcohol use was lower and increased over time, ranging from approximately 0.20 in early adolescence to 0.60 in young adulthood. The ICC for education was 0.820, which indicates that 82% of the variance across the five measurements of educational level was due to differences between persons. This finding is in line with the high stability in educational level we found in CLPMs. The ICC for alcohol use was 0.293, suggesting that a substantial proportion of variance in alcohol use represents within-person fluctuations over time.

TABLE 2.2 Alcohol use of adolescents and young adults participating in the TRAILS study (the Netherlands, 2000–2017, N = 2,229) according to concurrent educational level

	Wave 2		**Wave 3**		**Wave 4**		**Wave 5**		**Wave 6**	
	N = 2,148		**N = 1,818**		**N = 1,880**		**N = 1,781**		**N = 1,616**	
Date range	2003-2005		2005-2008		2008-2010		2012-2014		2016-2017	
Age, mean (SD)	13.57	(0.53)	16.28	(0.71)	19.08	(0.60)	22.29	(0.65)	25.66	(0.60)
Male gender, N (%)	1,054	(49.07)	867	(47.69)	898	(47.77)	843	(47.33)	735	(45.48)
Educational level, N (%)										
Lower vocational & special education	635	(32.20)	349	(22.83)	161	(10.68)	136	(9.52)	78	(6.54)
Intermediate vocational	497	(25.20)	405	(26.49)	498	(33.02)	354	(24.77)	273	(22.90)
Higher vocational	383	(19.42)	362	(23.68)	475	(31.50)	594	(41.57)	489	(41.02)
Academic	457	(23.17)	413	(27.01)	374	(24.80)	345	(24.14)	352	(29.53)
Alcohol use										
Quantity-frequency score, mean (SD)										
All levels	1.64	(4.56)	6.95	(9.56)	10.18	(11.64)	10.18	(11.01)	-	-
Lower vocational & special education	2.20	(6.12)	9.70	(13.52)	11.12	(15.48)	11.35	(15.78)	-	-
Intermediate vocational	1.76	(3.73)	6.74	(8.29)	9.47	(9.97)	8.56	(8.96)	-	-
Higher vocational	1.58	(4.14)	5.87	(8.81)	10.61	(11.50)	10.70	(11.08)	-	-
Academic	0.84	(3.45)	4.69	(5.05)	9.46	(10.46)	10.69	(10.98)	-	-
AUDIT-C score, mean (SD)										
All levels	-	-	-	-	-	-	-	-	4.60	(2.41)
Lower vocational & special education	-	-	-	-	-	-	-	-	3.81	(2.42)
Intermediate vocational	-	-	-	-	-	-	-	-	4.23	(2.43)
Higher vocational	-	-	-	-	-	-	-	-	4.55	(2.33)
Academic	-	-	-	-	-	-	-	-	4.92	(2.30)

SD = standard deviation.

FIGURE 2.2 Bidirectional associations between educational level and alcohol use in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) from cross-lagged panel models without (Model 1 and 2) and with fixed effects (Model 3)



	CFI	TLI	SRMR	RMSEA
Model 1	0.984	0.969	0.032	0.045
Model 2	0.994	0.967	0.014	0.033
Model 3				
a) Education → Alcohol use	0.999	0.996	0.009	0.016
b) Alcohol use → Education	0.997	0.989	0.007	0.030

Model 1: bivariate cross-lagged panel model. Model 2: cross-lagged panel model adjusted for age, gender, area of residence, ethnicity, parental socioeconomic status, IQ, and effortful control at baseline (wave 1). Model 3: cross-lagged panel models with fixed effects–adjustment for time-invariant characteristics was performed by inclusion of a latent variable. Edu = educational level; Alc = alcohol use. ***Boldface*** *denotes statistical significance at p < 0.05.*

When considering social causation paths from educational level to alcohol use, lower education around age 14 predicted increases in alcohol use around age 16 (ß coefficient -0.140, Standard Error [SE] 0.022, p<0.001). Conversely, from age 16 onwards we found consistent, though relatively weaker, associations between higher education and increases in subsequent alcohol use (from around age 16 to 19: ß 0.069, SE 0.023, p=0.003; from around age 19 to 22: ß 0.069, SE 0.026, p=0.007; from around age 22 to 26: ß 0.069, SE 0.026, p=0.008). In multivariate CLPMs, adjustment for parental SES led to the greatest change in the model (**Figure 2.S4**, Model 3), rendering associations between education around 16 and increases in alcohol use around 19 (ß 0.033, SE 0.026, p=0.198), as well as between education around 22 and increases in alcohol use around 26 (ß 0.035, SE 0.029, p=0.233) insignificant. In the full covariate-adjusted model (**Figure 2.2**, Model 2), only the association between lower education around age 14 and increases in alcohol use around age 16 remained statistically significant (ß -0.113, SE 0.031, p<0.001). Results from the fixed effects model (**Figure 2.2**, Model 3a) somewhat resembled the full covariate-adjusted model (**Figure 2.2**, Model 2). Lower education around age 14 significantly predicted increases in alcohol use around 16 (ß -0.169, SE 0.039, p<0.001). From age 19 to 26 we found tendencies towards opposite associations, though only the association between higher education around age 22 and increases in alcohol use around age 26 was once more significant in the fixed effects model (ß 0.092, SE 0.044, p=0.037). However, in our sensitivity analysis (**Figure 2.S5**) this association failed to reach significance both in the fixed effects model (ß 0.073, SE 0.045, p=0.106) and in the bivariate CLPM (ß 0.044, SE 0.028, p=0.114) once the binge drinking item was removed from the AUDIT-C. Besides this, our sensitivity analysis yielded very similar results to the main analysis.

When considering health-related selection related to alcohol use in the bivariate CLPM (**Figure 2.2**, Model 1), we found a weak prospective association between higher alcohol use and decreases in subsequent educational level from around age 19 to 22 (ß -0.039, SE 0.015, p=0.011). This result was robust to adjustment for covariates (ß -0.047, SE 0.016, p=0.004) (**Figure 2.2**, Model 2), but did not survive in the fixed effects model (ß -0.024, SE 0.016, p=0.142) (**Figure 2.2**, Model 3b). In our sensitivity analysis with the Bayes estimator (**Figure 2.S6**), in which educational level was declared as 'categorical', we additionally found a significant path from higher alcohol use around age 14 to decreases in education around age 16 in the bivariate CLPM (ß -0.039, posterior SD 0.014, one-tailed p=0.002). However, similar to our main result, this effect was absent in the fixed effects model (ß -0.011, posterior SD 0.016, one-tailed p=0.255). Model fit of all cross-lagged models was good (Hu & Bentler, 1999).

3.3. Associations of childhood predictors with educational level and alcohol use in early adolescence

In **Table 2.3**, we evaluated the role of childhood (around age 11) psychological characteristics (i.e., health-related selection) and parental SES (i.e., social causation) as predictors of initial educational level (around age 14) following adolescents' selection into educational tracks, in the full covariate-adjusted CLPM shown in **Figure 2.2**, Model 2. Furthermore, we evaluated the role of parental SES (i.e., social causation) and childhood psychological characteristics in early adolescent alcohol use (around age 14). The associations of the childhood predictors with educational level and alcohol use from wave 3 to 6 are shown in **Table 2.S3**.

Higher parental SES (ß 0.280, SE 0.017, p<0.001), IQ (ß 0.462, SE 0.016, p<0.001), and effortful control (ß 0.249, SE 0.017, p<0.001) significantly predicted higher educational level around age 14. Higher parental SES (ß -0.086, SE 0.021, p<0.001) and IQ (ß -0.052, SE 0.026, p=0.045), but not effortful control (ß -0.042, SE 0.024, p=0.079), also independently predicted lower alcohol use around age 14, although to a lesser extent. In addition, we conducted a post-hoc analysis to assess whether associations of baseline IQ and parental SES with alcohol use around age 14 would remain significant after further adjusting for adolescents' concurrent educational level (**Table 2.S4**). While the association of parental SES with drinking was not impacted (ß -0.079, SE 0.024, p=0.001), baseline IQ was no longer significantly associated with drinking around age 14 in this model (ß -0.041, SE 0.034, p=0.222).

TABLE 2.3 The association between baseline characteristics (wave 1) and educational level and alcohol use at wave 2 in the TRAILS study (the Netherlands, 2000–2017, N = 2,229) in the multivariate-adjusted cross-lagged panel model (model 2) in Figure 2.2; linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value); all predictors are mutually adjusted

	Educational level	**Alcohol quantity-frequency score**
Male gender	-0.029 (0.015), p=0.060	-0.024 (0.022), p=0.267
District		
City of Groningen	ref	ref
Leeuwarden	**-0.037 (0.017), p=0.034**	0.004 (0.027), p=0.896
Assen	**-0.039 (0.019), p=0.046**	**-0.052 (0.027), p=0.049**
Other regions	**-0.057 (0.019), p=0.003**	-0.008 (0.029), p=0.767
Non-Dutch ethnicity	0.009 (0.016), p=0.570	0.000 (0.030), p=0.995
Age	0.001 (0.018), p=0.949	0.002 (0.029), p=0.956
Parental socioeconomic status (SES)	**0.280 (0.017), p<0.001**	**-0.086 (0.021), p<0.001**
Wechsler Intelligence Deviation Quotient (IQ)	**0.462 (0.016), p<0.001**	**-0.052 (0.026), p=0.045**
Effortful control	**0.249 (0.017), p<0.001**	-0.042 (0.024), p=0.079

All predictors are mutually adjusted.
***Boldface** denotes statistical significance at p < 0.05.*

4. DISCUSSION

Investigating bidirectional associations between alcohol use and educational level in adolescence and young adulthood, we mainly found evidence in favour of the 'direct' social causation hypothesis in early adolescence, with lower educational level around age 14 strongly predicting subsequent increases in alcohol use around age 16. From age 19 onward, we found tendencies towards opposite associations, with higher education predicting increases in alcohol use. However, these associations failed to reach significance in the adjusted models, except for the path from higher education around age 22 to increases in alcohol use around age 26 in the fixed effects model when using the full AUDIT-C at wave 6. Also, a small health-related selection effect from higher alcohol use to lower subsequent education between age 19 and 22 was no longer significant after adjusting for unmeasured stable differences. The lack of significant cross-lagged associations throughout most of adolescence in adjusted models points to the importance of relatively stable 'third variables' associated with both education and alcohol use. Furthermore, educational track membership showed very high stability, suggesting that the potential for health-related selection later in adolescence may be limited in educational systems with early selection into educational tracks. It is therefore important to also consider the role of childhood predictors, which determine this initial selection. Indeed, we found that parental SES, as well as IQ and temperamental effortful control in childhood, strongly predicted children's educational level in early adolescence. Parental SES and IQ also predicted early alcohol use, although to a lesser extent.

4.1. Strengths and limitations

Our study has several limitations. First, attrition and missing data may have affected our results. While we addressed missing data with FIML (Peeters et al., 2019), more drop-out and missingness in participants with lower educational level, higher alcohol use, and less favourable psychological dispositions (**Tables 2.S1 and 2.S2**) may still have influenced our results. Studies suggest that low IQ, effortful control, and parental SES are important predictors of adverse outcomes in young adulthood (Caspi et al., 2016). Future research on at-risk groups is therefore essential. Second, TRAILS used a different indicator for alcohol use at wave 6 (i.e., the AUDIT-C) compared to the other waves, which additionally includes an item assessing binge drinking. However, almost similar results were found when the binge drinking item was removed from the AUDIT-C (**Figure 2.S5**). Third, our models do not address time-varying confounding. Certain characteristics that change substantially throughout adolescence, such as delinquency, may both be causally related to educational level and alcohol use, and may confound cross-lagged associations (Moffitt, 1993). Future research could investigate the contribution of such influences in relation to health-related selection and social causation effects. Fourth, by design, our study may

not have captured the whole range of effects of alcohol use on educational attainment. For example, alcohol use may still have adversely affected adolescents' GPA (Balsa et al., 2011; Latvala et al., 2014), but these effects may not have been consequential enough to lead to a decline in educational track. Likewise, it may still be possible that adolescents experience downward educational mobility in case of severe alcohol-related problems, e.g., repeated hospitalization with acute intoxication (Van Hoof et al., 2018), or polysubstance use (Vergunst et al., 2021). Our quantity-frequency measure might not have adequately captured severe forms of alcohol use or the use of alcohol in combination with other substances (i.e., polysubstance use). Further research on the educational consequences of severe alcohol-related problems and polysubstance use in adolescence is warranted. Fifth, the social meaning of alcohol use might change over time, which could lead to longitudinal measurement non-invariance. However, recent research suggests that alcohol use measures are quite reliable and consistent in measuring alcohol use during adolescence and young adulthood (Fish et al., 2017; Peeters et al., 2019). Sixth, whilst CLPMs account for the longitudinal structure of our data regarding the order of measurement occasions, they assume equal distances between waves. If the time intervals between measurement occasions are strongly unequal, CLPMs may yield biased results (Kuiper & Ryan, 2018). There were some differences in the spacing between the waves in TRAILS, which might be difficult to avoid in studies covering very long time periods (between wave 2 and 3 on average 2.71 years; between wave 3 and 4 on average 2.80 years; between wave 4 and 5 on average 3.21 years, and between wave 5 and 6 on average 3.37 years). While this might have introduced minor bias in the regression coefficients, we do not expect that this should have led to major changes in the relationships we found. Some waves started a bit later than others, and some adolescents participated in the one wave a bit later and in the following wave a bit earlier, but overall, the differences in the spacing between waves were small and on average only concerned several months.

Finally, limitations relate to the generalizability of our findings. The TRAILS participants were likely to start drinking during times when the Netherlands was at the top of international rankings of alcohol consumption amongst 12-16-year-olds. Dutch parents have since adopted more restrictive alcohol-related parenting practices (de Looze et al., 2017). Higher socioeconomic and educational groups tend to be faster at adopting behavioural innovations, which often leads to a (temporary) widening in inequalities in health behaviours (de Looze et al., 2013a; Mackenbach, 2012). In the Netherlands, differences in the prevalence of early adolescents´ past month drunkenness between the lowest and the highest educational tracks have increased from 4.6% in 2003 to 9.8% in 2015 (de Looze et al., 2017). Therefore, we hypothesize that associations might be stronger in more recent Dutch cohorts than in TRAILS. Generalizability of our findings to other geographical contexts may also be limited, because both educational systems and adolescents' drinking cultures vary widely across countries (de Looze et al., 2017; ESPAD Group, 2015).

The long follow-up and high response rate are key strengths of our study. By using CLPMs, we take into account reciprocity between educational level and alcohol use and disentangle their temporal direction (Allison et al., 2017). By comparing multivariate adjusted CLPMs to CLPMs with fixed effects, we adjust for both measured and unmeasured time-invariant confounding (Allison et al., 2017; Latvala et al., 2014). We add to the literature by for the first time modelling bidirectional associations between educational level and alcohol use in a selective educational system, which provides a consistent and age-appropriate measure of educational attainment, as proxy for developing SES, over the course of adolescence. The selection into educational tracks at an age as early as 11-12 years means that Dutch adolescents grow up in distinct educational environments that are characterized by different social norms, future expectations, cognitive resources, and occupational prospects (Berten et al., 2012; Van Houtte & Stevens, 2008) – characteristics that are closely related to conceptualizations of SES in adulthood (Mackenbach, 2012). One could therefore argue that in selective educational systems, such as in the Netherlands, youngsters move into 'their own' SES at a much earlier age than in comprehensive systems, such as in Finland or the USA. Therefore, TRAILS provides a unique opportunity to investigate both the antecedents and consequences, in terms of health-related characteristics, of this differentiation and subsequent intragenerational social mobility in adolescents and young adults.

4.2. Interpretation of findings

We found that lower educational level significantly predicted a stronger escalation in alcohol use in early adolescence only, and not later throughout adolescence and young adulthood. This result may point towards an important role for educational differences in peer group composition and social norms in early adolescence, processes which have been found to be amongst the strongest determinants of underage alcohol use (Francois et al., 2017). Previous research has shown that schools in the lower educational tracks more commonly feature a culture characterized by feelings of futility, poor future prospects, and low self-esteem. Consequently, students may turn to alternative means to attain status amongst their peers, which may include substance use (Berten et al., 2012; Elstad, 2010; Van Houtte & Stevens, 2008). Indeed, early adolescents in the lower educational tracks in the Netherlands more frequently perceive substance use as 'adult-like' behaviour (de Looze et al., 2013b), which may be used to gain popularity with drinking peers (Scott et al., 2019). As a result, young adolescents in these tracks may show an earlier escalation in their alcohol use.

From late adolescence onward, we found tendencies towards opposite associations, with higher educational level predicting increases in drinking. However, most of these associations did not survive statistical adjustment, and were partially explained by differences in parental SES (**Figure 2.S4** and **Table 2.S3**). This is in line with previous

research, which found negative correlations between parental SES and underage drinking, and positive correlations with drinking in young adulthood (Pedersen & von Soest, 2013). Changes in social norms within educational tracks as adolescents get older could also have contributed to positive associations between higher education and increases in alcohol use in young adulthood. Adolescents moving into higher education may experience a lifting of constraints on drinking combined with a strong peer pressure towards alcohol use in the context of university culture (Robertson & Tustin, 2018; Verster, 2009). Meanwhile, adolescents who complete the vocational tracks begin fulltime employment and experience earlier transitions to adult work and family roles (Green et al., 2017; Peeters et al., 2019; Staff et al., 2010). They may as a result be less likely to further escalate in their drinking. Past studies from the US and other countries show increased alcohol use amongst young adults in higher education. This 'college effect', however, may only be of temporary nature, as many young adults mature out of heavy drinking sometime after leaving university (Crosnoe & Riegle-Crumb, 2007; Jang et al., 2019; Latvala et al., 2014; Lui et al., 2018; O'Malley & Johnston, 2002; Slutske, 2005).

We found no evidence of health-related selection resulting from alcohol use, as the small lagged association we found in late adolescence did not survive in the fixed effects model. Importantly, we also found no clear evidence for confounding by IQ and effortful control in the CLPMs. The selection effect we found in the bivariate CLPM may therefore be attributable to other time-invariant background variables that we have not assessed, such as differences in parenting practices, personality characteristics, or genetics (Davies et al., 2017).

The absence of significant associations in adjusted CLPMs for most of the study period, in combination with the high stability of educational level over time, highlights the importance of the transition to secondary school in educational systems characterised by early stratification. In line with past studies (Brody, 1997; Veronneau et al., 2014), children's IQ and effortful control strongly predicted into which educational track participants were selected in early adolescence, which subsequently predicted alcohol use in CLPMs. By determining initial educational level, these characteristics also predict subsequent trajectories in alcohol use. This explanation is further supported by the fact that we no longer find a significant association between IQ and early alcohol use once concurrent educational level is added as covariate. Furthermore, our findings highlight the importance of parental SES as determinant of both selection into educational tracks and early alcohol use. Studies from the Netherlands have consistently shown that children from lower SES households more frequently enter lower educational tracks, regardless of their performance on standardized tests (van Spijker et al., 2017). Studies considering associations between parental SES and adolescent alcohol use have been less consistent (Hanson & Chen, 2007; Kwok & Yuan, 2016; Lemstra et al., 2008), but a meta-analysis that specifically focused on early adolescence (age 10-15) found that youngsters from lower

SES households consume more alcohol (Lemstra et al., 2008). In our study, the association between parental SES and early alcohol use remains significant even after further adjusting for adolescents' educational level around age 14. This suggests that parental SES is an important determinant of alcohol use in early adolescence, above the effect of own educational level; perhaps because parental SES reflects differences in parental attachment, alcohol use, and alcohol-related permissiveness (Pape et al., 2017).

5. CONCLUSIONS AND IMPLICATIONS

We mainly found evidence in support of the social causation theory in early adolescence, when lower education predicted increases in subsequent alcohol use. In young adulthood, we found tendencies towards opposite associations, with a stronger escalation of alcohol use amongst the higher educational tracks, though most of these effects failed to reach statistical significance in adjusted CLPMs. We found no evidence for health-related selection attributable to alcohol use throughout adolescence and young adulthood. The very high stability in educational level throughout adolescence might be typical of countries with educational systems characterized by an early selection and highlights the importance of determinants already present in childhood, which predict the initial selection into educational tracks. By determining educational level in early adolescence, these characteristics also predict subsequent inequalities in alcohol use.

Our findings emphasize the need for interventions to delay the early escalation of alcohol use amongst adolescents in the lower educational tracks. Early drinking is an important predictor of later problematic use and alcohol use disorders (Grant & Dawson, 1997). Background characteristics were not able to explain this early escalation, pointing towards educational differences in social norms and peer group composition in the educational context. Interventions may aim to integrate adolescents' social networks and popular peers in particular, who my act as key opinion leaders promoting good health behaviours (Campbell et al., 2008; Peeters et al., 2021). Peer-led interventions have been shown to reduce adolescent alcohol use (MacArthur et al., 2016). While less is known about peer-led interventions for young adults, targeting social norms may also be effective in reducing drinking in young adults in college, (Pischke et al., 2021; Wolter et al., 2021).

DATA AVAILABILITY STATEMENT

Under the General Data Protection Regulation (GDPR), our dataset is considered pseudonymized rather than anonymized, and is still regarded as personal data. When participants were invited to the cohort more than 20 years ago, they were not asked to give

informed consent to make their personal data publicly available in pseudonymized form. As a result of this, legal and ethical restrictions prevent the authors from making data from the TRAILS study publicly available. Data are available upon request from the TRAILS data manager (trails@umcg.nl). Detailed information about the participation agreements with TRAILS participants is available from the ethics committee; Central Committee on Research Involving Human subjects (CCMO; tc@ccmo.nl). For more information about accessing data from the TRAILS study, please see https://www.trails.nl/en/hoofdmenu/data/data-use. The Mplus syntax for our analyses can be obtained from: https://github.com/hschmengler/Educational-level-and-alcohol-use-in-adolescence-and-early-adulthood---the-TRAILS-Study.

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COMPETING INTERESTS

The authors have declared that no competing interests exist.

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2

CHAPTER 3

EDUCATIONAL LEVEL, ATTENTION PROBLEMS, AND EXTERNALIZING BEHAVIOUR IN ADOLESCENCE AND EARLY ADULTHOOD: THE ROLE OF SOCIAL CAUSATION AND HEALTH-RELATED SELECTION – THE TRAILS STUDY

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ABSTRACT

Social causation and health-related selection may contribute to educational differences in adolescents' attention problems and externalizing behaviour. The social causation hypothesis posits that the social environment influences adolescents' mental health. Conversely, the health-related selection hypothesis proposes that poor mental health predicts lower educational attainment. From past studies it is unclear which of these mechanisms predominates, as attention problems and externalizing behaviour have the potential to interfere with educational attainment, but may also be affected by differences in the educational context. Furthermore, educational gradients in mental health may reflect the impact of 'third variables' already present in childhood, such as parental socioeconomic status (SES), and IQ. We investigated both hypotheses in relation to educational differences in externalizing behaviour and attention problems throughout adolescence and young adulthood. We used data from a Dutch cohort (TRAILS study; N = 2,229), including five measurements of educational level, externalizing behaviour, and attention problems from around age 14 to 26 years. First, we evaluated the directionality in longitudinal associations between education, externalizing behaviour, and attention problems with and without adjusting for individual differences using fixed effects. Second, we assessed the role of IQ and parental SES in relation to attention problems, externalizing behaviour, and educational level. Attention problems predicted decreases in education throughout all of adolescence and young adulthood. Differences in parental SES contributed to increases in externalizing behaviour amongst the lower educational tracks in mid-adolescence. Childhood IQ and parental SES strongly predicted education around age 14. Parental SES, but not IQ, also predicted early adolescent attention problems and externalizing behaviour. Our results provide support for the health-related selection hypothesis in relation to attention problems and educational attainment. Further, our results highlight the role of social causation from parental SES in determining adolescent educational level, attention problems, and externalizing behaviour.

1. BACKGROUND

Externalizing behaviour and attention problems have been associated with socioeconomic adversity over the life course (Fergusson & Horwood, 1998; Galéra et al., 2012). These associations begin in childhood, when children growing up in lower SES households are more likely to show these problem behaviours and subsequently to be assigned to lower educational tracks in selective educational systems (Miller et al., 2018; Piotrowska et al., 2015; van Spijker et al., 2017). Selective educational systems are defined by an early selection into different educational tracks and thus different social contexts, while allowing for mobility between tracks post-selection (Berten et al., 2012; Delaruelle et al., 2019). Externalizing behaviour comprises a wide range of disruptive behaviours, including overt aggressiveness, as well as more covert rule-breaking (delinquent) behaviours (Farrington, 2004; Palmu et al., 2018). Externalizing behaviour is often comorbid with attention problems, and both syndromes share common risk factors (e.g., low effortful control, low parental SES) (Atherton et al., 2020b; Du Rietz et al., 2020; Miller et al., 2018; Piotrowska et al., 2015). Both attention problems and externalizing behaviour can be highly disruptive in educational contexts, hence predisposing to academic problems (Arnold, 1997; Maguin & Loeber, 1996; Polderman et al., 2010) and lower educational attainment and SES in young adulthood (Galéra et al., 2012; Havas et al., 2009; Jayakody et al., 1998; Veldman et al., 2014). At the same time, the social environment at school can have a considerable impact on both problem behaviours (Van Houtte & Stevens, 2008). Despite these shared features, attention problems and externalizing behaviour involve different behaviours that may affect and be affected by the environment in different ways. Therefore, attention problems and externalizing behaviour might be linked to educational outcomes through different mechanisms, and hence need to be studied separately (Palmu et al., 2018).

Two mechanisms may contribute to the entrenchment of educational inequalities in attention problems and externalizing behaviour: social causation and health-related selection (Mackenbach, 2012, 2019). The health-related selection hypothesis posits that health problems, including problem behaviours, can lead to a decline in educational level (Reiss, 2013). Adolescents with high levels of attention problems experience difficulties following teachers' instructions and staying on-task in class, as well as when completing their homework. As a result, these adolescents achieve lower grades, have higher risks of repeating classes, and may eventually decline in their educational level (DuPaul & Langberg, 2015; Jangmo et al., 2019). Also externalizing behaviour may interfere with adolescents' education, as adolescents with high levels of externalizing behaviour often experience rejection by peers with low levels of externalizing behaviour and may gravitate towards academically unengaged aggressive or delinquent classmates. This may lead to a loss of interest in school and a further escalation in externalizing behaviour, which, in its more

extreme forms, can lead to expulsion from class, and eventually to a decline in educational track (Moilanen et al., 2010).

As opposed to health-related selection, social causation explanations emphasize the role of differences in the social environment in explaining educational differences in externalizing behaviour and attention problems. The selective educational system in the Netherlands entails that Dutch students grow up in distinct educational environments that are characterized by different social norms, future expectations, cognitive resources, and occupational prospects (Berten et al., 2012; Delaruelle et al., 2019; Van Houtte & Stevens, 2008). The lower educational tracks focus on skills training, and often lead to lower-paid vocations that carry less societal esteem than vocations that require attending a higher, more theoretically focused, educational track (Delaruelle et al., 2019; Van Houtte & Stevens, 2008). Students in the lower tracks may therefore be less able to seek a socio-occupational position valued by society in terms of secure, well-paid, and esteemed employment (Van Houtte & Stevens, 2008). These adolescents may then turn to alternative social fields to gain recognition, which may involve affiliation with delinquent peers and externalizing behaviour, as rebellion against meritocratic social norms (Elstad, 2010). Furthermore, peer contagion effects may contribute to stronger increases in externalizing behaviour in the lower educational tracks. Adolescents with long-term behavioural problems are overrepresented in these tracks (Veldman et al., 2014), possibly resulting from health-related selection effects, as discussed above. As a result of this, normatively developing adolescents in the lower tracks are more frequently exposed to externalizing behaviour in the classroom context. Studies have shown that adolescents with initially low levels of externalizing behaviours often mimic the behaviours of aggressive/antisocial peers, themselves becoming more aggressive over time (Dishion & Tipsord, 2011; Moffitt, 1993). These peer contagion effects may tilt classroom norms in the lower tracks more strongly toward externalizing behaviours (Dishion & Tipsord, 2011; Moffitt, 1993). A higher prevalence of externalizing behaviours amongst the lower tracks may also lead to noisier and unrulier classrooms (Berger et al., 2017; Maxwell, 2010), potentially contributing to higher levels of attention problems in those tracks.

Furthermore, it is pivotal to evaluate the social causation and health-related selection hypotheses in relation to stable background characteristics, which are already present in childhood and predict later educational level, attention problems, and externalizing behaviour. These risk factors may act as time-invariant 'third variables' (i.e., confounders) in longitudinal associations between educational level and problem behaviours, and therefore need to be adjusted for in statistical models. From a social causation perspective, family SES is especially important. Lower SES parents may have fewer resources to support the academic performance of their child, leading to a lower initial placement in the educational system (van Spijker et al., 2017). Additionally, these parents often lack the financial means to provide a safe and stimulating environment, which is highly important for children's

cognitive and emotional development (Ng-Knight & Schoon, 2017; Rosen et al., 2018), leading to increased risks of attention problems and externalizing behaviour (Piotrowska et al., 2015; Piotrowska et al., 2019; Reiss, 2013; Russell et al., 2016). From a health-related selection perspective, it is important to take into account common early psychological risk factors of attention problems, externalizing behaviour, and low educational attainment, such as low IQ, as well as genetic risk factors (Du Rietz et al., 2020; Moffitt, 1993; Odgers et al., 2008; Rommelse et al., 2017; Tistarelli et al., 2020). These risk factors are not always measured (e.g., in the case of genetics), but can still partly be adjusted for by using statistical models that allow for the decomposition of variance into within- and between-person components (Allison et al., 2017; Hamaker et al., 2015).

In order to evaluate social causation and health-related selection explanations in relation to the association between adolescents' educational level and attention problems and externalizing behaviour, it is necessary to investigate whether educational level more strongly predicts these problem behaviours (i.e., social causation), or vice versa (i.e., health-related selection). This can be done using longitudinal datasets and cross-lagged panel models (CLPMs). To the best of our knowledge, all previous studies addressing this issue used either some form of cognitive testing or Grade Point Average (GPA)-based measures to assess educational performance (Chen et al., 2010; Defoe et al., 2013; Deighton et al., 2018; Lee, 2012; Masten et al., 2005; Metsäpelto et al., 2015; Okano et al., 2020; Palmu et al., 2018; Vaillancourt et al., 2013; Van der Ende et al., 2016; Weeks et al., 2016; Yong et al., 2013; Zhang et al., 2019; Zimmermann et al., 2013), and most were conducted in comprehensive educational systems, which are characterized by a late differentiation into educational tracks (e.g., in the USA at around age 18 upon completing high school) (Chen et al., 2010; Defoe et al., 2013; Deighton et al., 2018; Masten et al., 2005; Metsäpelto et al., 2015; Okano et al., 2020; Palmu et al., 2018; Vaillancourt et al., 2013; Weeks et al., 2016; Yong et al., 2013; Zhang et al., 2019). This late differentiation means that unlike in the Dutch system adolescents' educational level (as indicator of developing SES) does not become entrenched until the transition out of secondary school. It may therefore be quite difficult to assess adolescents' developing SES in comprehensive educational systems, and GPA-based measures may indeed be the best indication in such systems. We identified only two studies from selective educational systems. These studies also used GPA-based measures, but did not take into account the role of adolescents' educational tracks, which characterise selective educational systems (Van der Ende et al., 2016; Zimmermann et al., 2013). In selective educational systems, GPA-based measures only express performance relative to other students within the same track and are therefore not appropriate for predicting adolescents' socioeconomic prospects.

Furthermore, many existing studies focus on specific developmental periods (e.g., early adolescence) (Defoe et al., 2013; Okano et al., 2020; Palmu et al., 2018; Vaillancourt et al., 2013; Yong et al., 2013; Zhang et al., 2019; Zimmermann et al., 2013). However, it is pivotal

to consider all phases of adolescent development simultaneously, as the importance of social causation and health-related selection mechanisms may differ across age groups. In addition, attention problems, externalizing behaviour, and academic outcomes may form developmental cascades (Defoe et al., 2013). For example, in a US study, attention problems predicted lower academic achievement in adolescents, which in turn predicted increased delinquent behaviour (Defoe et al., 2013). It is important to account for these cascading effects across phases of development, to strengthen causal inference.

Past studies based on GPA-based measures or cognitive tests have found different results for attention problems and externalizing behaviour (Palmu et al., 2018). When considering externalizing behaviour, four studies found mainly evidence for the social causation hypothesis (Defoe et al., 2013; Lee, 2012; Palmu et al., 2018; Vaillancourt et al., 2013), with lower academic achievement predicting more problem behaviours, whilst three found mainly evidence for the health-related selection hypothesis, with externalizing behaviour predicting decreases in subsequent academic achievement (Chen et al., 2010; Deighton et al., 2018; Masten et al., 2005). Bidirectional associations between academic achievement and externalizing behaviours were found in seven studies (Metsäpelto et al., 2015; Okano et al., 2020; Van der Ende et al., 2016; Weeks et al., 2016; Yong et al., 2013; Zhang et al., 2019; Zimmermann et al., 2013). In contrast, when considering attention problems, studies mainly support the health-related selection hypothesis, with symptoms more strongly predicting academic achievement than vice versa in two studies (Defoe et al., 2013; Palmu et al., 2018). These results underline the importance of making a distinction between attention problems and externalizing behaviour for answering our research questions rather than combining these concepts into one underlying dimension.

1.1. Aims of the study

In the present study, we aimed to contribute to the literature by modelling reciprocal relationships (i.e., social causation and health-related selection) between attention problems and externalizing behaviour and adolescent educational level over a period of 16 years, using educational track membership as proxy for developing socioeconomic status (SES) in a selective educational system. Using educational track membership in the Dutch system as measure for educational attainment allows for assessing social mobility in adolescents at an earlier age than possible in comprehensive educational systems, as social stratification already occurs around the beginning of adolescence. Furthermore, we aimed to evaluate the role of family socioeconomic status (SES) and childhood IQ both as predictors of educational level, attention problems, and externalizing behaviour around age 14, and as confounders ('third variables') in cross-lagged paths. Finally, we address unmeasured time-stable confounding in cross-lagged paths using fixed effects.

2. MATERIALS AND METHODS

2.1. Study population

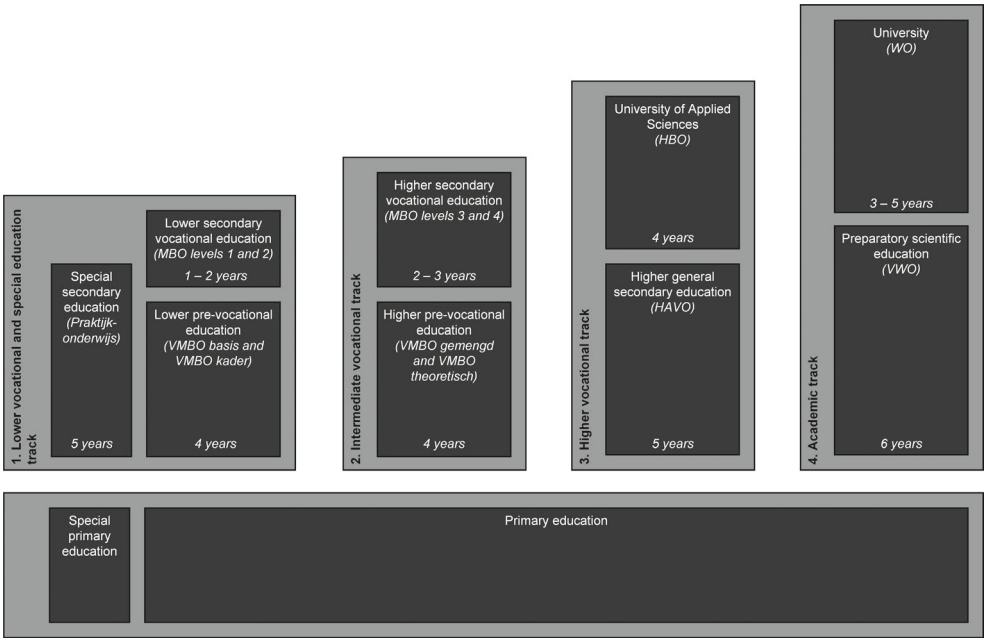
We used data from the first six waves (T1 – T6) of the TRacking Adolescents' Individual Lives Survey (TRAILS), a population-based prospective cohort study of Dutch adolescents. A detailed description of the cohort can be obtained elsewhere (Oldehinkel et al., 2015). At the beginning of the study, 135 schools in the province of Groningen were invited, of which 122 decided to participate (de Winter et al., 2005). Adolescents were followed between 2000 and 2017 with assessments around age 11 (N = 2,229), 14 (N = 2,148), 16 (N = 1,818), 19 (N = 1,880), 22 (N = 1,781), and 26 (N = 1,616). Attrition analyses (**Table 3.S1**) revealed that male gender, non-Dutch ethnicity, lower educational level, IQ, and parental SES were associated with dropout. Higher externalizing behaviour also predicted dropout, but only at wave 3 and 4. Attention problems were not associated with attrition. Some similar differences were found when comparing participants with complete information on educational level to those whose educational level was missing or could not be classified (**Table 3.S2**). To deal with missing information, full-information maximum likelihood (FIML) was implemented, allowing to incorporate information from all participants.

2.2. Adolescents' educational level

The Dutch educational system is characterized by an early (age 11-12) selection into a particular educational track, based on cognitive tests and the advice of the primary school. There are four tracks in the Dutch educational system, each consisting of a specific type of secondary school followed by tertiary education at the corresponding level (**Figure 3.1**): 1. lower vocational track, 2. intermediate vocational track, 3. higher vocational track, 4. academic track. In addition, there is a special education track, attended by students who are unable to attend regular education. This track was collapsed with the lower vocational track in our analyses. While in secondary education, adolescents can be recommended by their school to move between educational tracks, depending on their academic performance. Furthermore, after attaining specific milestones of their track, students can become eligible to continue their education in the next higher track. For example, students who finish the intermediate vocational track with an MBO level 4 diploma may continue their education by attending a University of Applied Sciences of the higher vocational track (Wet op het hoger onderwijs en wetenschappelijk onderzoek, 2021). Overall, a substantial proportion of students is mobile between educational tracks: 24.66% of adolescents moved to a different track between wave 2 and 3, 25.41% between wave 3 and 4, 26.79% between wave 4 and 5, and 12.98% between wave 5 and 6, respectively. 47.00% of TRAILS participants were in a different educational track around age 26 (wave 6) than around age 14 (wave 2). Educational track membership was assessed at each wave by asking for participants' current enrolment, as well as their highest completed diploma. Participants who finished

the final diploma of a given track received the value corresponding to that level for all subsequent waves, unless they continued education at a higher level. Our measure of educational level allows us to assign a score that represents an age-appropriate measure of educational attainment as proxy of developing SES. Missing information on educational track membership from waves 2 through 6 was imputed using retrospective event history calendars conducted at wave 3 and wave 5. Participants who were still in elementary education or in a combined class at wave 2 were assigned according to their elementary school teachers' recommended level. If this information was not available, pupils were classified according to the first track they attended after leaving elementary education or the combined class. It was not possible to classify participants who had not been in education for a longer period, were not classifiable into an educational track (e.g., because of education abroad), whose educational level was assessed incompletely, who did not respond to questions on education, or who had left the educational system permanently (wave 2: N = 221, 10.29%; wave 3: N = 289, 15.90%; wave 4: N = 373, 19.84%; wave 5: N = 352, 19.76%; wave 6 = 424, 26.24%). Educational level was set to missing for these participants.

FIGURE 3.1 The Dutch educational system



2.3. Adolescents' mental health

Attention problems were assessed by calculating the mean scores (possible range 0 – 2) of the attention problem subscales of the Achenbach System of Empirically Based

Assessment (ASEBA) Youth Self-report (YSR) (waves 2 – 3; 8 items) and Adult Self-report (ASR) (waves 4 – 6; 15 items) (Achenbach & Rescorla, 2001, 2003). The YSR and ASR contain lists of questions on emotional and behavioural problems in the preceding 6 months, with three response categories: 0 = 'not true', 1 = 'somewhat or sometimes true', 2 = 'very or often true' (Veldman et al., 2014). The item 'my schoolwork is bad' was removed from the YSR attention problem scale, as it reflects the main mechanism through which attention problems affect educational level rather than attention problems themselves. Sample items of the attention problem scale are 'I have difficulties concentrating' and 'I don't finish tasks I begin'. Cronbach's alphas for the attention problem scales ranged from 0.71 to 0.73 for the YSR, and from 0.84 to 0.86 for the ASR.

Externalizing behaviour was assessed by taking the mean score (possible range 0 – 2) of the externalizing subscales of the YSR (waves 2 – 3; 29 items) and ASR (waves 4 – 6; 33 items) (Achenbach & Rescorla, 2001, 2003). Items on substance use were removed from the scales (3 for the YSR and 2 for the ASR). This decision was made because adolescents' substance use is heavily influenced by cultural factors and often shows unique relationships with educational level, which diverge from other externalizing behaviours (Peeters et al., 2019). For example, increased alcohol use has been associated with lower educational level in early adolescence, and higher educational level in late adolescence and young adulthood (de Looze et al., 2017; Jang et al., 2019). Sample items from the externalizing behaviour scales are 'I steal' and 'I physically attack others'. Cronbach's alphas for the YSR ranged from 0.85 to 0.86, and for the ASR from 0.87 to 0.89.

2.4. Characteristics at baseline (wave 1)

Demographic baseline characteristics were adolescent age, gender, area of residence (City of Groningen, Leeuwarden, Assen, other regions), and ethnicity. Children were classified as having non-Dutch ethnicity if at least one of their parents was born outside the Netherlands (Vollebergh et al., 2005). In addition, we included the following two baseline characteristics that were hypothesized to be associated with attention problems, externalizing behaviour, and education based on earlier studies (Brody, 1997; Moffitt, 1993; Odgers et al., 2008; Piotrowska et al., 2015; Piotrowska et al., 2019; Reiss, 2013; Rommelse et al., 2017; Russell et al., 2015; Russell et al., 2016; van Spijker et al., 2017):

1. *Parents' socioeconomic status (SES)*, constructed as the mean score (observed range -1.94 – 1.73) of the following five indicators (standardized): maternal and paternal educational attainment, maternal and paternal occupational position (according to the International Standard Classification of Occupations), and family income (Amone-P'Olak et al., 2009).
2. *Intelligence Quotient (IQ)* was estimated using the Block Design and Vocabulary subtests of the Revised Wechsler Intelligence Scale for Children (WISC-R) (observed range 45 – 149) (Brunnekreef et al., 2007).

2.5. Analytic approach

First, we computed descriptive statistics of the study population by cross-tabulating baseline characteristics (mean age 11) with early adolescent educational track membership at wave 2 (mean age 14), and attention problems and externalizing behaviour with concurrent educational level from wave 2 through wave 6 (mean age 26). Second, we conducted standardized linear regression models to estimate the relative importance of parental SES and adolescents' IQ at baseline in predicting attention problems, externalizing behaviour, and educational level at wave 2. Third, we computed cross-lagged panel models (CLPMs) between attention problems and externalizing behaviour and educational level from wave 2 through wave 6. The CLPM estimates prospective associations between educational level and changes in subsequent attention problems and externalizing behaviour, and between attention problems and externalizing behaviour and changes in subsequent educational level, whilst taking into account temporal stability and reciprocity (Allison et al., 2017). We first conducted separate CLPMs for attention problems and externalizing behaviour (see **Figure 3.S1** for a schematic illustration), both unadjusted and adjusted for demographics, parental SES, and IQ. Subsequently, we combined attention problems and externalizing behaviour in a single CLPM to estimate their unique relationships with educational level (**Figure 3.S2**).

In addition, we conducted CLPMs with fixed effects, according to a specification by Allison et al. (2017). These models only use within-person variance to estimate associations between cross-lagged variables, hereby adjusting for all measured and unmeasured time-invariant characteristics. Separate one-sided fixed effects models were fit to assess lagged associations from education to changes in subsequent attention problems or externalizing behaviour, and from attention problems or externalizing behaviour to changes in subsequent education (**Figure 3.S3**). The fixed effects terms were represented by a latent variable of all measurements of the outcome with each having its factor loading constrained to be 1. This latent variable was allowed to be correlated freely with all time-varying exogenous variables in the model. Reciprocal causation was accommodated by including correlations between the error term of the outcome at each measurement occasion and all future values of the time-varying exposure (Allison et al., 2017). Unlike in the traditional CLPM, we were not able to combine attention problems, externalizing behaviour, and education in a fixed effects model, when assessing the direction from education to attention problems/externalizing behaviour (i.e., social causation), as this led to convergence problems in Mplus (correlations >1). We were able to compute such a fixed effects model, which additionally included baseline covariates, for the direction from attention problems/externalizing behaviour to education, with results identical to the fixed effects models assessing attention problems and externalizing behaviour separately, and which did not include baseline covariates (**Figure 3.S4**). We hence assessed cross-lagged

associations between externalizing behaviour and education, and attention problems and education, respectively, in separate fixed effects models without covariates (**Figure 3.S3**).

Model fit in Structural Equation Modelling (SEM) analyses was assessed using the Comparative Fit Index (CFI), the Tucker Lewis Index (TLI), the Root Mean Square Error of Approximation (RMSEA), and the Standardised Root Mean Residual (SRMR). Following the suggestions by Hu & Bentler (1999), model fit was judged as 'good' if the CFI and TLI were >0.95, the RMSEA was <0.06, and the SRMR was <0.08. Standard errors were estimated using robust maximum likelihood (MLR). Analyses were conducted in Mplus 8.6.

2.6. Sensitivity analyses

In theory, fixed effects models require consistent measures of constructs over time (Hamaker, 2018). In practice, this is often difficult to achieve in developmental research, which frequently aims to study multiple phases of development simultaneously. For example, oftentimes different, yet closely related, instruments are used to increase validity when assessing the same types of problem behaviours at different developmental stages. In TRAILS, the YSR is used to assess attention problems and externalizing behaviour in early and mid-adolescence, while the ASR is used in late adolescence and young adulthood. While it is common practice to combine YSR and ASR in the same models (Kan et al., 2013; Peeters et al., 2019; Veldman et al., 2015), we have conducted a sensitivity analysis of the fixed effects models using amended scales consisting only of corresponding items between the YSR and ASR (**Table 3.S3**), to test whether using these slightly different instruments at different time points may have affected our findings**.**

Externalizing behaviour, attention problems, and educational problems frequently co-occur with internalizing problems (Tistarelli et al., 2020; Van der Ende et al., 2016). In order to study the unique associations between externalizing behaviour, attention problems, and education, some researchers have therefore suggested to additionally adjust for internalizing symptoms in CLPMs (Van der Ende et al., 2016). We have hence conducted a second sensitivity analysis in which the multivariate CLPM was additionally adjusted for the mean score of the summed anxious/depressed and withdrawn/depressed subscales of the YSR and ASR. Finally, we checked whether there were sex differences in cross-lagged associations in our fixed effects models, using multigroup modelling (van Lier et al., 2012).

3. RESULTS

3.1. Descriptive statistics

Table 3.1 shows the characteristics of TRAILS participants around age 11 according to educational level around age 14. Children with less affluent or non-Dutch parents more commonly attended the lower educational tracks. Girls more frequently attended the

academic and intermediate vocational tracks than boys. Children in the lower vocational track and the academic track were slightly older at baseline than those in the intermediate and higher vocational tracks. Further, higher IQ around age 11 was associated with higher education around age 14.

Table 3.2 shows educational differences in attention problems and externalizing behaviour around ages 14, 16, 19, 22, and 26. Over the course of adolescence, educational differences in externalizing behaviour increased, with lower educational level being associated with more externalizing behaviour. When considering attention problems, adolescents in the academic track had lower scores than the lowest two tracks around age 14, and lower scores than all other tracks at 16, but we did not find clear differences among the other educational tracks. From age 19 onwards, no ordered relationships between educational level and attention problems were found anymore.

TABLE 3.1 Characteristics of adolescents participating in the TRAILS study (the Netherlands, 2000–2017, N = 2,229) at wave 1 (2000-2002) according to educational level at wave 2 (2003-2005)

	All levels		**Lower vocational & special education**		**Intermediate vocational**		**Higher vocational**		**Academic**	
	N = 2,229		**N = 635**		**N = 497**		**N = 383**		**N = 457**	
Male gender, N (%)	1,098	(49.26)	341	(53.70)[a]	217	(43.66)[b]	196	(51.17)[a]	195	(42.67)[b]
Non-Dutch ethnicity, N (%)	301	(13.50)	108	(17.01)[a]	61	(12.27)[b]	39	(10.18)[b]	45	(9.85)[b]
Age, mean (SD)	11.11	(0.56)	11.16	(0.56)[a]	11.07	(0.54)[b]	11.05	(0.56)[b]	11.14	(0.56)[a]
Parental socioeconomic status (SES), mean (SD)	-0.05	(0.80)	-0.53	(0.70)[a]	-0.16	(0.67)[b]	0.21	(0.68)[c]	0.55	(0.70)[d]
Wechsler Intelligence Deviation Quotient (IQ), mean (SD)	97.19	(15.00)	86.05	(12.49)[a]	95.20	(10.98)[b]	102.68	(11.20)[c]	111.14	(11.91)[d]

SD = standard deviation.
Parameters with different superscripts differ significantly from each other at $p < 0.05$, as determined by chi-squared tests (categorical variables) and one-way ANOVAs with pairwise comparisons (continuous variables).

3.2. Prospective associations between childhood characteristics and early adolescent educational level, attention problems, and externalizing behaviour

Table 3.3 shows prospective associations between baseline (around age 11) characteristics and educational level, attention problems, and externalizing behaviour around age 14. In bivariate results, higher parental SES and child IQ strongly predicted higher educational level around age 14. These results remained robust after adjusting for covariates, be it that a relatively large proportion of the association between SES and education was explained by differences in IQ. Higher parental SES also predicted lower attention problems and

TABLE 3.2 Mental health characteristics of adolescents and young adults in the TRAILS study (wave 2 – 6; the Netherlands, 2000–2017, N = 2,229) according to concurrent educational level

	Wave 2		**Wave 3**		**Wave 4**		**Wave 5**		**Wave 6**	
	N = 2,148		**N = 1,818**		**N = 1,880**		**N = 1,781**		**N = 1,616**	
Date range	2003-2005		2005-2008		2008-2010		2012-2014		2016-2017	
Age, mean (SD)	13.57	(0.53)	16.28	(0.71)	19.08	(0.60)	22.29	(0.65)	25.66	(0.60)
Male gender, N (%)	1,054	(49.07)	867	(47.69)	898	(47.77)	843	(47.33)	735	(45.48)
Educational level, N (%)										
Lower vocational & special education	635	(32.20)	349	(22.83)	161	(10.68)	136	(9.52)	78	(6.54)
Intermediate vocational	497	(25.20)	405	(26.49)	498	(33.02)	354	(24.77)	273	(22.90)
Higher vocational	383	(19.42)	362	(23.68)	475	(31.50)	594	(41.57)	489	(41.02)
Academic	457	(23.17)	413	(27.01)	374	(24.80)	345	(24.14)	352	(29.53)
Mental health characteristics										
Attention problems, mean (SD)										
All levels	0.56	(0.34)	0.59	(0.35)	0.45	(0.32)	0.43	(0.32)	0.45	(0.34)
Lower vocational & special education	0.57	(0.34)[a]	0.64	(0.36)[a]	0.45	(0.34)[a]	0.44	(0.32)[a/b]	0.48	(0.33)[a]
Intermediate vocational	0.59	(0.34)[a]	0.61	(0.34)[a]	0.45	(0.33)[a]	0.40	(0.29)[a]	0.41	(0.29)[a]
Higher vocational	0.57	(0.34)[a/b]	0.61	(0.32)[a]	0.48	(0.31)[a]	0.45	(0.32)[b]	0.45	(0.33)[a]
Academic	0.53	(0.33)[b]	0.54	(0.34)[b]	0.44	(0.31)[a]	0.41	(0.34)[a/b]	0.43	(0.36)[a]
Externalizing behaviour, mean (SD)										
All levels	0.31	(0.21)	0.32	(0.21)	0.23	(0.21)	0.19	(0.18)	0.20	(0.19)
Lower vocational & special education	0.32	(0.21)[a]	0.37	(0.24)[a]	0.28	(0.26)[a]	0.22	(0.19)[a]	0.25	(0.21)[a]
Intermediate vocational	0.32	(0.21)[a]	0.34	(0.22)[a]	0.23	(0.21)[b]	0.20	(0.18)[a]	0.19	(0.18)[b/c]
Higher vocational	0.30	(0.20)[a/b]	0.31	(0.20)[b]	0.23	(0.20)[b]	0.19	(0.19)[a]	0.19	(0.18)[b]
Academic	0.28	(0.18)[b]	0.27	(0.18)[c]	0.19	(0.18)[c]	0.16	(0.16)[b]	0.17	(0.16)[c]

SD = standard deviation. Parameters with different superscripts differ significantly from each other at p < 0.05, as determined by one-way ANOVAs with pairwise comparisons.

externalizing behaviour, albeit to a lesser extent. Baseline IQ was neither associated with attention problems nor externalizing behaviour at around age 14.

TABLE 3.3 The association between baseline characteristics (wave 1) and educational level, externalizing behaviour, and attention problems at wave 2 in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); unadjusted and adjusted linear regression models (stdyx-standardized ß-coefficient, robust standard error, p-value)

	Model 1	**Model 2**	**Model 3**
	Educational level		
Parental socioeconomic status (SES)	**0.506 (0.017), p<0.001**	**0.498 (0.018), p<0.001**	**0.303 (0.018), p<0.001**
Wechsler Intelligence Deviation Quotient (IQ)	**0.621 (0.013), p<0.001**	**0.629 (0.013), p<0.001**	**0.516 (0.016), p<0.001**
	Externalizing behaviour		
Parental socioeconomic status (SES)	**-0.067 (0.022), p=0.002**	**-0.068 (0.023), p=0.003**	**-0.067 (0.025), p=0.006**
Wechsler Intelligence Deviation Quotient (IQ)	-0.029 (0.023), p=0.215	-0.028 (0.023), p=0.239	-0.002 (0.025), p=0.924
	Attention problems		
Parental socioeconomic status (SES)	**-0.045 (0.022), p=0.046**	**-0.064 (0.023), p=0.005**	**-0.062 (0.025), p=0.012**
Wechsler Intelligence Deviation Quotient (IQ)	-0.023 (0.023), p=0.311	-0.028 (0.023), p=0.219	-0.005 (0.025), p=0.833

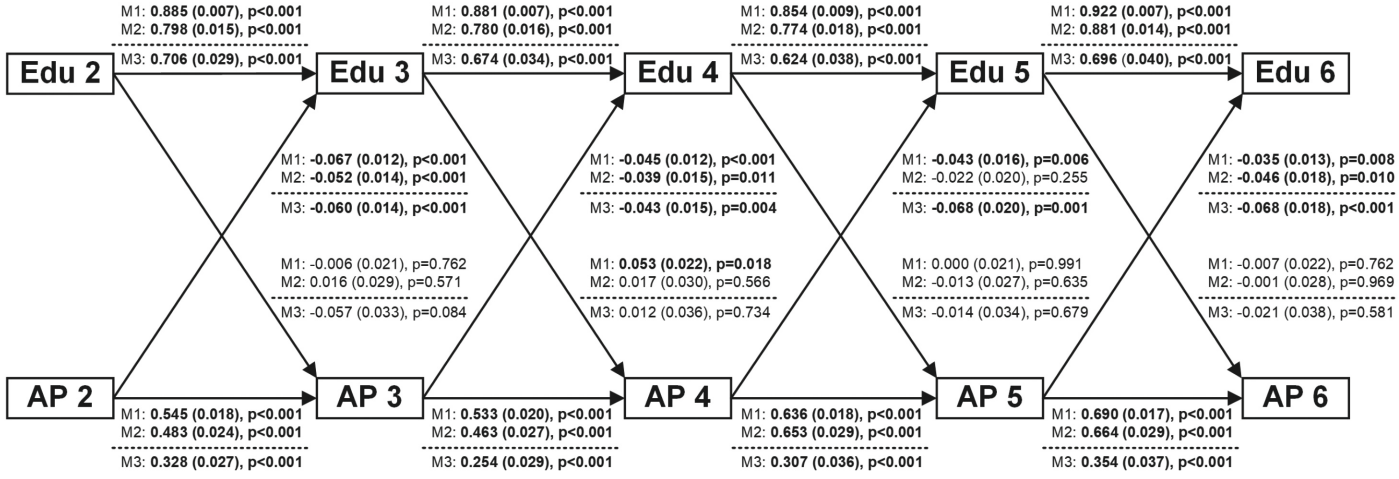
Model 1: unadjusted models.
Model 2: adjusted for age, sex, municipality, and ethnicity at baseline (wave 1).
Model 3: adjusted for age, sex, municipality, ethnicity, and adjusted for parental socioeconomic status or IQ at baseline (wave 1).
***Boldface** denotes statistical significance at p < 0.05.*

3.3. Cross-lagged associations for attention problems and education

In bivariate CLPMs, educational level exhibited consistently high stability (standardized ß > 0.800; **Figure 3.2**, **Figure 3.3**). The stability of attention problems was also high and increased from a standardized ß of 0.545 in early adolescence to 0.690 in young adulthood (**Figure 3.2**). Bivariate analyses showed that attention problems robustly predicted subsequent decreases in education throughout the entire study period. The same results were found in the fixed effects model, suggesting that these associations were not explained by time-invariant confounders. These associations also remained significant in the covariate-adjusted models (i.e., adjusted for demographics, parental SES, IQ, and concurrent externalizing behaviour), except for the path between age 19 and 22, which was no longer significant after adjusting for externalizing behaviour (**Figure 3.S5**).

When considering social causation, we only found one bivariate association between higher education around age 16 and increases in attention problems around age 19. This

FIGURE 3.2 Bidirectional associations between educational level and attention problems in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) from cross-lagged panel models without (Model 1 and 2) and with fixed effects (Model 3)

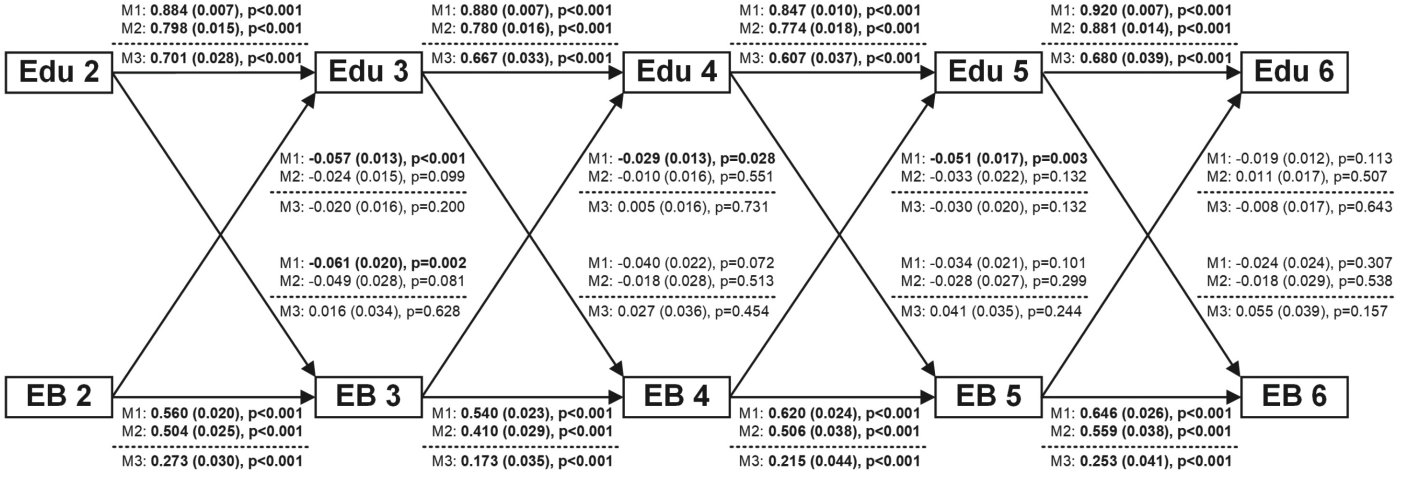


	Chi-square	df	CFI	TLI	SRMR	RMSEA
Model 1	271.691	24	0.977	0.957	0.064	0.068
Model 2	457.851	54	0.976	0.900	0.040	0.058
Model 3						
a) Education → AP	59.055	9	0.994	0.977	0.023	0.050
b) AP → Education	18.742	9	0.999	0.996	0.005	0.022

Model 1: bivariate cross-lagged panel model. Model 2: cross-lagged panel model adjusted for age, gender, area of residence, ethnicity, parental SES, and IQ at baseline (wave 1), and externalizing behaviour at each preceding wave. Model 3: cross-lagged panel models with fixed effects – adjustment for time-invariant characteristics was performed by inclusion of a latent variable.

Edu = educational level; AP = attention problems. ***Boldface*** *denotes statistical significance at p < 0.05.*

FIGURE 3.3 Bidirectional associations between educational level and externalizing behaviour in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) from cross-lagged panel models without (Model 1 and 2) and with fixed effects (Model 3)



	Chi-square	df	CFI	TLI	SRMR	RMSEA
Model 1	342.449	24	0.964	0.933	0.079	0.077
Model 2	457.851	54	0.976	0.900	0.040	0.058
Model 3						
a) Education → EB	50.302	9	0.994	0.977	0.025	0.045
b) EB → Education	32.017	9	0.997	0.990	0.007	0.034

Model 1: bivariate cross-lagged panel model. Model 2: cross-lagged panel model adjusted for age, gender, area of residence, ethnicity, parental SES, and IQ at baseline (wave 1), and attention problems at each preceding wave. Model 3: cross-lagged panel models with fixed effects – adjustment for time-invariant characteristics was performed by inclusion of a latent variable. Edu = educational level; EB = externalizing behaviour. ***Boldface*** *denotes statistical significance at p < 0.05.*

association was no longer significant after adjusting for parental SES, and disappeared almost completely after further taking into account adolescents' IQ (**Figure 3.S5**). We found no significant associations between education and subsequent changes in attention problems in the fixed effects model.

The fixed effects models with the amended scales (**Figure 3.S7**) were in line with the main results, with significant paths from attention problems to subsequent decreases in educational level throughout the entire followup, and no significant paths from educational level to subsequent changes in attention problems. Also, further adjusting CLPMs for anxiety/depression did not change cross-lagged associations between attention problems and education (**Figure 3.S5**). While we were not able to find significant differences in any cross-lagged associations between boys and girls in fixed effects models, we noted substantial increases in standard errors in the multigroup models, particularly regarding the social causation paths (**Figure 3.S9**). Furthermore, allowing for sex differences in cross-lagged paths did not improve model fit, as compared to models where cross-lagged paths were constrained to be equal across sexes.

3.4. Cross-lagged associations for externalizing behaviour and education

Externalizing behaviour showed similar stability to attention problems, with slight increases over time, ranging from a standardized ß of 0.560 in early adolescence to 0.646 in young adulthood, in bivariate models (**Figure 3.3**). When considering health-related selection, we found that externalizing behaviour predicted decreases in education from around age 14 through 22, but not from around age 22 to 26. These associations remained robust after adjusting for baseline covariates, but lost significance after additionally taking into account attention problems (**Figure 3.S6**). When considering social causation, we only found a significant association in early adolescence, with higher education around 14 predicting decreases in externalizing behaviour around 16. However, after adjusting for parental SES (**Figure 3.S6**), this association was no longer significant. We did not find any cross-lagged associations between education and externalizing behaviour in fixed effects models when using the unamended YSR/ASR scales.

Our sensitivity analyses showed very similar results, with two exceptions: First, a path from externalizing behaviour to decreases in subsequent education from around age 19 to around age 22 became borderline significant (p=0.049) in the fixed effects model with amended scales (**Figure 3.S8**). Second, additional adjustment for anxiety/depression made the path from externalizing behaviour around age 14 to decreases in education around age 16 significant in the multivariate-adjusted CLPM (**Figure 3.S6**). However, the change in coefficients was only minor. Similar to the results concerning attention problems, we did not find significant differences in cross-lagged paths between education and externalizing behaviour in males and females in fixed effects models, and allowing for sex differences in cross-lagged paths did not improve model fit (**Figure 3.S10**).

Model fit of all cross-lagged models was adequate to good (Hu & Bentler, 1999). Overall, the fixed effects CLPMs fit the data better than the CLPMs without fixed effects.

4. DISCUSSION

In this study, we aimed to evaluate the social causation and health-related selection hypotheses by modelling reciprocal relationships of externalizing behaviour and attention problems with educational level throughout adolescence and young adulthood in a selective educational system. Attention problems almost consistently predicted subsequent decreases in educational level throughout adolescence and into young adulthood (i.e., health-related selection). This result was robust to adjustment for covariates and fixed effects. We also found bivariate associations between externalizing behaviour and subsequent decreases in educational level, but these paths were no longer significant after adjusting for concurrent attention problems, and neither in the fixed effects model when using the unamended scales. When considering social causation, two time-specific associations between education and subsequent changes in attention problems or externalizing behaviour were found, but these were explained by time-stable confounders. Surprisingly, there were only few cross-sectional differences in attention problems across educational tracks.

4.1. Interpretation of findings

Our results show that attention problems are consistently associated with subsequent decreases in educational level (i.e., health-related selection) throughout adolescence and young adulthood. Our findings are consistent with previous studies that highlight the direct detrimental effects of attention problems on educational outcomes above and beyond other mental health problems (Galéra et al., 2012; Veldman et al., 2014), as well as with previously published CLPMs on GPA-based measures and attention problems (Defoe et al., 2013; Palmu et al., 2018). We add to this literature by showing that health-related selection effects may extend beyond GPA and could be sufficiently consequential for adolescents to change to a lower degree programme in the Dutch selective educational system.

Attention problems were associated with health-related selection similarly throughout all phases of adolescence and young adulthood. While symptoms associated with attention problems, such as impulsivity and hyperactivity, tend to decline with age, attention problems often persist into young adulthood (Biederman et al., 2000). This persistence, in combination with gradual increases in self-management demands, may explain why health-related selection effects hardly diminish over time (Anastopoulos et al., 2018). With extensive teacher and parental support, some adolescents may be able to attain recommendations for the higher educational tracks in primary school despite high levels

of attention problems. However, once in secondary school, many of these adolescents may struggle with the increased workload, eventually leading them to switch to a lower educational track. Other adolescents grappling with attention problems may nevertheless successfully finish the secondary school of one of the higher tracks, but run into difficulties upon entering higher education, which requires much more autonomy and self-directed learning (Anastopoulos et al., 2018). Matters are further complicated for these young adults due to a steep increase in personal responsibilities after leaving the parental home, often combined with a loss of contact with youth mental health care institutions (Anastopoulos et al., 2018; Tuomainen et al., 2018).

Unexpectedly, we found only few cross-sectional differences in attention problems across educational tracks. Only around age 14 we found lower scores in the academic track, as compared to the lowest two tracks, and around age 16 compared to all other tracks. We did not find clear differences amongst the other tracks, suggesting that the cross-sectional association between education and attention problems might not be linear in early and mid-adolescence. We found no increase in cross-sectional differences in attention problems over time, which would be expected if there is persistent downward educational mobility related to attention problems. Instead, from age 19 onwards, we no longer found an ordered relationship between educational level and attention problems. One explanation for these results could be selective attrition or missingness. Indeed, attrition analyses revealed higher dropout of adolescents from the lower educational tracks. However, attention problems were not associated with attrition, and participants with incomplete information on educational level only reported more attention problems at wave 6. This suggests that attrition or missingness may be no sufficient explanation of the scarcity of cross-sectional associations between attention problems and education from late adolescence onwards. Instead, this finding could be related to lower academic demands in combination with an earlier career choice in the vocational tracks. The theoretically focussed work in the higher educational tracks may elicit more attention problems in adolescents than the more practical work in the vocational tracks. Furthermore, adolescents in the vocational tracks are able to choose their desired profession earlier, and can subsequently train in the job that suits their individual talents best. Taken together, this could have led to an equalization in cross-sectional educational differences in self-perceived attention problems.

Regarding social causation and attention problems, we found one bivariate path from higher education around age 16 to increases in attention problems around age 19, which was driven by adolescents' IQ, and to a lesser extent parental SES. While attention problems have often been associated with lower IQ scores in the literature (Rommelse et al., 2017; Rommelse et al., 2016), there is some evidence that individuals with high IQ may also be at increased risk of attention problems (Karpinski et al., 2018). Similarly, higher parental SES is associated with both lower attention problems and higher educational attainment in the offspring (Miller et al., 2018; van Spijker et al., 2017).

When considering health-related selection related to externalizing behaviour in bivariate CLPMs, we found significant associations of externalizing behaviour with subsequent decreases in education throughout all of adolescence and up to around age 22. These associations lost significance after additionally adjusting for attention problems and were absent in fixed effects models. It is therefore likely that decreases in educational level in adolescence are not predicted by adolescents' delinquent or aggressive behaviour, but by co-occurring attention problems and associated traits, such as low effortful control, adversely affecting school performance (Beauchaine & McNulty, 2013; Snyder et al., 2015; Veronneau et al., 2014).

We found a bivariate social causation path from lower educational level to increases in externalizing behaviour in early adolescence only, which was no longer significant after further adjusting for parental SES. The propensity of the Dutch educational system to place children from lower SES households in the lower educational tracks (van Spijker et al., 2017), in combination with the higher prevalence of stress factors predictive of externalizing behaviour in these families, such as neighbourhood disadvantage, stressful life events, and unhealthy family functioning (Piotrowska et al., 2019), could thus partially explain how adolescents' educational level and externalizing behaviour become associated.

The high stability of educational track membership over the course of adolescence and young adulthood highlights the importance of the transition from primary to secondary school around age 12 in explaining educational differences in adolescents' attention problems and externalizing behaviour. Both parental SES and IQ strongly predicted adolescents' educational level in early adolescence, which is in line with previous studies showing that these characteristics are amongst the strongest determinants of adolescents' educational attainment (Brody, 1997; van Spijker et al., 2017). Low parental SES, but not IQ, predicted increased externalizing behaviour and attention problems around age 14, highlighting the importance of parental SES in adolescents' problem behaviours (Piotrowska et al., 2015; Russell et al., 2016), as explained above.

4.2. Limitations and strengths

Some limitations of this study may have affected our results and conclusions. First, we chose to operationalize adolescents' attention problems and externalizing behaviour using the empirically-based syndrome scales of the YSR/ASR, which are less closely related to diagnosable conditions than the DSM-based clinical scales. This choice was made because the empirically based scales are more comparable in the YSR and the ASR than the DSM-based scales. Different diagnoses (and hence different DSM-based scales) are used to represent externalizing behaviour in adolescents (i.e., oppositional defiant disorder and conduct disorder) and adults (i.e., antisocial personality disorder). Likewise, in adolescents with ADHD, symptoms of impulsivity and hyperactivity often diminish over time, while attention problems tend to remain more stable and often persist into adulthood (Biederman

et al., 2000). Moreover, previous research on ADHD patients has shown that attention problems are more predictive of educational outcomes than impulsivity and hyperactivity (DuPaul & Langberg, 2015). That said, the empirically based YSR and ASR scales are strongly correlated with the DSM-oriented scales of related conditions. Achenbach et al. (2003, 2005) reported correlations of the attention problems scale with the ADHD scale of r=0.91 for the ASR (Achenbach et al., 2005), and a Cohen's kappa of 0.70 for the correlation between scoring in the borderline/clinical range of the attention problems and the ADHD scale of the YSR (Achenbach et al., 2003). Although the empirically based scales of the YSR and ASR were far more comparable than the DSM-based scales, still slightly different questionnaires were used to capture attention problems and externalizing behaviour in adolescents and young adults, in order to account for developmental differences. While it is commonplace in the literature to combine these scales in longitudinal models of development (Kan et al., 2013; Peeters et al., 2019; Veldman et al., 2015), we have conducted sensitivity analyses to rule out the possibility that this affected the outcomes of the fixed effects models (Hamaker, 2018). Overall, these analyses (which used only concordant items between the YSR and ASR) yielded similar results as the main analyses.

Second, attrition might have influenced the results of our study. Although we implemented FIML to manage missing data, higher dropout of adolescents with less favourable conditions (e.g., lower education, parental SES, IQ) may still have affected our results. As these characteristics are also important determinants of adverse outcomes in young adulthood, further research on at-risk groups is necessary (Caspi et al., 2016). Third, while we have used fixed effects to address unmeasured time-invariant confounding, residual confounding may still have affected our results. For example, our fixed effects models did not take into account that unmeasured time-stable characteristics (e.g., genetics) may have time-varying effects. Further residual confounding may stem from other time-varying variables we have not measured. Fourth, while we did not find significant differences in cross-lagged associations between genders, we found increased standard errors in the multigroup models, suggesting that we might need larger samples to detect smaller differences between groups. Further studies with larger samples are needed to investigate variations in cross-lagged associations by gender, parental SES, and ethnicity.

Our study has several key strengths. First, the TRAILS study is characterized by a long follow-up (16 years) and a high response rate (Oldehinkel et al., 2015). Second, by using innovative statistical techniques we simultaneously took into account reciprocity between educational level and mental health, disentangled their temporal direction, and addressed time-invariant unmeasured confounding using fixed effects (Allison et al., 2017). Third, we add to the literature by, for the first time, modelling bidirectional associations between educational level and externalizing behaviour in a selective educational system, which provides a consistent and age-appropriate measure of educational attainment, as proxy for developing SES over the course of adolescence. The selection into educational tracks

as early as at age 11-12 years means that Dutch adolescents grow up in distinct educational environments that are characterized by different social norms, future expectations, cognitive resources, and occupational prospects (Berten et al., 2012; Van Houtte & Stevens, 2008) — characteristics that are closely related to conceptualizations of SES in adulthood (Mackenbach, 2012). One could therefore argue that in selective educational systems, such as in the Netherlands, youngsters move into 'their own' SES at a much earlier age than in comprehensive systems, such as in Finland or the USA. TRAILS provides a unique opportunity to investigate both the antecedents and consequences, in terms of health-related characteristics, of this differentiation and subsequent intragenerational social mobility in adolescents and young adults.

5. CONCLUSION AND IMPLICATIONS

In line with the health-related selection hypothesis, our findings suggest that attention problems pose a risk for decreases in educational attainment during all phases of adolescence and young adulthood, as youngsters with these kinds of problems may face substantial academic difficulties in the higher educational tracks. Although the cross-lagged paths as such may represent small effects, their cumulative effect throughout adolescence may be considerable. Our results emphasize the need for interventions to address the negative impact of attention problems on educational attainment. There is some evidence that pharmacological treatment (Langberg & Becker, 2012; Punja et al., 2016; Storebø et al., 2015) and certain non-pharmacological interventions (Evans et al., 2021; Lopez et al., 2018) may provide short-term improvements in GPA. However, only few studies have investigated to what extent these improvements are sufficient to meaningfully alter adolescents' and young adults' educational careers (Langberg & Becker, 2012, 2020). Reassuringly, a registry-based study has found that ADHD treatment was associated with a decreased risk of non-eligibility to upper secondary school in Sweden (Jangmo et al., 2019). Other studies have concluded that treatment effects may not be strong enough to address disparities in educational attainment (Fleming et al., 2017; Langberg & Becker, 2012, 2020). Future research on interventions should focus on their long-term effectiveness, effect sizes, and clinically meaningful indicators (Langberg & Becker, 2012, 2020), such as long-term educational attainment.

Regarding social causation, differences in parents' SES across educational tracks seem to contribute to educational differences in adolescents' externalizing behaviour in mid-adolescence. Interventions targeting externalizing behaviours should therefore take into account risk factors related to low family SES, such as lack of economic resources, unhealthy family functioning, stressful life events, and neighbourhood disadvantage (Piotrowska et al., 2019).

DATA AVAILABILITY

Under the General Data Protection Regulation (GDPR), our dataset is considered pseudonymized rather than anonymized, and is still regarded as personal data. When participants were invited to the cohort more than 20 years ago, they were not asked to give informed consent to make their personal data publicly available in pseudonymized form. As a result of this, legal and ethical restrictions prevent the authors from making data from the TRAILS study publicly available. Data are available upon request from the TRAILS data manager (trails@umcg.nl). Detailed information about the participation agreements with TRAILS participants is available from the ethics committee; Central Committee on Research Involving Human subjects (CCMO; tc@ccmo.nl). For more information about accessing data from the TRAILS study, please see https://www.trails.nl/en/hoofdmenu/data/data-use. The Mplus syntax for our analyses can be obtained from: https://github.com/hschmengler/Educational-level-attention-problems-and-externalizing-behaviour-in-adolescence-and-early-adulthood

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CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

ETHICAL STANDARD STATEMENT

Ethical approval for TRAILS was obtained from the Dutch national ethics committee Central Committee on Research Involving Human Subjects (#NL38237.042.11). Written informed consent was obtained from both adolescents (all waves) and their parents (first three waves) prior to each assessment wave.

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AUTHOR CONTRIBUTIONS

Heiko Schmengler: Conceptualization, Methodology, Formal analysis, Visualization, Writing - Original Draft, Writing - Review & Editing, Margot Peeters: Conceptualization, Supervision, Project administration, Writing - Review & Editing, Gonneke W. J. M. Stevens: Writing - Review & Editing, Anton E. Kunst: Writing - Review & Editing, Catharina A. Hartman: Writing - Review & Editing, Albertine J. Oldehinkel: Supervision, Writing - Review & Editing, Wilma A. M. Vollebergh: Conceptualization, Supervision, Project administration, Funding acquisition, Writing - Review & Editing.

3

CHAPTER 4

DISENTANGLING THE INTERPLAY BETWEEN GENES, COGNITIVE SKILLS, AND EDUCATIONAL LEVEL IN ADOLESCENT AND YOUNG ADULT SMOKING – THE TRAILS STUDY

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ABSTRACT

Recent studies suggest that smoking and lower educational attainment may have genetic influences in common. However, little is known about the mechanisms through which genetics contributes to educational inequalities in adolescent and young adult smoking. Common genetic liabilities may underlie cognitive skills associated with both smoking and education, such as IQ and effortful control, in line with indirect health-related selection explanations. Additionally, by affecting cognitive skills, genes may predict educational trajectories and hereby adolescents' social context, which may be associated with smoking, consistent with social causation explanations. Using data from the Dutch TRAILS study (N = 1,581), we estimated the extent to which polygenic scores (PGSs) for ever smoking regularly (PGS_{SMOK}) and years of education (PGS_{EDU}) predict IQ and effortful control, measured around age 11, and whether these cognitive skills then act as shared predictors of smoking and educational level around age 16, 19, 22, and 26. Second, we assessed if educational level mediated associations between PGSs and smoking. Both PGSs were associated with lower effortful control, and PGS_{EDU} also with lower IQ. Lower IQ and effortful control, in turn, predicted having a lower educational level. However, neither of these cognitive skills were directly associated with smoking behaviour after controlling for covariates and PGSs. This suggests that IQ and effortful control are not shared predictors of smoking and education (i.e., no indirect health-related selection related to cognitive skills). Instead, PGS_{SMOK} and PGS_{EDU}, partly through their associations with lower cognitive skills, predicted selection into a lower educational track, which in turn was associated with more smoking, in line with social causation explanations. Our findings suggest that educational differences in the social context contribute to associations between genetic liabilities and educational inequalities in smoking.

1. INTRODUCTION

Lower education has been consistently associated with higher risks of smoking over the life course (Alves et al., 2023). In selective educational systems like in the Netherlands, which are defined by an early selection into different educational tracks based on academic aptitude, educational inequalities in smoking emerge already in early adolescence (de Looze et al., 2013a). For example, in 2021, 22.5% of Dutch adolescents (mean age 13.9) in the lower vocational track reported ever experimenting with smoking and 5.5% reported daily smoking, whilst that was only the case for respectively 11.1% and 0.4% of adolescents in the academic track (Boer et al., 2022). Educational trajectories remain strongly associated with smoking after leaving the educational system, and some, but not all, studies even found increases in educational inequalities in smoking between late adolescence and young adulthood (Alves et al., 2023).

Educational inequalities in smoking are thought to emerge in the context of a complex interplay between differences in the social environment and individual differences (e.g., in genetics and cognitive skills), which is not well understood. For example, currently little is known about the mechanisms, including those related to genetically influenced phenotypes, as well as the social context, through which genetic factors may contribute to educational inequalities in smoking in adolescence and young adulthood. This is surprising, given that recent studies have found substantial genetic correlations between smoking and educational attainment (Jang et al., 2022; Quach et al., 2020; Wedow et al., 2018), which suggests that genetic variants associated with smoking and lower educational attainment to some extent overlap. This phenomenon is known as pleiotropy and means that genetic dispositions for smoking are also associated with lower educational attainment, and, similarly, genetic dispositions for lower educational attainment are also associated with smoking, inducing genetic correlations between both phenotypes (Jang et al., 2022). Genetic dispositions for observed outcomes like educational attainment or smoking can be measured with polygenic scores (PGSs). PGSs sum up the effects of a person's many genetic variants on an outcome of interest, or phenotype, using effect sizes estimated in large genome-wide association studies (GWASs) (Allegrini et al., 2022).

Two types of mechanisms may link correlated genetic risk factors for smoking and lower educational attainment to educational inequalities in smoking (i.e., phenotypic correlations between lower education and smoking behaviours). First, genetic dispositions for smoking and lower educational attainment may influence phenotypic characteristics proximally associated with both smoking and lower educational attainment, which can therefore be considered shared risk factors of both. These may include cognitive skills known to be associated with both smoking and educational outcomes, such as IQ (Brody, 1997; Daly & Egan, 2017; Weiser et al., 2010) and effortful control (Daly et al., 2016; deBlois & Kubzansky, 2016; Piehler et al., 2012; Veronneau et al., 2014). The phenomenon

that individual characteristics (e.g., genetic risk factors, cognitive skills) related to health behaviours (e.g., smoking) also influence the chance that individuals end up in a certain educational trajectory is known as indirect health-related selection in the public health literature (Mackenbach, 2012). If these characteristics are genetically influenced phenotypes directly associated with both smoking and education (in this case, cognitive skills), this mechanism is also called confounding pleiotropy in the genetics literature (Davies et al., 2019). Alternatively, and also consistent with indirect health-related selection, it is possible that genetic variants affect smoking and education through separate phenotypic mechanisms, which is known as horizontal pleiotropy (Davies et al., 2019).

In a second type of developmental mechanism, like in the first type of mechanism, genes predict into which educational trajectory adolescents are selected, including by means of predicting cognitive skills in childhood. However, unlike in the first type of mechanism, educational differences in the social context then drive associations between genetic risk factors and smoking behaviour. This is referred to as social causation in the public health literature (Mackenbach, 2012). If genetic variants predict one of the two phenotypes of interest (e.g., educational level), which in turn influences the other phenotype of interest (e.g., smoking), this is also referred to as vertical pleiotropy in the genetics literature (Davies et al., 2019). More specifically, adolescents entering the lower educational tracks are much more likely to encounter smoking peers, as classroom social norms in these tracks more strongly encourage smoking (de Looze, ter Bogt, et al., 2013; Huisman & Bruggeman, 2012; Peeters et al., 2021), making these adolescents more likely to initiate tobacco use themselves. Educational trajectories substantially predict which socio-occupational groups young adults enter (e.g., in terms of occupational class, prestige, and income) (Andersen & Van De Werfhorst, 2010; Behrens et al., 2016; Bol, 2015), and associated differences in social norms, privileges, and stressors, all of which may influence educational differences in smoking behaviour in young adulthood (Huisman et al., 2005; Schaap et al., 2008).

Consistent with both types of mechanisms, associations between PGSs for lower educational attainment and smoking, and between PGSs for smoking and lower educational attainment have been found repeatedly across studies (Hicks et al., 2021; Pasman et al., 2021; Salvatore et al., 2020; Wedow et al., 2018). These associations, which are also referred to as cross-phenotype associations in the genetics literature (Solovieff et al., 2013), may even persist after adjusting for the overlap between PGSs (Hicks et al., 2021). For example, associations between a PGS for having ever smoked regularly and educational attainment, and between a PGS for years of education and smoking remained statistically significant after mutually adjusting for both PGSs (Hicks et al., 2021). This suggests that both PGSs are uniquely related to variance in later smoking and educational attainment and thus should be considered simultaneously when investigating developmental cascades from genetic variants to educational inequalities in smoking.

Research on the developmental mechanisms contributing to the cross-phenotype associations mentioned above is limited. For example, little is known about the processes through which genetic predictors of lower educational attainment are associated with smoking. Only one study among adults found that the association between a PGS for years of education and smoking was partially explained by educational attainment, rather than differences in cognitive ability (Wedow et al., 2018). To our knowledge, this finding has thus far not been replicated in adolescents, which is an important omission given that some research suggests that the contribution of genetics and the environment to smoking behaviour may vary over the course of development (Kendler et al., 2008). Concerning associations between genetic predictors of smoking and educational outcomes, a study among adolescents found that academic motivation, disciplinary problems, and grade point average (GPA) partially mediated the association between a PGS for smoking and educational attainment. These mediating characteristics were also correlated with tobacco use (Hicks et al., 2021). Another study that focussed on associations between PGSs and cognitive skills related to educational attainment (rather than educational attainment directly) found no associations between a PGS for smoking and cognitive ability and executive functioning (Paul et al., 2022). These studies provide mixed evidence on the explanatory mechanisms contributing to cross-phenotype associations between PGSs for smoking and educational attainment. Notably, to our knowledge, no study has thus far explored the interplay between genetic liabilities for smoking, cognitive skills, and educational level in the development of educational inequalities in smoking in adolescents and young adults.

1.1. Aim

In this study, the developmental pathways through which correlated genetic dispositions are associated with educational inequalities in smoking throughout adolescence and young adulthood were investigated. Hereby, this study provides novel insights into the mechanisms underlying the phenotypic associations between educational level and smoking behaviour. We first evaluated whether cross-phenotype associations exist between a PGS for smoking and lower educational attainment, and a PGS for lower educational attainment and smoking, to determine the presence of any form of pleiotropy. Second, we studied the role of indirect health-related selection as developmental pathway linking PGSs to educational differences in smoking. To do so, we evaluated the extent to which both PGSs predict IQ and effortful control measured in childhood (around age 11), and whether these cognitive skills in turn act as shared predictors of both educational level and smoking behaviour in adolescence and young adulthood. We also evaluated whether PGSs serve as shared predictors of smoking and educational level through other mechanisms than IQ and effortful control, which might point at indirect health-related selection via phenotypic mediators we have not measured. Subsequently, we conducted

sequentially adjusted regression analyses of associations between educational level and smoking. If indirect health-related selection related to genetic influences and/or cognitive skills is present, these associations should weaken once controlled for PGSs, IQ, and effortful control. Third, we examined the extent to which associations between PGSs and smoking are mediated by the educational trajectories into which adolescents are selected based on their genetic differences, and hereby educational differences in the social context, consistent with social causation explanations. Our longitudinal approach allows us to consider all phases of adolescent development simultaneously, as the associations between genetic factors, educational level, and smoking may differ across age groups.

2. METHODS

2.1. Study population

The TRacking Adolescents' Individual Lives Survey (TRAILS) is a prospective cohort study based in the Netherlands, consisting of a population cohort recruited from primary schools (N = 2,229), and a clinical cohort of adolescents recruited from psychiatric outpatient clinics (N = 543), followed from around age 11 onwards. We used data collected during the first six (biennial or triennial) assessment waves, which spanned the period between around age 11 and age 26. The population cohort was invited from 135 schools in the provinces of Groningen, Friesland, and Drenthe, of which 122 decided to participate (de Winter et al., 2005). The clinical cohort consists of adolescents who had been referred to child and adolescent psychiatric outpatient clinics at any point in their life before age 11 for consultation or treatment. The initial response rate was 76% for the general population cohort and 43% for the clinical cohort. A detailed description of TRAILS can be obtained elsewhere (Oldehinkel et al., 2015).

2.2. Genotyping

DNA was available for N = 1,694 participants and was collected from blood samples (N = 1,334) or, in a minority of participants (N = 360), from buccal swabs (wave 3: general population cohort; wave 2: clinical cohort), and extracted using a manual salting out procedure as discussed in Miller, Dykes, and Polesky (1988). The Golden Gate Illumina BeadStation 500 and the Infinium™ HumanCytoSNP-12 v2.1 BeadChip platforms (Illumina Inc., San Diego, CA) were used for genotyping. The quality of the genotyping was checked for SNP call rate (>95%), minor allele frequency (>1%), Hardy-Weinberg equilibrium ($p>1x10^{-6}$), sample call rate (>95%), and heterozygosity (<4SD from mean). Subsequently, datasets were merged, checked for genotype concordance, and imputed using the Haplotype Reference Consortium's global reference panel on the Michigan Imputation Server (Das et al., 2016; McCarthy et al., 2016). Next, we removed participants where at least one parent

was born abroad (N = 97). This was done because PGSs based on currently available GWAS, which are mainly based on European-ancestry samples, have inferior prediction accuracy when applied to other ethnic groups (Lee et al., 2018; Mostafavi et al., 2020). Unfortunately, detailed information on participants' ancestry allowing to distinguish between participants with European and non-European migration backgrounds was not available, which is why we decided to exclude all participants with at least one parent born abroad. Lastly, for the small number of sibling pairs in TRAILS, one sibling per pair was randomly removed if genetic data were available for both (N = 16), leading to a final sample of N = 1,581 participants.

2.3. Smoking

Adolescents and young adults were asked to report on their tobacco use in the previous four weeks. Responses were recoded to approximate the average number of cigarettes smoked per day. At wave 3 (around age 16), adolescents' answers were coded as follows: 0 (non-smokers), 1 (less than one cigarette per day), 3 (1 - 5 cigarettes per day), 8 (6 - 10 cigarettes per day), 15 (11 - 20 cigarettes per day), 21 (>20 cigarettes per day). From wave 4 to wave 6 (age around 19 – 26), response options were expanded to capture heavy smoking in more detail: 0 (non-smokers), 1 (less than one cigarette per day), 3 (1 - 5 cigarettes per day), 8 (6 - 10 cigarettes per day), 15 (11 - 20 cigarettes per day), 25 (21 - 30 cigarettes per day), 31 (>30 cigarettes per day).

2.4. Cognitive skills

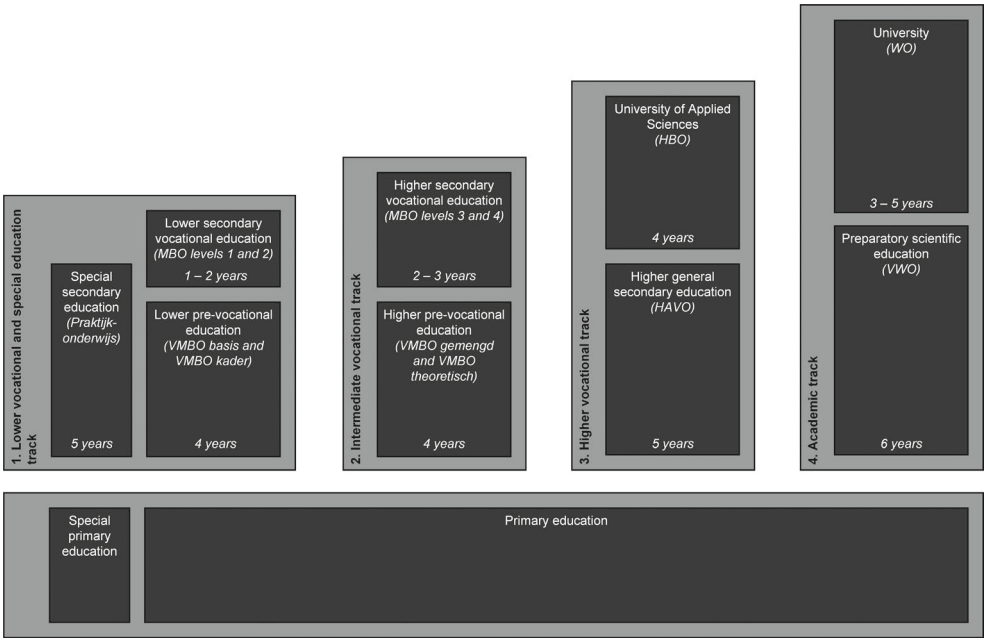
Childhood cognitive skills were captured by effortful control and the Intelligence Quotient (IQ) assessed around age 11 (wave 1). Effortful control was assessed using the corresponding subscale from the parent-report Early Adolescent Temperament Questionnaire (EATQ-R), which consists of 11 items with 5 response categories (Cronbach's alpha = 0.86) (Oldehinkel et al., 2004). IQ was estimated using the Block Design and Vocabulary subtests of the Revised Wechsler Intelligence Scale for Children (WISC-R) (Brunnekreef et al., 2007).

2.5. Educational level

The Dutch educational system is characterized by an early (age 11–12) selection into a secondary educational track, based on cognitive tests and the advice of the primary school. There are four tracks in the Dutch educational system, each consisting of a specific type of secondary school followed by tertiary education at the corresponding level (**Figure 4.1**): (1) lower vocational track, (2) intermediate vocational track, (3) higher vocational track, (4) academic track. In addition, there is a special education track, followed by students unable to attend regular education. This track was collapsed with the lower vocational track in our analyses. While in secondary education, adolescents can be recommended by their school to move between educational tracks, depending on their academic performance. Furthermore, after attaining specific milestones of their track, students can become eligible

to continue their education in the next higher track. For example, students who finish the intermediate vocational track with an MBO level 4 diploma may continue their education by attending a University of Applied Sciences of the higher vocational track. Overall, a proportion of students was mobile mainly between adjacent educational tracks: 26.70% of adolescents moved to a different track between wave 3 and 4, 26.88% between wave 4 and 5, and 12.51% between wave 5 and 6, respectively. 41.83% of participants were in a different educational track around age 26 (wave 6) than around age 16 (wave 3). Educational track membership was assessed at each wave by asking for participants' current enrolment, as well as their highest completed diploma. Participants who finished the final diploma of a given track received the value corresponding to that level for all subsequent waves, unless they continued education at a higher level.

FIGURE 4.1 The Dutch educational system



If information on current and completed education was not available at waves 3 or 4, retrospective event history calendars completed at wave 3 and wave 5 were used to ascertain adolescents' educational level at these waves. It was not possible to classify participants who had not been in education for a longer period, whose educational level was not classifiable in terms of one of the four tracks described above (e.g., because of education abroad), whose educational level was assessed incompletely, who did not respond to questions on education, or who had left the educational system permanently

(wave 3: N = 206, 13.32%; wave 4: N = 222, 14.82%, wave 5: N = 240, 16.49%, wave 6: N = 342, 24.95%). Education was considered as missing for these participants.

2.6. Polygenic scores (PGSs)

PGSs for smoking (PGS_{SMOK}) and educational attainment (PGS_{EDU}) were computed as the weighted sum of alleles using LDPred (Vilhjálmsson et al., 2015). Weights (i.e., effect sizes) for PGS_{EDU} were obtained from a large GWAS for years of schooling completed (EduYears; N = 1,131,881; 1,271 genome-wide significant loci), based on the total sample with the exception of 23andme (Lee et al., 2018). Weights for PGS_{SMOK} were calculated based on a large GWAS for having ever smoked regularly (SmkInit; N = 1,232,091; 378 associated variants), also based on the total sample with the exception of 23andme (Liu et al., 2019). In prediction analyses, EduYears was able to explain 11–13% of the variance in educational attainment (Lee et al., 2018). SmkInit accounted for approximately 4% of variance in ever smoking regularly (Liu et al., 2019). The weights were multiplied by the inverse of the linkage disequilibrium scores, as calculated by LDPred from the combined data set of the TRAILS general population and clinical cohort. The most liberal threshold (fraction of causal variants = 1.00) including all SNPs was used, in line with suggestions that this approach best captures the genetic architecture of complex phenotypes, such as education and smoking (Boyle et al., 2017). PGSs were z-score transformed to facilitate interpretability. Higher scores on PGS_{SMOK} represent higher genetic risk for smoking, and higher scores on PGS_{EDU} represent higher genetic risk for lower educational attainment.

2.7. Covariates

Environmentally mediated effects of parental genotypes on offspring phenotypes can induce 'backdoor paths' confounding associations between individual genotypes and phenotypes. This type of confounding, which is also known as dynastic effects (Morris et al., 2020; Pingault et al., 2022), was addressed by controlling for parental educational attainment and smoking. To assess parental educational attainment, the responding parent was asked about their own and their partner's highest educational attainment (wave 1), of which the mean was taken: 1 (elementary education), 2 (lower tracks of secondary education), 3 (higher tracks of secondary education), 4 (higher vocational education), 5 (university). For smoking, the responding parent was asked about their own and their partner's tobacco use in the preceding year (wave 1). Answers were recoded to 0 (neither parent smokes daily), 1 (one parent smokes daily), 2 (both parents smoke daily). We further adjusted for age at baseline, sex, and cohort type (i.e., clinical vs. general population cohort).

2.8. Missing data handling

We performed attrition analyses to evaluate the extent to which dropout may have influenced our findings. Attrition analyses showed that at wave 6 13.28% (N = 210) of our

analytic sample no longer participated in the study. Higher scores on both PGSs, lower IQ, lower effortful control, male sex, lower parental education, parental and adolescent smoking, and lower educational level were significantly related to dropout (**Table 4.S1**). Similar differences were found when comparing participants with classifiable educational level to those whose educational level could not be determined (**Table 4.S2**). Missing values were addressed using multiple imputations by chained equations under fully conditional specification (van Buuren, 2007) and under the assumption of missingness at random. 90 imputed datasets were created with 50 iterations between datasets.

2.9. Analytical approach

We conducted structural equation models (SEM) in Mplus 8.10 to represent the hypothesized relationships between PGSs, cognitive skills around age 11, and educational level and smoking from around age 16 to 26 (**Figure 4.2**). Separate models were conducted to predict educational level and smoking in each age group (i.e., around age 16, 19, 22, and 26). We first also ran separate models for each PGS, and then combined PGS_{SMOK} and PGS_{EDU} in a single model to account for their overlap and to explore whether each PGS contains variance uniquely associated with educational level and smoking. All regression coefficients were adjusted for all covariates (i.e., parental education and smoking, adolescent age at baseline, sex, and cohort type). We evaluated the potential developmental mechanisms linking PGSs to educational inequalities in smoking by computing total, direct, and indirect effects using the 'model indirect' (mediation) command in Mplus. Besides our SEM models based on **Figure 4.2**, we conducted sequentially adjusted regression models of associations between educational level and smoking around age 16, 19, 22, and 26, to explore the extent to which these associations are explained by individual differences in PGSs, IQ, and effortful control. Smoking was modelled using negative binomial regression to accommodate the zero-inflated nature of this outcome (Allison, 2012). Linear regressions were used to predict IQ, effortful control, and educational level. Non-normality was accounted for by using robust maximum likelihood (MLR).

We conducted our analyses in three steps:

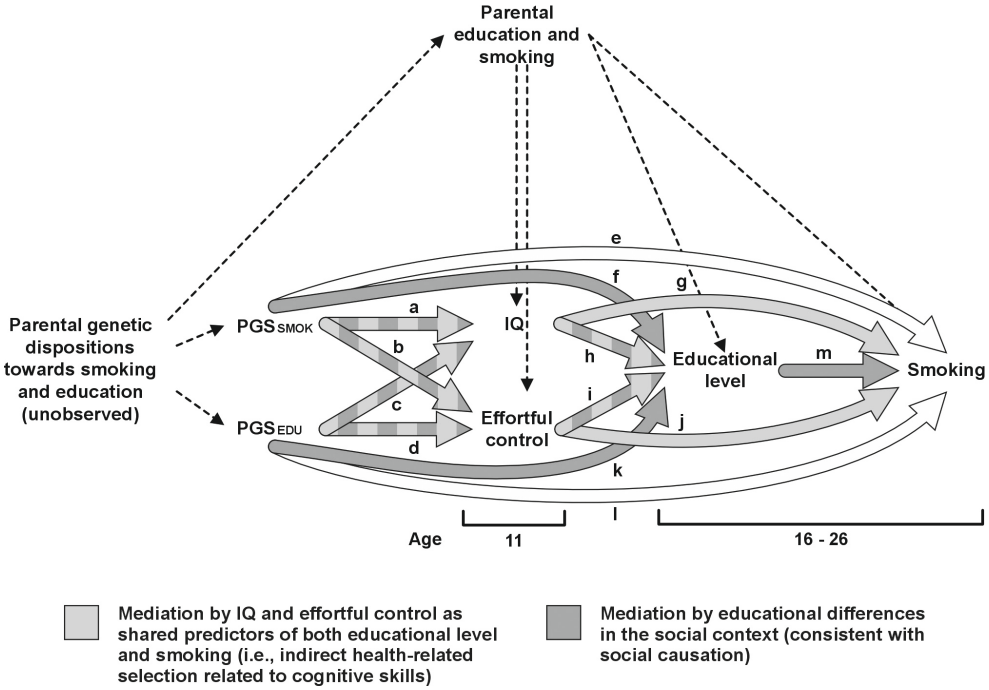
1.) To determine the existence of cross-phenotype associations (and therefore any form of pleiotropy), we calculated the total effects (i.e., combination of direct and all indirect effects) of PGS_{SMOK} and PGS_{EDU} on both smoking and educational level around age 16, 19, 22, and 26.

2.) To assess the role of indirect health-related selection, we first evaluated whether the PGSs predicted IQ and effortful control at around age 11, and whether these cognitive skills then acted as shared predictors of smoking and educational level measured around ages 16, 19, 22, and 26. We therefore compared the indirect effects of PGS_{SMOK} and PGS_{EDU} on educational level mediated by IQ and effortful control (paths a/h, b/i, c/h, d/i) to

the indirect effects of PGS_{SMOK} and PGS_{EDU} on smoking, mediated by IQ and effortful control, but not educational level (paths a/g, b/j, c/g, and d/j). Second, we evaluated direct effects of PGSs on smoking (paths e and l) and educational level (paths f and k). If significant direct effects of a PGS on both smoking and educational level are found, this could, for instance, point at indirect health-related selection through other phenotypic mechanisms than IQ or effortful control. Lastly, we explored potential changes in associations between educational level and smoking (path m) after controlling for PGSs, IQ, and effortful control in sequentially adjusted regression models.

3.) To evaluate the extent to which genes are associated with selection into different educational tracks, and hereby different social contexts, which may then predict smoking behaviour (consistent with social causation), we estimated the indirect effects of PGS_{SMOK} and PGS_{EDU} on smoking from around age 16 to 26 sequentially mediated by IQ or effortful control around age 11 and educational level, assessed at the same age as smoking (i.e., around age 16, 19, 22, and 26) (paths a/h/m, b/i/m, c/h/m, and d/i/m). We also considered indirect effects of PGSs on smoking via educational level which were not also mediated by IQ or effortful control (paths f/m and k/m).

FIGURE 4.2 Conceptual model



Dashed lines represent potential confounding paths. Additional covariates included in all regression equations (omitted from Figure 4.2 for clarity) were age at baseline, sex, and cohort type.

2.10. Sensitivity analyses

We repeated the SEM models with smoking recoded as binary variable capturing daily smoking (yes/no) using the WLSMV estimator and probit regression. These models were also able to treat educational level as ordinal variable. Currently, it is not possible to conduct mediation analysis with categorical mediators combined with outcomes modelled with negative binomial regression in Mplus. In mediation models it is usually preferable to measure mediators and outcomes in consecutive waves. However, the time lags between measurements in TRAILS are rather long (about three years). This means that adolescents are frequently in different social contexts in one wave compared to the next. To adequately assess the consequences of educational level in terms of smoking, a fairly short time interval between measurements is preferable. This is why we modelled educational level and smoking contemporaneously in our main analyses. Nevertheless, we conducted sensitivity analyses in which we allowed for one wave time lag between measurements of educational level and smoking, to assess whether the choice of time lags affected our results. Furthermore, we assessed whether results differed if the analytical sample was restricted to participants of the general population cohort (N = 1,248). Lastly, we conducted a complete case analysis to assess how our way of handling missing data affected results.

3. RESULTS

3.1. Descriptive statistics

Descriptive statistics of the study sample are presented in **Table 4.1**. Adolescents' tobacco use increased from around age 16 to around age 22, and decreased again from around age 22 to 26. Lower educational level was consistently associated with more smoking in all age groups. The correlation between PGS_{SMOK} and PGS_{EDU} was r = 0.29.

3.2. Structural equation models

3.2.1. Cross-phenotype associations

As expected, both PGS_{SMOK} and PGS_{EDU} were strongly associated with their respective phenotypes, as indicated by significant total effects on smoking and, respectively, educational level in all age groups (**Tables 4.2 and 4.5**). Associations between PGS_{SMOK} and smoking were similar around age 16 and 19, increased slightly by around age 22, and then decreased again by around age 26. Associations between PGS_{EDU} and educational level were similar at all measurement occasions. Significant cross-phenotype associations were also found (**Table 4.3 and 4.4**). PGS_{EDU} had significant total effects on smoking at all measurement occasions, which decreased in strength as adolescents became young adults and remained significant after controlling for PGS_{SMOK}. Similarly, PGS_{SMOK} had significant total

effects on educational level over the entire follow up, which increased between around age 19 and 22. However, the weaker total effects of PGS_{SMOK} on education around age 16 and 19 did not survive adjustment for PGS_{EDU}.

TABLE 4.1 Characteristics of adolescents and young adults in the TRAILS study

	Wave 1		Wave 3		Wave 4		Wave 5		Wave 6	
N participants	1,581		1,547		1,498		1,455		1,371	
Male sex, N (%)	821	(51.93)	798	(51.58)	770	(51.40)	748	(51.41)	690	(50.33)
Age, mean (SD)	11.08	(0.54)	16.15	(0.68)	19.02	(0.60)	22.17	(0.66)	25.70	(0.65)
Parental education, mean (SD)	3.15	(0.88)								
Parental smoking, mean (SD)	0.64	(0.77)								
Clinical cohort, N (%)	333	(21.06)								
IQ, mean (SD)	99.71	(14.67)								
Effortful control, mean (SD)	3.11	(0.72)								
PGS_{EDU}, mean (SD)	0.00	(1.00)								
PGS_{SMOK}, mean (SD)	0.00	(1.00)								
Correlation PGS_{EDU} and PGS_{SMOK}	0.29									
Educational level, N (%)										
Lower vocational & special education			322	(24.01)	162	(12.70)	128	(10.53)	82	(7.97)
Intermediate vocational			372	(27.74)	441	(34.56)	322	(26.50)	257	(24.98)
Higher vocational			303	(22.60)	364	(28.53)	478	(39.34)	398	(38.68)
Academic			344	(25.65)	309	(24.22)	287	(23.62)	292	(28.38)
Smoking, mean (SD)										
All levels			2.30	(5.13)	3.42	(6.55)	3.57	(6.67)	2.65	(5.47)
Lower vocational & special education			4.14	(6.68)	6.57	(9.45)	8.09	(10.21)	6.08	(8.08)
Intermediate vocational			2.35	(5.10)	3.75	(6.42)	4.66	(6.93)	3.02	(5.61)
Higher vocational			1.23	(3.43)	2.01	(4.57)	2.24	(4.79)	1.91	(4.19)
Academic			0.70	(2.73)	1.21	(3.48)	1.18	(3.53)	0.72	(2.67)

SD = standard deviation.

TABLE 4.2 Total, direct, and indirect effects of PGS_{SMOK} on smoking around age 16, 19, 22, and 26 in the TRAILS study; potential mediators were measured around age 11 (IQ and effortful control) and concurrently with smoking (educational level); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Smoking around age 16	**Smoking around age 19**	**Smoking around age 22**	**Smoking around age 26**
Model 1				
Total effect	**0.375 (0.075), <0.001**	**0.358 (0.064), <0.001**	**0.423 (0.067), <0.001**	**0.306 (0.069), <0.001**
Direct effect	**0.322 (0.074), <0.001**	**0.296 (0.062), <0.001**	**0.353 (0.065), <0.001**	**0.233 (0.067), <0.001**
Total indirect effect	**0.053 (0.016), 0.001**	**0.062 (0.017), <0.001**	**0.071 (0.016), <0.001**	**0.073 (0.018), <0.001**
Specific indirect effects				
PGS_{SMOK}→effortful control→smoking	0.008 (0.008), 0.345	0.008 (0.006), 0.201	0.009 (0.006), 0.140	0.009 (0.007), 0.165
PGS_{SMOK}→IQ→smoking	-0.001 (0.003), 0.641	-0.001 (0.002), 0.655	0.000 (0.001), 0.960	0.000 (0.002), 0.839
PGS_{SMOK}→education→smoking	**0.030 (0.012), 0.016**	**0.038 (0.013), 0.004**	**0.047 (0.013), <0.001**	**0.049 (0.015), 0.001**
PGS_{SMOK}→effortful control→education→smoking	**0.014 (0.005), 0.004**	**0.014 (0.005), 0.003**	**0.012 (0.004), 0.002**	**0.012 (0.004), 0.003**
PGS_{SMOK}→IQ→education→smoking	0.003 (0.005), 0.543	0.003 (0.006), 0.542	0.003 (0.005), 0.543	0.003 (0.005), 0.543
Model 2				
Total effect	**0.322 (0.075), <0.001**	**0.299 (0.064), <0.001**	**0.384 (0.068), <0.001**	**0.266 (0.069), <0.001**
Direct effect	**0.295 (0.075), <0.001**	**0.265 (0.063), <0.001**	**0.345 (0.066), <0.001**	**0.224 (0.068), 0.001**
Total indirect effect	0.027 (0.015), 0.062	**0.033 (0.015), 0.028**	**0.039 (0.016), 0.013**	**0.042 (0.017), 0.012**
Specific indirect effects				
PGS_{SMOK}→effortful control→smoking	0.005 (0.006), 0.454	0.005 (0.005), 0.284	0.007 (0.005), 0.181	0.007 (0.005), 0.205
PGS_{SMOK}→IQ →smoking	0.004 (0.005), 0.353	0.003 (0.004), 0.364	0.000 (0.003), 0.951	0.001 (0.003), 0.690
PGS_{SMOK}→education→smoking	0.016 (0.011), 0.158	0.023 (0.012), 0.060	**0.029 (0.012), 0.019**	**0.032 (0.014), 0.022**
PGS_{SMOK}→effortful control→education→smoking	**0.010 (0.004), 0.022**	**0.010 (0.004), 0.018**	**0.009 (0.004), 0.017**	**0.009 (0.004), 0.019**
PGS_{SMOK}→IQ→education→smoking	-0.007 (0.005), 0.157	-0.008 (0.005), 0.150	-0.006 (0.004), 0.149	-0.006 (0.004), 0.151

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{EDU}.

TABLE 4.3 Total, direct, and indirect effects of PGS_{EDU} on smoking around age 16, 19, 22, and 26 in the TRAILS study; potential mediators were measured around age 11 (IQ and effortful control) and concurrently with smoking (educational level); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Smoking around age 16	**Smoking around age 19**	**Smoking around age 22**	**Smoking around age 26**
Model 1				
Total effect	**0.338 (0.076), <0.001**	**0.330 (0.058), <0.001**	**0.255 (0.057), <0.001**	**0.234 (0.066), <0.001**
Direct effect	**0.227 (0.076), 0.003**	**0.214 (0.060), <0.001**	0.112 (0.059), 0.057	0.097 (0.069), 0.158
Total indirect effect	**0.111 (0.026), <0.001**	**0.116 (0.022), <0.001**	**0.142 (0.023), <0.001**	**0.136 (0.027), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→smoking	0.008 (0.010), 0.423	0.009 (0.007), 0.194	0.013 (0.007), 0.069	0.011 (0.008), 0.152
PGS_{EDU}→IQ→smoking	-0.031 (0.020), 0.129	-0.023 (0.015), 0.142	-0.003 (0.015), 0.834	-0.013 (0.017), 0.469
PGS_{EDU}→education→smoking	**0.068 (0.018), <0.001**	**0.067 (0.016), <0.001**	**0.081 (0.017), <0.001**	**0.085 (0.020), <0.001**
PGS_{EDU}→effortful control→education→smoking	**0.018 (0.006), 0.002**	**0.017 (0.005), 0.001**	**0.015 (0.004), 0.001**	**0.015 (0.005), 0.001**
PGS_{EDU}→IQ→education→smoking	**0.048 (0.011), <0.001**	**0.045 (0.009), <0.001**	**0.036 (0.007), <0.001**	**0.038 (0.008), <0.001**
Model 2				
Total effect	**0.272 (0.077), <0.001**	**0.262 (0.061), <0.001**	**0.171 (0.059), 0.004**	**0.179 (0.067), 0.007**
Direct effect	**0.172 (0.077), 0.026**	**0.152 (0.062), 0.015**	0.036 (0.061), 0.551	0.050 (0.070), 0.478
Total indirect effect	**0.099 (0.026), <0.001**	**0.110 (0.022), <0.001**	**0.134 (0.024), <0.001**	**0.129 (0.026), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→smoking	0.007 (0.009), 0.444	0.007 (0.006), 0.247	0.009 (0.006), 0.148	0.010 (0.007), 0.167
PGS_{EDU}→IQ→smoking	-0.026 (0.021), 0.227	-0.019 (0.016), 0.229	-0.001 (0.016), 0.956	-0.008 (0.018), 0.664
PGS_{EDU}→education→smoking	**0.059 (0.017), <0.001**	**0.061 (0.016), <0.001**	**0.075 (0.017), <0.001**	**0.076 (0.019), <0.001**
PGS_{EDU}→effortful control→education→smoking	**0.014 (0.005), 0.007**	**0.014 (0.005), 0.005**	**0.012 (0.004), 0.004**	**0.012 (0.004), 0.006**
PGS_{EDU}→IQ → education→smoking	**0.046 (0.011), <0.001**	**0.047 (0.010), <0.001**	**0.039 (0.008), <0.001**	**0.039 (0.008), <0.001**

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{SMOK}.

TABLE 4.4 Total, direct, and indirect effects of PGS_{SMOK} on educational level around age 16, 19, 22, and 26 in the TRAILS study; potential mediators were measured around age 11 (IQ and effortful control); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Educational level around age 16	**Educational level around age 19**	**Educational level around age 22**	**Educational level around age 26**
Model 1				
Total effect	**-0.094 (0.026), <0.001**	**-0.091 (0.023), <0.001**	**-0.105 (0.023), <0.001**	**-0.107 (0.024), <0.001**
Direct effect	**-0.060 (0.021), 0.005**	**-0.062 (0.019), 0.001**	**-0.079 (0.020), <0.001**	**-0.082 (0.022), <0.001**
Total indirect effect	**-0.034 (0.015), 0.019**	**-0.029 (0.012), 0.019**	**-0.025 (0.011), 0.017**	**-0.024 (0.011), 0.020**
Specific indirect effects				
PGS_{SMOK}→effortful control→educational level	**-0.028 (0.008), 0.001**	**-0.023 (0.007), 0.001**	**-0.021 (0.006), 0.001**	**-0.020 (0.006), 0.001**
PGS_{SMOK}→IQ→educational level	-0.007 (0.011), 0.538	-0.006 (0.009), 0.539	-0.005 (0.008), 0.539	-0.005 (0.008), 0.539
Model 2				
Total effect	-0.038 (0.026), 0.149	-0.043 (0.023), 0.062	**-0.055 (0.023), 0.017**	**-0.057 (0.024), 0.019**
Direct effect	-0.033 (0.022), 0.133	**-0.039 (0.020), 0.048**	**-0.051 (0.021), 0.014**	**-0.054 (0.022), 0.015**
Total indirect effect	-0.005 (0.014), 0.725	-0.004 (0.012), 0.724	-0.004 (0.010), 0.668	-0.004 (0.010), 0.723
Specific indirect effects				
PGS_{SMOK}→effortful control→educational level	**-0.020 (0.008), 0.010**	**-0.017 (0.007), 0.010**	**-0.015 (0.006), 0.011**	**-0.014 (0.006), 0.011**
PGS_{SMOK}→IQ→educational level	0.016 (0.010), 0.134	0.013 (0.009), 0.135	0.011 (0.007), 0.136	0.011 (0.007), 0.137

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{EDU}.

TABLE 4.5 Total, direct, and indirect effects of PGS_{EDU} on educational level around age 16, 19, 22, and 26 in the TRAILS study; potential mediators were measured around age 11 (IQ and effortful control); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Educational level around age 16	**Educational level around age 19**	**Educational level around age 22**	**Educational level around age 26**
Model 1				
Total effect	**-0.256 (0.026), <0.001**	**-0.220 (0.023), <0.001**	**-0.230 (0.023), <0.001**	**-0.230 (0.025), <0.001**
Direct effect	**-0.131 (0.022), <0.001**	**-0.114 (0.020), <0.001**	**-0.142 (0.021), <0.001**	**-0.142 (0.023), <0.001**
Total indirect effect	**-0.125 (0.015), <0.001**	**-0.106 (0.013), <0.001**	**-0.089 (0.011), <0.001**	**-0.088 (0.011), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→educational level	**-0.034 (0.009), <0.001**	**-0.029 (0.008), <0.001**	**-0.025 (0.007), <0.001**	**-0.024 (0.006), <0.001**
PGS_{EDU}→IQ→educational level	**-0.091 (0.011), <0.001**	**-0.077 (0.010), <0.001**	**-0.063 (0.009), <0.001**	**-0.063 (0.009), <0.001**
Model 2				
Total effect	**-0.246 (0.027), <0.001**	**-0.209 (0.024), <0.001**	**-0.217 (0.024), <0.001**	**-0.215 (0.026), <0.001**
Direct effect	**-0.122 (0.023), <0.001**	**-0.104 (0.021), <0.001**	**-0.129 (0.022), <0.001**	**-0.128 (0.024), <0.001**
Total indirect effect	**-0.124 (0.016), <0.001**	**-0.105 (0.013), <0.001**	**-0.088 (0.012), <0.001**	**-0.087 (0.012), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→educational level	**-0.029 (0.009), 0.001**	**-0.024 (0.008), 0.001**	**-0.021 (0.007), 0.001**	**-0.020 (0.006), 0.002**
PGS_{EDU}→IQ→educational level	**-0.095 (0.012), <0.001**	**-0.081 (0.010), 0.010**	**-0.067 (0.009), <0.001**	**-0.067 (0.009), <0.001**

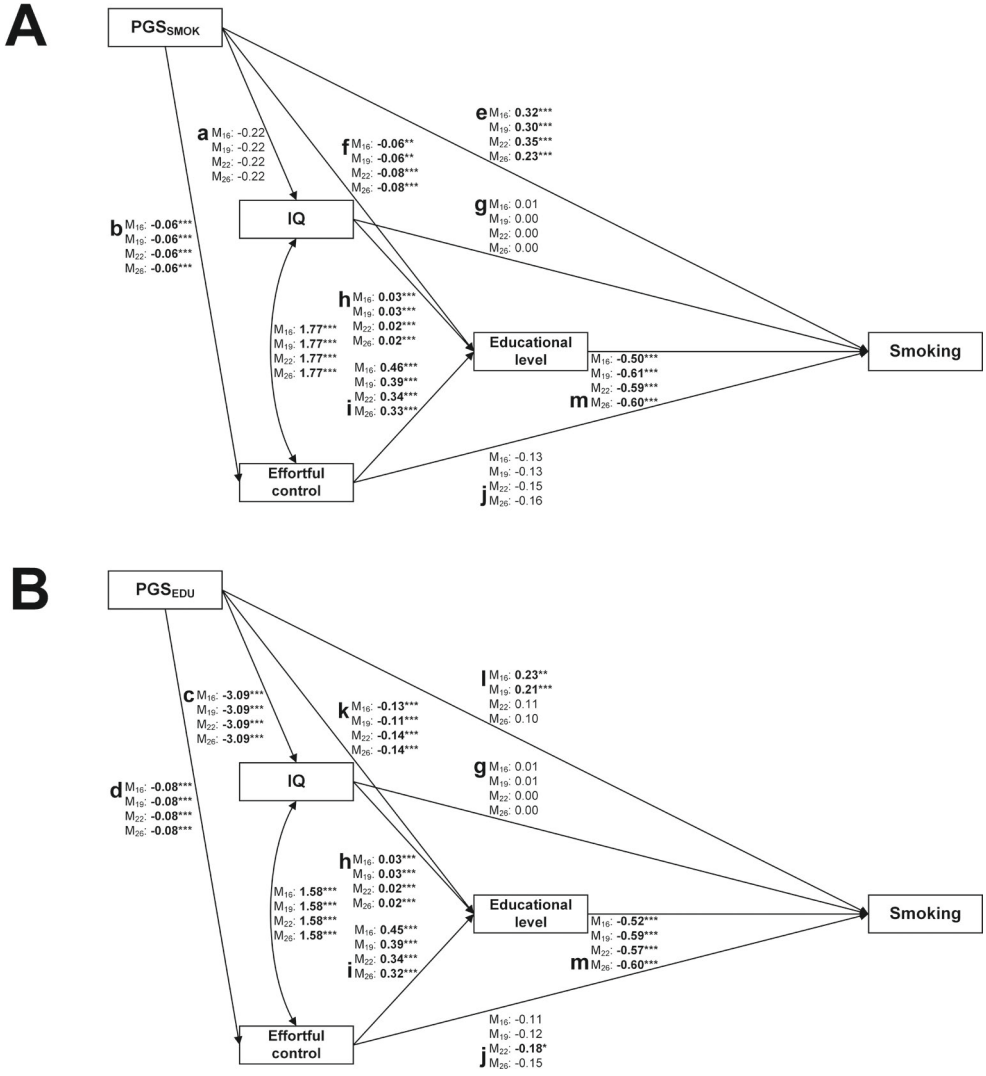
Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{SMOK}.

3.2.2. PGSs and cognitive skills as shared predictors of smoking and education

PGS_{SMOK} and PGS_{EDU} significantly predicted lower effortful control (paths b and d), whereas PGS_{EDU}, but not PGS_{SMOK}, also predicted lower IQ (path c) (**Figures 4.3-4**). Both higher IQ and effortful control were, in turn, associated with a higher educational level (paths h and i). Accordingly, we found significant indirect effects of PGS_{EDU} on lower education via both lower IQ and effortful control (paths c/h and d/i), and of PGS_{SMOK} on lower education via lower effortful control only (path b/i) (**Table 4.4-5)**. However, when considering direct associations between cognitive skills and smoking, we found only one significant direct association between higher effortful control and less smoking around age 22 (path j) when controlling for PGS_{EDU} but not PGS_{SMOK} (**Figure 4.3B**). This association did not lead to any significant indirect effects of PGS_{EDU} on smoking via effortful control but not educational level (path d/j) (**Table 4.3**). We found no direct associations of IQ with smoking, and therefore also no significant indirect effects of PGSs on smoking via IQ besides those via educational level (path c/h/m) (**Tables 4.2 and 4.3**).

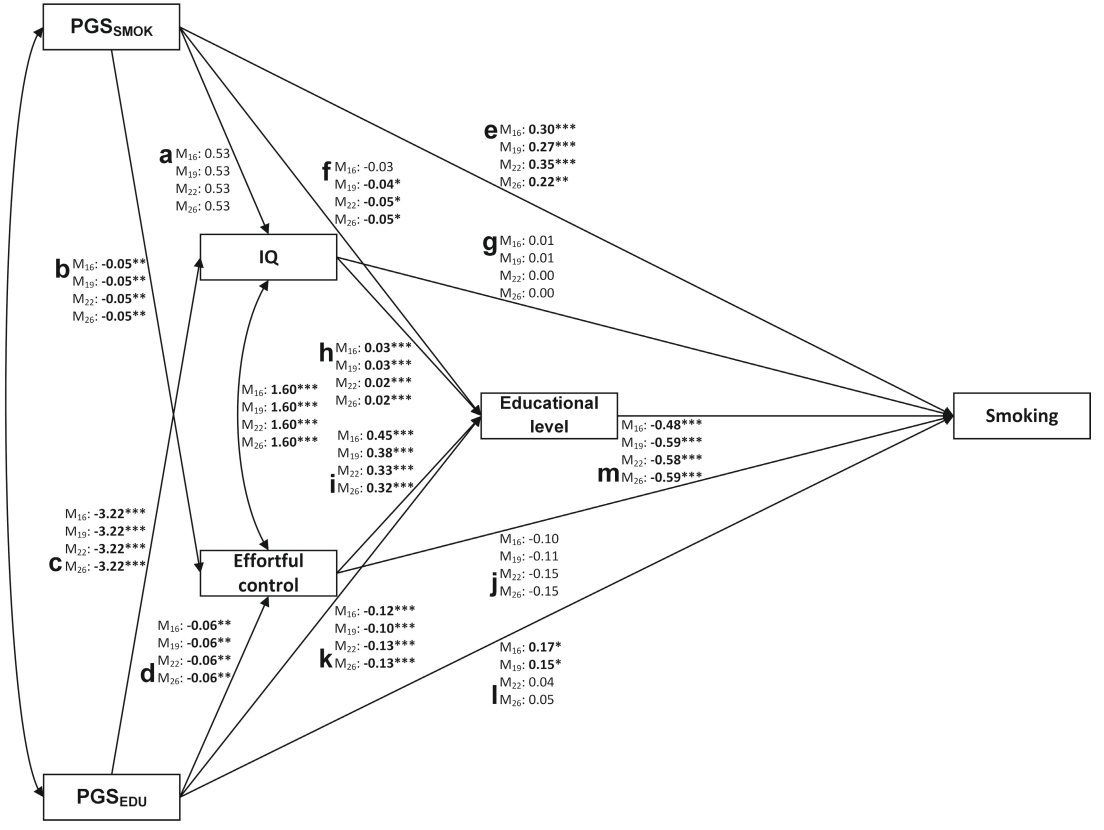
We found significant direct effects of PGS_{SMOK} and PGS_{EDU} on both smoking and educational level, suggesting that both PGSs serve as shared predictors of smoking and education through mechanisms other than IQ and effortful control. PGS_{EDU} had significant direct effects on educational level in all age groups (**Table 4.5**), as well as on smoking around age 16 and 19 (**Table 4.3**). PGS_{SMOK} had very large direct effects on smoking (**Table 4.2**), and smaller direct effects on educational level in all age groups, be it that the direct effect of PGS_{SMOK} on educational level around age 16 was no longer significant after adjusting for PGS_{EDU} (**Table 4.4**). Lastly, sequentially adjusted regression models (**Table 4.S3**) revealed only very minor changes in associations between lower educational level and smoking, with the largest reduction in the association between educational level and smoking around age 16 after controlling for PGSs. In the fully adjusted models, the association between educational level and smoking increased from about age 16 to 19 and remained stable from age 19 onwards.

FIGURE 4.3 The associations of PGS_{SMOK} (A) and PGS_{EDU} (B) with smoking around age 16, 19, 22, and 26 in separate models; potential mediators were IQ and effortful control (around age 11) and educational level measured concurrently with smoking; linear and negative binomial regression models (MLR estimator; beta-coefficient)



** p<0.05; **p<0.01; ***p<0.001. All regressions were adjusted for age, sex, cohort type, parental education, and parental smoking. Separate models were used to predict smoking around age 16 (M_{16}), 19 (M_{19}), 22 (M_{22}), and 26 (M_{26}). Educational level was measured concurrently with smoking.*

FIGURE 4.4 The associations of PGS_{EDU} and PGS_{SMOK} with smoking around age 16, 19, 22, and 26 in models combining both PGSs; potential mediators were IQ and effortful control (around age 11) and educational level measured concurrently with smoking; linear and negative binomial regression models (MLR estimator; beta-coefficient)



** p<0.05; **p<0.01; ***p<0.001. All regressions were adjusted for age, sex, cohort type, parental education, and parental smoking. Separate models were used to predict smoking around age 16 (M_{16}), 19 (M_{19}), 22 (M_{22}), and 26 (M_{26}). Educational level was measured concurrently with smoking.*

3.2.3. PGSs as predictors of educational trajectories, and educational trajectories as predictors of smoking

Large proportions of the associations between PGS_{EDU} and smoking were mediated by being in a lower educational track. The proportions mediated increased from less than 40% around age 16 to around 60-80% in young adulthood (**Table 4.3**). PGS_{EDU} was strongly associated with a lower IQ and less effortful control in childhood (paths c and d), which then predicted selection into a lower educational track (paths h and i). PGS_{EDU} also significantly predicted selection into a lower educational track via other, unmeasured mechanisms (path k). Having a lower educational level, in turn, predicted more smoking behaviour (path m). Accordingly, all indirect effects of PGS_{EDU} on smoking via educational level were significant throughout adolescence and young adulthood (i.e., paths c/h/m, d/i/m, and k/m), and remained so after adjusting for PGS_{SMOK} (**Table 4.3**).

Mediation by educational level was also present for the associations between PGS_{SMOK} and smoking, but the corresponding indirect effects tended to be smaller than the indirect effects of PGS_{EDU} on smoking via educational level, and not always remained significant after adjusting for PGS_{EDU} (**Table 4.2**). PGS_{SMOK} predicted lower effortful control (path b), which was subsequently associated with being in a lower educational track (path i). PGS_{SMOK} was also associated with lower education via other mechanisms than IQ or effortful control that we have not measured (path f). Lower educational level in turn predicted increased risks of smoking (path m), as described above. Accordingly, indirect effects of PGS_{SMOK} on smoking via lower effortful control and lower education (path b/i/m), as well as via other unmeasured predictors of lower education were found in all age groups (path f/m) (**Table 4.2**). After adjusting for PGS_{EDU}, indirect effects of PGS_{SMOK} via educational level but not effortful control on smoking around age 16 and 19 were no longer significant (path f/m), while the indirect effects via effortful control and educational level remained significant (path b/i/m). Also, the total indirect effect (i.e., indirect effect via IQ, effortful control, and educational level combined) of PGS_{SMOK} on smoking around age 16 was no longer significant after adjusting for overlap with PGS_{EDU}.

3.3. Sensitivity analyses

Analyses with education evaluated as ordinal variable and daily smoking (yes/no) as outcome (**Figures 4.S1-S2, Tables 4.S4-S7**), those with smoking measured one wave after educational level (**Figures 4.S3-S4, Tables 4.S8-S9**), and those restricted to participants of the general population cohort were largely consistent with our main analyses (**Figures 4.S5-S6, Tables 4.S10-S13**). We found some significant direct associations between cognitive skills and smoking in models evaluating PGSs separately, which all lost significance once both PGSs were included in the models. Only in the complete case analysis we found a weakly significant direct association (p=0.044) of IQ on smoking around age 16 that survived adjustment for both PGSs, be it that this association did not result in a significant

indirect effect of either PGS on smoking. Otherwise, the complete case analyses were mostly consistent with our main results (**Figures 4.S7-S8, Tables 4.S14-S17**).

4. DISCUSSION

4.1. Summary of results

We investigated the developmental pathways through which genetic dispositions contribute to educational inequalities in smoking throughout adolescence and young adulthood. Genetic vulnerability for smoking (PGS_{SMOK}) was associated with having a lower educational level throughout adolescence and young adulthood. Similarly, a PGS for having a lower educational attainment (PGS_{EDU}) was associated with smoking in all age groups. Associations between PGS_{SMOK} and lower education strengthened, while associations between PGS_{EDU} and smoking weakened as adolescents became young adults. Most of these associations remained significant after mutually adjusting for both PGSs. Whereas PGS_{SMOK} and PGS_{EDU} were both significantly associated with lower effortful control, and PGS_{EDU} also with lower IQ, direct associations between cognitive skills and smoking were absent once controlled for covariates and both PGSs. This suggests that cognitive skills largely do not serve as shared predictors of educational level and smoking. Accordingly, changes in associations between educational level and smoking after adjusting for IQ and effortful control were negligible, suggesting that the role of indirect health-related selection related to these variables may be minor. Instead, associations between both PGSs and smoking seemed partially driven by educational differences in the social context. Partly through associations with lower cognitive skills, PGS_{EDU} and PGS_{SMOK} predicted selection into a lower educational track, which in turn predicted increased smoking behaviour, consistent with social causation explanations.

4.2. Interpretation of findings

Most of our results do not support the indirect health-related selection hypothesis in relation to cognitive skills. Whilst both IQ and effortful control were strongly associated with educational level, neither of these cognitive skills showed significant direct associations with smoking behaviour once controlled for covariates and both PGSs. Past studies on direct associations of cognitive skills with smoking have yielded inconsistent results, with some research still finding substantial independent associations after adjusting for educational level (Daly & Egan, 2017; Davies et al., 2017; Sanderson et al., 2019). Differences in time lags between assessments of cognitive skills, educational level, and smoking, as well as in the measures used to capture educational level and cognitive skills may have contributed to this heterogeneity in results. Future research could focus on further disentangling under what conditions measures of cognitive skills remain associated with

smoking, net of differences in the social context. It is also possible that other genetically influenced aspects of impulsivity, such as sensation-seeking, are stronger proximal risk factors for smoking than effortful control (Mitchell & Potenza, 2014).

Consistent with the idea that there may be other genetically influenced phenotypic shared predictors of both smoking behaviour and education than IQ and effortful control, we found small reductions in associations between lower education and smoking after controlling for PGSs, as well as some significant direct effects of PGSs simultaneously on smoking and educational level. It is also possible that genetic variants influence smoking and educational attainment through separate phenotypic mechanisms (i.e., horizontal pleiotropy) (Davies et al., 2019). Taken together, our results suggests that indirect health-related selection related to unmeasured genetically influenced mediators may still play a role. It is, however, important to note that due to, among other reasons, the limited power of GWAS, currently PGSs capture only part of the genetic variance predictive of smoking and educational attainment (Pingault et al., 2021). Therefore, statistical adjustment for PGSs likely underestimates the contribution of genetics to associations between educational level and smoking.

We found that educational level substantially mediated the associations between PGS_{EDU} and smoking behaviour, and to a lesser extent the associations between PGS_{SMOK} and smoking behaviour. These indirect effects were partly driven by lower cognitive skills in childhood, which predicted selection into a lower educational trajectory. Being in a lower educational trajectory was, in turn, associated with more smoking behaviour, consistent with social causation explanations. Genes, by affecting selection into educational tracks, strongly predict adolescents' options for friendship formation (Huisman & Bruggeman, 2012), exposure to substance use-related social norms, stressors, and perceived future prospects, which may all be related to smoking (Elstad, 2010). Lower educational tracks are more frequently characterized by a culture of futility (Van Houtte & Stevens, 2008) and lower prospects with respect to job/income, potentially leading to increased short-term orientation, and seeking alternative means to attain status amongst peers, which may include risk behaviours like substance use (Elstad, 2010). Correspondingly, classrooms in the vocational educational tracks more strongly feature popularity norms endorsing smoking, and these norms in turn predict adolescents' tobacco use within classrooms (Peeters et al., 2021). The importance of peer effects is further highlighted by social network research demonstrating that the influence of friends on smoking remains strong, even after controlling for friendship selection processes, and that friendship network effects contribute to educational differences in adolescent smoking (Huisman & Bruggeman, 2012). We found that the association between lower education and smoking increased between around age 16 and 19, which is consistent with previous research also showing increases in educational inequalities in smoking towards young adulthood (Alves et al., 2023; Widome et al., 2013). Future research could explore how the transition from education to adult work

roles, which often takes place earlier in young adults who followed the vocational tracks (de Looze et al., 2013b), may contribute to these increases.

4.3. Strengths and limitations

Key strengths of our study are its high response rate, long follow-up, and consistency of measures over time, allowing to capture multiple developmental periods simultaneously (Oldehinkel et al., 2015). In particular, the selective educational system of the Netherlands provided us a consistent and age-appropriate measure of educational attainment, as proxy for developing socioeconomic status (SES). That is, the selection into educational tracks as early as at age 11-12 years means that Dutch adolescents grow up in distinct educational environments that are characterized by different social norms, future expectations, cognitive resources, and occupational prospects—characteristics that are closely related to conceptualizations of SES in adulthood. Furthermore, we used very large GWAS for both smoking and educational attainment to calculate PGSs, which (unlike PGSs based on older GWAS) predict similar amounts of variance in phenotypes as many environmental predictors (Lee et al., 2018; Liu et al., 2019). Our longitudinal approach allowed to study differences in associations between genetic propensities, educational level, and smoking behaviours over the course of development, an area that has not been investigated much in past research. Lastly, we were able to address dynastic effects as potential source of confounding by controlling for smoking and educational attainment in parents.

Nevertheless, our study has several limitations. First, attrition and missing data may have affected our results. While we addressed missing data with multiple imputations (van Buuren, 2007), selective missingness in participants with lower educational level, more tobacco use, higher genetic risk for smoking and lower educational attainment, lower parental SES, and lower IQ and effortful control (**Tables 4.S1 and 4.S2**) may still have influenced our results. Second, to achieve a sufficient sample size, we combined participants from a clinical with a general population cohort, which means that the study sample included more participants with diagnosable psychiatric conditions than would be expected in a representative population-based sample of adolescents. We addressed this issue by including cohort type as covariate in all analyses and by conducting sensitivity analyses that restricted the sample to participants of the general population cohort. Third, we did not control for past levels of smoking in our analyses, as our approach was not focussed on modelling changes in smoking over the course of adolescence, but instead sought to gain insight into the contribution of correlated genetic risk factors to the phenotypic correlations between smoking and educational level that emerge over the course of adolescence. Accordingly, our models could not consider reverse causality in the associations between educational level and smoking. While reverse causation may be less plausible given that nicotine is not intoxicating, smoking could still to some extent be

associated with decreases in education, for instance due to long-term effects of nicotine on the developing brain (Yuan et al., 2015).

Fourth, our sample did not include any participants of non-Dutch ethnicity, as large-scale GWAS are currently unavailable for non-European ethnic groups. PGSs based on GWAS from European-ancestry samples tend to have inferior prediction accuracy when applied to other ethnic groups (Lee et al., 2018; Mostafavi et al., 2020), as the frequency of causal alleles and the extent of linkage disequilibrium of SNPs with causal sites differ across populations (Mostafavi et al., 2020). Multiethnic GWAS are necessary to improve external validity, and so is further research on the portability of PGSs across (sub-)populations. Fifth, currently, PGSs capture only part of the genetic variance predictive of smoking and educational attainment. This means that we have only a partial view on the contribution of genetics to the association between adolescent educational level and smoking (Pingault et al., 2021; Wray et al., 2014). At the same time, by providing an individual-level summary measure of the level of genetic risk for a given phenotype, PGSs give a unique opportunity to gain novel insights into the developmental cascades linking correlated genetic risk factors for smoking and lower educational attainment to later educational differences in smoking.

5. CONCLUSION

Correlated genetic liabilities for smoking and lower educational attainment were significantly associated with both smoking and lower education throughout adolescence and in young adulthood. There was little support for an indirect pathway through cognitive skills (i.e., IQ and effortful control) subsequently acting as shared predictors of educational level and smoking (i.e., no indirect health-related selection related to cognitive skills). Instead, PGSs predicted, partly via their associations with lower cognitive skills, selection into a lower educational trajectory, which in turn predicted more smoking. Our findings shed further light on how social conditions, such as educational differences in the classroom context, add to the genetic relationship between smoking and lower educational attainment. The social contexts in the lower educational tracks (e.g., social norms, peer group composition, social stressors) may therefore be an important target for interventions.

DATA AVAILABILITY

Under the General Data Protection Regulation (GDPR), our dataset is considered pseudonymized rather than anonymized, and is regarded as personal data. When participants were invited to the cohort more than 20 years ago, they were not asked to give informed consent to make their personal data publicly available in pseudonymized form. As a result of this, legal and ethical restrictions prevent the authors from making data from the TRAILS study publicly available. Data are available upon request from the TRAILS data manager (trails@umcg.nl). Detailed information about the participation agreements with TRAILS participants is available from the ethics committee; Central Committee on Research Involving Human subjects (CCMO; tc@ccmo.nl). For more information about accessing data from the TRAILS study, please see https://www.trails.nl/en/hoofdmenu/data/data-use. The syntax for our analyses can be obtained from: https://github.com/hschmengler/Interplay-between-genes-cognitive-skills-and-educational-level-in-adolescent-and-young-adult-smoking

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CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest.

ETHICAL STANDARD STATEMENT

Ethical approval for TRAILS was obtained from the Dutch national ethics committee Central Committee on Research Involving Human Subjects (#NL38237.042.11). Written informed consent was obtained from both adolescents (all waves) and their parents (first three waves) prior to each assessment wave.

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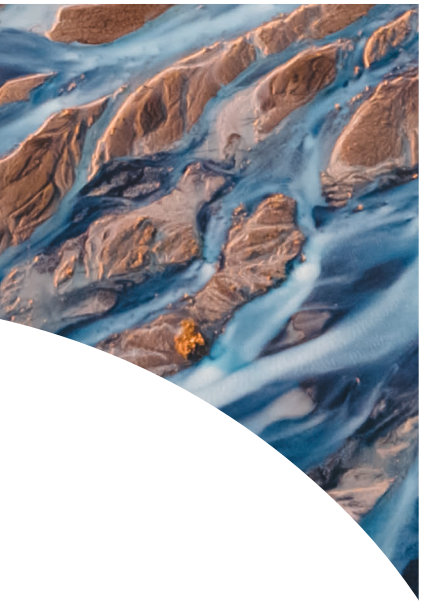
AUTHOR CONTRIBUTIONS

Heiko Schmengler: Conceptualization, Methodology, Formal analysis, Visualization, Writing - Original Draft, Writing - Review & Editing, Albertine J. Oldehinkel: Supervision, Writing - Review & Editing, Wilma A. M. Vollebergh: Conceptualization, Supervision, Project administration, Funding acquisition, Writing - Review & Editing, Joëlle A. Pasman: Writing - Review & Editing, Catharina A. Hartman: Project administration, Writing - Review & Editing, Gonneke W. J. M. Stevens: Writing - Review & Editing, Ilja M. Nolte: Formal analysis, Margot Peeters: Supervision, Project administration, Writing - Review & Editing.

CHAPTER 5

ADHD SYMPTOMS AND EDUCATIONAL LEVEL IN ADOLESCENTS: THE ROLE OF THE FAMILY, TEACHERS, AND PEERS



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ABSTRACT

Few studies have explored the contribution of family and school factors to the association between ADHD symptoms and lower education. Possibly, having more ADHD symptoms contributes to poorer family functioning and less social support, and consequently a lower educational level (i.e., mediation). Moreover, the negative effects of ADHD symptoms on education may be stronger for adolescents with poorer family functioning or less social support (i.e., interaction). Using data of the Dutch TRAILS study (N = 2,229), we evaluated associations between ADHD symptoms around age 11 and educational level around age 14, as well as between ADHD symptoms around age 14 and 16 years and subsequent changes in educational level around age 16 and 19, respectively. We assessed the potential mediating role of family functioning, and social support by teachers and classmates, all measured around ages 11, 14, and 16, while additionally evaluating interactions between ADHD symptoms and these hypothesized mediators. ADHD symptoms were associated with poorer family functioning, less social support by teachers and classmates, and lower education throughout adolescence. No conclusive evidence of mediation was found, because unique associations between family functioning and social support by teachers and classmates and education were largely absent. Furthermore, we found no interactions between ADHD symptoms and family functioning and social support by teachers and classmates. Although social support by teachers and classmates and good family functioning may benefit the wellbeing and mental health of adolescents with high levels of ADHD symptoms, they will not necessarily improve their educational attainment.

1. INTRODUCTION

Symptoms of attention-deficit/hyperactivity disorder (ADHD) are associated with significant impairments in adolescents' academic functioning, as well as a lower level of education (Schmengler et al., 2021). In selective educational systems like in the Netherlands, this association begins in childhood, when children with high levels of ADHD symptoms are more frequently assigned to lower educational tracks. Selective educational systems are defined by an early selection into different educational tracks while allowing for mobility between tracks post-selection. Subsequent to this initial selection, symptoms of ADHD have been consistently associated with moving to a lower educational track throughout all phases of adolescence and in young adulthood (Schmengler et al., 2021).

While the association between ADHD symptoms and lower education is well-established, little research has focussed on risk or protective factors in the social context, which may contribute to this association (Dvorsky & Langberg, 2016; Dvorsky et al., 2018; Zendarski et al., 2017). This is surprising, considering that it is well known that adolescents with high levels of ADHD symptoms commonly experience problems in their relationships with parents, teachers, and peers (Ewe, 2019; Glatz et al., 2011; McQuade, 2020). Meanwhile, studies have consistently highlighted the importance of these relationships for adolescents' academic development in general (Lin et al., 2019; Robertson & Reynolds, 2010; Roorda et al., 2017; Tao et al., 2022; Wentzel et al., 2021). It is therefore plausible that poorer social relationships with parents, teachers, and peers might act as mechanisms (mediators) contributing to the association between ADHD symptoms and lower education. Furthermore, adolescents with relatively poor relationships with their family, peers, or teachers and high levels of ADHD symptoms may be at a particularly high risk of poor educational outcomes, which would imply interactions between ADHD symptoms and adolescents' social context. From a clinical perspective, it is critical to explore these interactions to identify especially vulnerable subgroups, who should be prioritized for interventions.

A better understanding of social relationships in the context of ADHD and educational outcomes may also be informative for the development of new psychosocial interventions, as previous studies have highlighted the benefits of involving both the family and school to achieve positive outcomes (DuPaul et al., 2020). For example, parent-teen behaviour therapy has yielded stronger reductions in ADHD symptom severity, as well as in impairments in organization, time management, and planning in the home setting than treatment as usual (Sibley et al., 2016). A peer-delivered intervention prevented declines in class attendance, organization skills, and academic motivation throughout the school year in adolescents with symptoms of ADHD (Sibley et al., 2020). Lastly, a strong student-teacher relationship was one of the most frequently endorsed facilitators by teachers for the use of behavioural classroom interventions for ADHD (Lawson et al., 2022). In this study, we aimed to evaluate the role of three important family and school factors that could be targets

in psychosocial interventions (i.e., family functioning, and social support by classmates and teachers) as mediators in the association between ADHD symptoms and (changes in) adolescent educational track in Dutch adolescents, whilst also taking into account potential interactions between ADHD symptoms and these family and school factors.

1.1. Family and school factors as mediators in the association between ADHD symptoms and lower educational level

While the evidence for the direct and harmful effects of primary ADHD symptoms on adolescents' education is strong (Schmengler et al., 2021), ADHD symptoms might also affect educational outcomes indirectly, by causing impairments in aspects of adolescents' social context important for their academic development, such as relationships with parents, teachers, and peers. Indeed, extensive research has characterized the harmful impact of ADHD symptoms on these relationships, which may lead to poorer family functioning and receiving less social support at school.

In the parental home, adolescents with high levels of ADHD symptoms frequently have difficulties following directions and are less responsive to cues and punishment, rendering parental rule-setting less effective (Glatz et al., 2011). This can lead to perceptions of powerlessness in parents (Glatz et al., 2011), who may react with less responsiveness and emotional support (Glatz et al., 2011; Jones et al., 2015). In families with adolescents with high levels of ADHD symptoms, studies reveal more parental stress, marital conflict, higher rates of divorce, as well as poorer overall family functioning (Moen et al., 2014; Schroeder & Kelley, 2009; Theule et al., 2010; Wiener et al., 2016; Wymbs et al., 2008).

The classroom context, which requires students to sit still and pay attention, can make ADHD symptoms particularly salient. In comparison to their typically developing peers, adolescents with high levels of ADHD symptoms show significantly shorter attentive states during class, more off-task behaviours, and less overall engagement in school (Rogers et al., 2015). Often teachers lack awareness of ADHD, leading them to believe that students' inattentive and impulsive behaviour is intentional (Wiener & Daniels, 2015), and they may hence resort to frequent criticism and disciplinary penalties, leading to further negative responses from adolescents (Honkasilta et al., 2016). Indeed, studies report more conflictual and less emotionally close relationships between students with ADHD and their teachers (Ewe, 2019).

ADHD symptoms can also cause difficulties in relationships with peers. For example, adolescents with ADHD more often have problems waiting for their turn in give-and-take exchanges, often talk excessively, and more frequently interrupt others (McQuade, 2020). Restlessness and fidgeting may be misinterpreted as disinterest or impatience (McQuade, 2020). As a result of these disruptive behaviours, adolescents with ADHD symptoms tend to report fewer friends, and are more commonly rejected by their classmates (McQuade, 2020; Wiener & Daniels, 2015).

Impairments in the family life and in relationships with teachers and peers caused by ADHD symptoms may adversely affect academic performance, and in this way contribute to the association between ADHD symptoms and lower educational attainment. Past research has highlighted poor social relationships within the family and with teachers and peers as risk factors for academic problems and poor educational outcomes. These studies have mainly focussed on academic development in general, and not specifically in the context of ADHD. For example, it was found that adolescents from poorly functioning families tend to have lower math, logic, and reasoning skills (Lin et al., 2019), lower school engagement and academic self-efficiency (Stubbs & Maynard, 2017), higher risks of school disruption (Sun et al., 2021), lower academic achievement (Blackson, 1995), and ultimately lower educational attainment (Robertson & Reynolds, 2010; Roy et al., 2017). The less effective parenting styles that frequently characterize poorly functioning families may act as a mechanism connecting family dysfunction and poorer adolescent educational outcomes (Chan & Koo, 2010; Lin et al., 2019; Matejevic et al., 2014; Spera, 2005).

Accordingly, it was found that lack of social support by or poor relationships with teachers predict worse academic outcomes. Meta-analyses found associations of the quality of teacher-student relationships and social support from teachers with academic achievement (Roorda et al., 2017; Tao et al., 2022). Meta-analytic mediation analyses revealed that the association between social support from teachers and academic achievement was partially driven by behavioural (participation in academic activities, e.g., homework completion), emotional (feelings about school/academics, e.g., interest, enjoyment, school belonging and identification), and cognitive engagement (level of psychological investment in academics, e.g., intrinsic motivation, self-efficacy, self-regulation, learning strategies, goals and values) (Tao et al., 2022). Also, social support by and the quality of relationships with peers have been associated with adolescents' educational outcomes (Ahmed et al., 2010; Fang et al., 2020; Lorijn et al., 2022; Wentzel et al., 2021; Woodward & Fergusson, 2000). Adolescents who do not perceive much support from their peers tend to be less motivated to learn new skills, enjoy studying less, feel less competent and interested in subject-matter knowledge, and are less able to cope with academic difficulties, which in turn was found to predict lower academic achievement (Ahmed et al., 2010; Fang et al., 2020; Patrick et al., 2007).

Regarding children and adolescents with high levels of ADHD symptoms, one study demonstrated that parental marital problems in childhood (which could be indicative of poor family functioning) are associated with lower educational attainment in young adults with a previous ADHD diagnosis (Roy et al., 2017), and one study showed that not having a close bond with teachers is associated with lower academic motivation (Rogers et al., 2015). While these two studies demonstrated associations between family and school factors and education amongst children and adolescents with ADHD, they did not assess whether these factors act as explanatory mechanisms in the association between ADHD symptoms

and educational level. We aimed to address this omission by investigating whether having more ADHD symptoms contributes to poorer family functioning and less social support by teachers and classmates, and consequently a lower educational level (i.e., mediation).

1.2. Poor family functioning and lack of social support by teachers and classmates may amplify the association between ADHD symptoms and lower educational level (i.e., interaction)

Adverse family functioning and poor relationships with teachers and peers may also amplify the association between ADHD symptoms and lower education, yet past research on this topic is limited. We were able to identify only one study showing weaker associations between ADHD symptoms and lower Grade Point Average (GPA) in adolescents who felt socially accepted by peers (Dvorsky et al., 2018). This suggests that adolescents with low social support by peers and high levels of ADHD symptoms may be at particularly high risk of poorer educational outcomes, as they, for instance, cannot rely on academic support from classmates, for example through sharing resources, such as notes and books, which may reduce the impact of their symptoms on education (Dvorsky et al., 2018). To our knowledge, it has not yet been investigated whether the findings of Dvorsky et al. (2018) concerning fluctuations in GPA extend to long-term outcomes, such as educational track membership. Furthermore, we are not aware of any studies that assessed interactions between family functioning or social support by teachers and ADHD symptoms in affecting adolescent's educational level. It is conceivable that ADHD symptoms may affect educational level more strongly in the presence of poor family functioning, as poorly functioning families are less able to provide a supportive learning environment at home, for example by helping with homework, or vouch for their children at school. Similarly, ADHD symptoms may be particularly detrimental for the education of adolescents whose relationships with teachers are conflictual, given that adolescents with ADHD frequently have to rely on their teachers for accommodations to meet their academic needs (Harrison et al., 2020).

1.3. Aims of the study

In the present study, we aimed to contribute to the literature by investigating the role of three important family and school factors (i.e., family functioning, social support by teachers, and social support by classmates) as mediators in the association between ADHD symptoms and (changes in) educational level from early adolescence to young adulthood in the Dutch educational system. While doing so, we allowed for potential interactions between ADHD symptoms and family and school factors using interventional effects for mediation analysis (VanderWeele et al., 2014; Vansteelandt & Daniel, 2017). By using a multi-informant approach, we avoided mono-informant bias and were able to consider both parents' and adolescents' views on ADHD symptoms. By covering the entire adolescent period and the transition into young adulthood, we took into account potential changes

in the role of parents, teachers, and peers in youngsters' education over the course of development. Our study sheds new light on how several systems (family, school) around adolescents may interact or contribute to the association between ADHD symptoms and lower educational attainment.

2. MATERIALS AND METHODS

2.1. Study population

We used data from the first four waves (T1 – T4) of the TRacking Adolescents' Individual Lives Survey (TRAILS), a population-based prospective cohort study of Dutch adolescents (n = 2,229, 49.26% male, 13.50% non-Dutch ethnicity). A detailed description of the cohort can be obtained elsewhere (Oldehinkel et al., 2015). At the beginning of the study, 135 schools in the provinces of Groningen, Friesland, and Drenthe were invited, of which 122 decided to participate. Adolescents were followed between 2000 and 2010, with assessments around age 11, 14, 16, and 19.

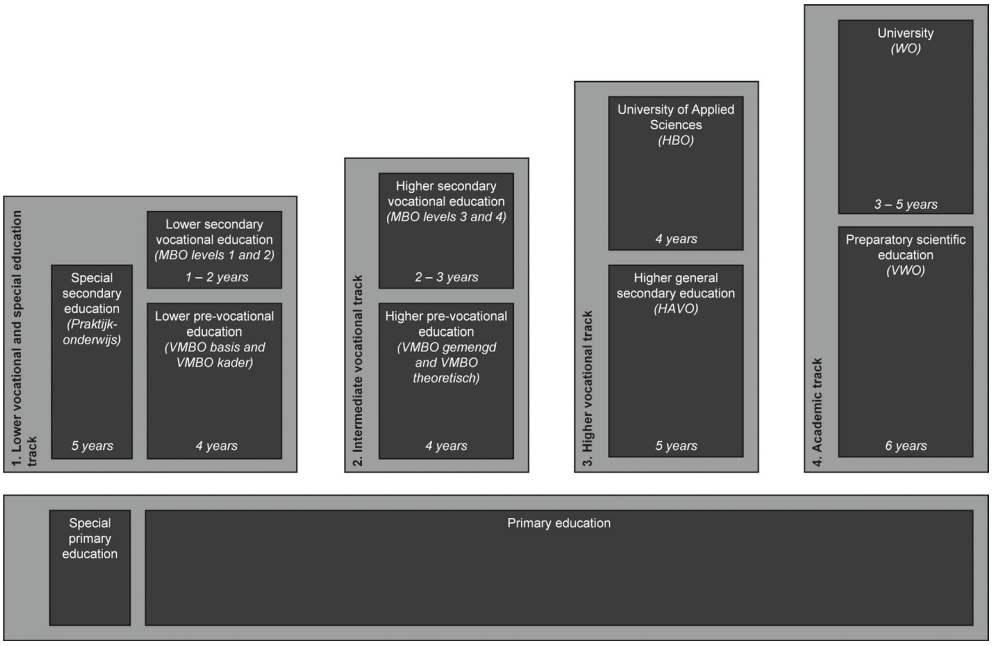
2.2. ADHD Symptoms

ADHD symptoms were assessed using a multi-informant approach, by computing the mean score of the DSM-oriented ADHD symptom scales of the parent-report Child Behavior Checklist (CBCL) and the Youth Self-report (YSR) of the Achenbach System of Empirically Based Assessment (ASEBA), completed at wave 1, 2, and 3 (i.e., around age 11, 14, and 16) (Achenbach & Rescorla, 2001). The YSR and CBCL contain lists of questions on emotional and behavioural problems in the preceding six months, with three response categories: 0 = 'not true', 1 = 'somewhat or sometimes true', 2 = 'very or often true'. Sample items from the scales include 'difficulties concentrating', 'not finishing tasks', and 'being unable to sit still'. Cronbach's alphas ranged from 0.68 to 0.74 for the YSR, and from 0.82 to 0.84 for the CBCL. Mean scores of the YSR and CBCL scales (7 items each) were computed separately, and then the mean of both scales was taken, yielding a scale ranging from 0 to 2, with higher scores indicating higher levels of ADHD symptoms. The DSM-oriented scales were constructed based on the ratings of experienced psychiatrists and psychologists in terms of the consistency of each item in the CBCL/YSR with DSM-IV ADHD diagnostic criteria. While the DSM-oriented scales do not measure all DSM symptom criteria of ADHD, and cannot take into account the age of onset, duration, and level of impairment, the scales have been able to distinguish between diagnosed and non-diagnosed children, and are strongly associated with other standardized rating scales, such as the Conners Scales (Achenbach et al., 2003).

2.3. Adolescents' educational level

The Dutch educational system is characterized by an early (age 11–12) selection into a particular educational track, based on cognitive tests and the advice of the primary school. There are four tracks in the Dutch educational system, each consisting of a specific type of secondary school followed by tertiary education at the corresponding level (**Figure 5.1**): (1) lower vocational track, (2) intermediate vocational track, (3) higher vocational track, (4) academic track. In addition, there is a special education track, attended by students who are unable to attend regular education. This track was collapsed with the lower vocational track in our analyses. While in secondary education, adolescents can be recommended by their school to move between educational tracks, depending on their academic performance. Furthermore, after attaining specific milestones of their track, students can become eligible to continue their education at a higher track. Overall, a substantial proportion of students is mobile between educational tracks: 24.66% of adolescents moved to a different track between wave 2 and 3 (i.e., between around age 14 and 16), and 25.41% between wave 3 and 4 (i.e., between around age 16 and 19). Educational track membership was assessed at each wave by asking for participants' current enrolment, as well as their highest completed diploma. Participants who finished the final diploma of a given track received the value corresponding to that level for all subsequent waves, unless they continued education at a higher level. Our measure of educational level allows us to assign a score that represents an age-appropriate measure of educational level as proxy of developing socioeconomic status (SES).

FIGURE 5.1 The Dutch educational system



2.4. Family functioning

Family functioning was assessed at wave 1, 2, and 3 (i.e., around ages 11, 14, and 16) by parent-report using a modified version of the Dutch version of the General Functioning Scale of the McMaster Family Assessment Device (FAD), which is a mean score of 12 items with each four response categories: 1 = 'totally disagree', 2 = 'disagree', 3 = 'agree', 4 = 'totally agree' (Bouma et al., 2008; Epstein et al., 1983). We recoded the items of the FAD such that higher scores indicate better family functioning. Six dimensions of family functioning were assessed: communications, problem solving, affective responsiveness, affective involvement, roles, and behaviour control. Example items include 'being able to count on each other's support', 'trusting each other', and 'avoiding talking about one's fears and worries' within the family. The scale has demonstrated adequate test-retest reliability, is moderately correlated with other self-report family functioning measures, and has shown utility in differentiating between clinician-rated healthy and unhealthy families (Hamilton & Carr, 2016; Wenniger et al., 1993). The Cronbach's alpha of the FAD in TRAILS ranged from 0.85 to 0.87.

2.5. Social support by teachers

Social support by teachers was assessed at wave 1, 2, and 3 (i.e., around age 11, 14, and 16) by adolescent-report using the mean score of the corresponding affection (4 items) and behavioural confirmation (4 items) subscales adapted from the Social Production Functions (SPF) Questionnaire (Ormel et al., 1997). The response options were 1 = 'never', 2 = 'almost never', 3 = 'sometimes', 4 = 'almost always', 5 = 'always'. Higher scores on the scale indicate higher levels of social support by teachers. Example items include 'most teachers are satisfied with the way I am' and 'I can trust most teachers'. The Cronbach's alpha ranged from 0.75 to 0.78 for the affection subscale, and from 0.72 to 0.74 for the behavioural confirmation subscale.

2.6. Social support by classmates

Social support by classmates was assessed at waves 1, 2, and 3 (i.e., around age 11, 14, and 16) by adolescent-report using the mean score of the corresponding affection (4 items) and behavioural confirmation (4 items) subscales adapted from the Social Production Functions (SPF) Questionnaire (Ormel et al., 1997). The response options were 1 = 'never', 2 = 'almost never', 3 = 'sometimes', 4 = 'almost always', 5 = 'always'). Higher scores on the scale indicate higher levels of social support by classmates. Example items include 'most classmates help me in case of a problem' and 'most classmates like to do things with me'. The Cronbach's alpha ranged from 0.80 to 0.84 for the affection subscale, and from 0.76 to 0.82 for the behavioural confirmation subscale.

2.7. Covariates

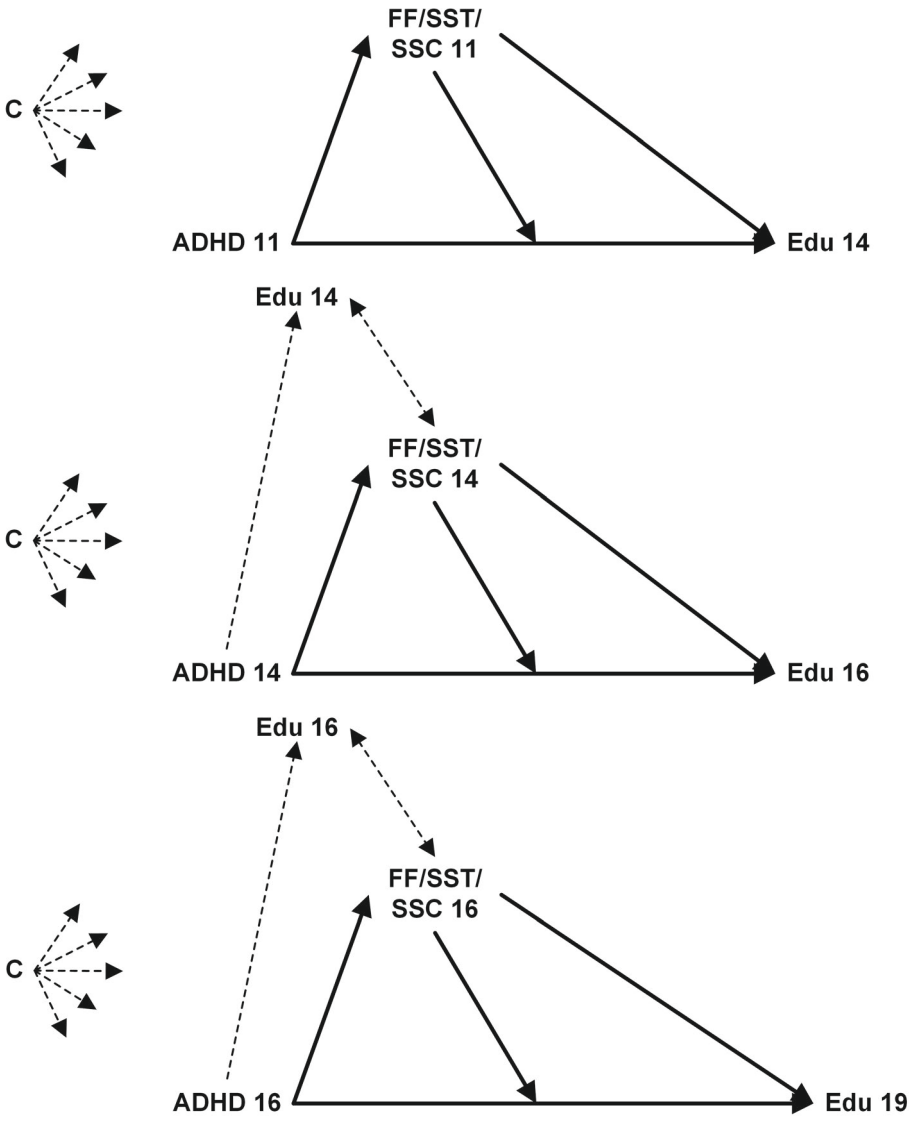
Covariates assessed at baseline around age 11 (wave 1) include children's IQ, which was estimated using the Block Design and Vocabulary subtests of the Revised Wechsler Intelligence Scale for Children (WISC-R), as well as parents' socioeconomic status (SES), constructed as the mean score of five indicators (standardized): maternal and paternal educational attainment, maternal and paternal occupational position (according to the International Standard Classification of Occupations), and family income. Furthermore, we included gender and ethnicity as demographic covariates. Children were classified as having non-Dutch ethnicity if at least one of their parents was born outside the Netherlands. Finally, we adjusted for adolescent age, which was measured contemporaneously with each assessment of ADHD symptoms.

2.8. Analytic approach

First, we computed descriptive statistics of the study population by cross-tabulating baseline characteristics (mean age 11) with early adolescent educational track membership at wave 2 (mean age 14), as well as ADHD symptoms and family and school factors with concurrent educational level from wave 2 to wave 3 (mean age 19). In mediation analyses, we modelled the association between ADHD symptoms and initial educational level in early adolescence (i.e., around age 14). Subsequent changes in educational level were estimated by regressing educational level around 16 and 19 on previous measurements of educational level (i.e., around 14 and 16, respectively). We assessed the potential mediating role of family functioning, and social support by teachers and classmates, all measured concurrently with ADHD symptoms (around ages 11, 14, and 16), in the association between ADHD symptoms and (changes in) education, whilst additionally evaluating interactions between ADHD symptoms and these hypothesized mediators (**Figure 5.2**). Separate models were run for each age category (i.e., between around age 11 and 14, 14 and 16, and 16 and 19) and for each potential mediator (i.e., family functioning, social support by classmates, social support by teachers). It is possible that ADHD symptoms also affect educational level after relatively short amounts of time. In this case, measures of educational level assessed concurrently with ADHD symptoms could function as exposure-induced mediator-outcome confounders, which can cause bias in many types of mediation models. We therefore used interventional effects mediation models, which can still yield valid results by treating these confounders as additional mediators (Chan & Leung, 2022; VanderWeele et al., 2014; Vansteelandt & Daniel, 2017). All continuous variables were z-score transformed to facilitate interpretability of coefficients. If change in education was estimated by regressing educational level on its past value, standardized beta-coefficients of 0.03 were judged as small, 0.07 as medium, and 0.12 as large effects, as recommended by Orth et al. (2022) for longitudinal autoregressive models. For all other estimates from mediator and outcome models, we followed recommendations by Funder and Ozer (2019),

who suggested classifying effects of 0.05 as very small, 0.10 as small, 0.20 as medium, 0.30 as large, and 0.40 as very large in psychological research. The effect sizes of indirect effects were given by the proportion mediated (Alwin & Hauser, 1975; Goldstein, 2016).

FIGURE 5.2 Illustrations of the hypothesized relationships between ADHD symptoms, family and school factors, and educational level across adolescence, as assessed with mediation analysis using interventional effects



Edu = educational level; FF = family functioning; SST = social support by teachers; SSC = social support by classmates; C = covariates, which were included in all regression equations (i.e., gender, ethnicity, IQ, and parental SES measured at baseline, and age assessed in the same wave ADHD symptoms and potential mediators were measured).

Attrition analyses showed that at wave 2 3.63% (N = 81) of the original participants no longer participated in the study. At wave 3 this was the case for 18.44% (N = 411), and at wave 4 for 15.66% (N = 349) of the original participants. Adolescents with greater age, male gender, non-Dutch ethnicity, lower educational level and IQ, as well as those from lower SES households were more likely to drop out of the study (**Table 5.S1**). ADHD symptoms were also related to dropout, but only significantly so at wave 1 (i.e., around age 11). Considering family and school factors, lower family functioning around age 14 was related to increased risks of having left the study by wave 4 (i.e., around age 19). Comparable differences were found between participants with complete information on educational level and those whose educational level was missing or could not be classified (**Table 5.S2**).

Missing information on educational track membership from wave 2 to 4 was imputed using retrospective event history calendar data collected at wave 3 and wave 5. Participants who were still in elementary education or in a combined class at wave 2 were assigned according to their elementary school teacher's recommended level. If this information was not available, pupils were classified according to the first track they attended after leaving elementary education or the combined class. It was not possible to classify participants who had not been in education for a longer period, were not classifiable into an educational track (e.g., because of education abroad), whose educational level was assessed incompletely, who did not respond to questions on education, or who had left the educational system permanently (wave 2: N = 221, 10.29%; wave 3: N = 289, 15.90%; wave 4: N = 373, 19.84%). Education was considered as missing for these participants. This missing information together with missing values on all other variables was addressed using multiple imputations by chained equations under fully conditional specification (van Buuren, 2007) and under the assumption of missingness at random. 90 imputed datasets were created with 50 iterations between datasets. Analyses were conducted in Stata 16.1 and in R 4.2.2, making use of the 'intmed' (version 0.1.2) package (Chan, 2022) for mediation analyses.

2.9. Sensitivity analyses

The 'intmed' package currently does not support ordinal outcomes. We therefore assessed whether the ordinal nature of our educational variable affected the linear regression results of the mediation models by repeating our analyses using structural equation modelling and the weighted least square means and variance adjusted (WLSMV) estimator in Mplus 8.8. These models allow for ordinal outcomes, while assuming the absence of exposure-mediator interactions (**Figures 5.S1-S3**). In mediation models it is usually preferable to measure exposures, mediators, and outcomes in consecutive waves. However, the time lags between measurements in TRAILS are rather long (about three years), which means that adolescents are frequently in different social contexts (e.g., in different classrooms with different teachers and peers) in one wave compared to the next. To adequately assess the

consequences of ADHD symptoms, in terms of social support in the classroom and family functioning, it is important to measure these variables within the same social context as ADHD symptoms, necessitating a fairly short time interval between measurements. This is why we modelled exposures and mediators contemporaneously in our main analyses. Nevertheless, we conducted sensitivity analyses in which we allowed for each one wave time lag between measurements of exposures, mediators, and outcomes, to assess whether the choice of time lags affects our results (**Figure 5.S4, Table 5.S3**).

3. RESULTS

3.1. Descriptive statistics

Table 5.1 shows the characteristics of TRAILS participants around age 11 according to educational level around age 14. More ADHD symptoms around age 11 were strongly associated with lower education around age 14. Children about to attend the lower educational tracks in early adolescence tended to experience poorer family functioning. No significant differences according to educational level around age 14 were found in terms of social support by teachers and classmates around age 11. Children with less affluent or non-Dutch parents were more commonly selected into the lower educational tracks. Girls more frequently went on to attend the academic and intermediate vocational tracks than boys. Further, higher IQ around age 11 was strongly associated with higher education around age 14. Being in a higher educational track was strongly and inversely associated with ADHD symptoms around age 14 and 16 (**Table 5.2**). Overall, with few exceptions, adolescents in the higher educational tracks tended to experience somewhat better family functioning, and more social support by teachers and classmates around 14 and 16.

TABLE 5.1: Characteristics of adolescents participating in the TRAILS study (wave 1 – 4, the Netherlands, 2000–2010, N = 2,229) at wave 1 (2000-2002) according to educational level at wave 2 (2003-2005)

	All levels		**Lower vocational & special education**		**Intermediate vocational**		**Higher vocational**		**Academic**	
	N = 2,229		**N = 635**		**N = 497**		**N = 383**		**N = 457**	
ADHD symptoms†, mean (SD)	0.58	(0.33)	0.69[a]	(0.35)	0.62[b]	(0.32)	0.55[c]	(0.31)	0.43[d]	(0.27)
Family functioning, mean (SD)	3.23	(0.36)	3.16[a]	(0.34)	3.23[b]	(0.38)	3.24[b]	(0.35)	3.32[c]	(0.36)
Social support by teachers, mean (SD)	3.81	(0.70)	3.79[a]	(0.79)	3.82[a]	(0.67)	3.78[a]	(0.66)	3.87[a]	(0.57)
Social support by classmates, mean (SD)	3.58	(0.73)	3.60[a]	(0.82)	3.62[a]	(0.71)	3.53[a]	(0.68)	3.53[a]	(0.64)
Male gender, N (%)	1,098	(49.26)	341[a]	(53.70)	217[b]	(43.66)	196[a]	(51.17)	195[b]	(42.67)
Non-Dutch ethnicity, N (%)	301	(13.50)	108[a]	(17.01)	61[b]	(12.27)	39[b]	(10.18)	45[b]	(9.85)
Age, mean (SD)	11.11	(0.56)	11.16[a]	(0.56)	11.07[b]	(0.54)	11.05[b]	(0.56)	11.14[a]	(0.56)
Parental socioeconomic status (SES), mean (SD)	-0.05	(0.80)	-0.53[a]	(0.70)	-0.16[b]	(0.67)	0.21[c]	(0.68)	0.55[d]	(0.70)
Wechsler Intelligence Deviation Quotient, mean (SD)	97.19	(15.00)	86.05[a]	(12.49)	95.20[b]	(10.98)	102.68[c]	(11.20)	111.14[d]	(11.91)

SD = standard deviation. Parameters with different superscripts differ significantly from each other at p<0.05, as determined by chi-squared tests (categorical variables) and one-way ANOVAs with pairwise comparisons (continuous variables). Higher scores indicate higher SES, higher intelligence, more ADHD symptoms, higher levels of family functioning, and more social support by teachers and classmates. †ADHD symptoms were assessed using mean scores of the YSR and CBCL DSM-oriented ADHD symptom scales.

TABLE 5.2 Characteristics of adolescents and young adults in the TRAILS study (wave 1 – 4, the Netherlands, 2000–2010, N = 2,229) according to concurrent educational level

	Wave 2		**Wave 3**		**Wave 4**	
N participants	2,148		1,818		1,880	
Date range	2003-2005		2005–2008		2008–2010	
Male gender, N (%)	1,054	(49.07)	867	(47.69)	898	(47.77)
Educational level, N (%)						
Lower vocational & special education	635	(32.20)	349	(22.83)	161	(10.68)
Intermediate vocational	497	(25.20)	405	(26.49)	498	(33.02)
Higher vocational	383	(19.42)	362	(23.68)	475	(31.50)
Academic	457	(23.17)	413	(27.01)	374	(24.80)
ADHD symptoms†, mean (SD)						
All levels	0.54	(0.32)	0.52	(0.32)	-	-
Lower vocational & special education	0.63[a]	(0.33)	0.64[a]	(0.34)	-	-
Intermediate vocational	0.58[b]	(0.31)	0.57[b]	(0.32)	-	-
Higher vocational	0.53[c]	(0.32)	0.50[c]	(0.28)	-	-
Academic	0.43[d]	(0.28)	0.40[d]	(0.27)	-	-
Family functioning, mean (SD)					-	-
All levels	3.36	(0.40)	3.35	(0.40)	-	-
Lower vocational & special education	3.26[a]	(0.41)	3.32[a]	(0.44)	-	-
Intermediate vocational	3.36[b]	(0.40)	3.31[a]	(0.41)	-	-
Higher vocational	3.41[b/c]	(0.38)	3.35[a/b]	(0.38)	-	-
Academic	3.44[c]	(0.38)	3.40[b]	(0.38)	-	-
Social support by teachers, mean (SD)						
All levels	3.48	(0.65)	3.43	(0.61)	-	-
Lower vocational & special education	3.47[a]	(0.74)	3.41[a/b]	(0.69)	-	-
Intermediate vocational	3.47[a]	(0.60)	3.44[a/b]	(0.63)	-	-
Higher vocational	3.46[a]	(0.62)	3.37[a]	(0.61)	-	-
Academic	3.56[b]	(0.56)	3.48[b]	(0.48)	-	-
Social support by classmates, mean (SD)						
All levels	3.59	(0.65)	3.57	(0.55)	-	-
Lower vocational & special education	3.50[a]	(0.74)	3.53[a]	(0.65)	-	-
Intermediate vocational	3.61[b]	(0.66)	3.55[a/b]	(0.54)	-	-
Higher vocational	3.63[b]	(0.60)	3.58[a/b]	(0.51)	-	-
Academic	3.64[b]	(0.57)	3.61[b]	(0.49)	-	-
Age, mean (SD)						
All levels	13.57	(0.53)	16.28	(0.71)	-	-
Lower vocational & special education	13.65[a]	(0.52)	16.15[a]	(0.66)	-	-
Intermediate vocational	13.53[b]	(0.56)	16.14[a]	(0.72)	-	-

TABLE 5.2 Continued

	Wave 2		Wave 3		Wave 4	
Higher vocational	13.49[b]	(0.54)	16.25[b]	(0.62)	-	-
Academic	13.55[b]	(0.49)	16.25[b]	(0.54)	-	-

SD = standard deviation. Parameters with different superscripts differ significantly from each other at p<0.05, as determined by one-way ANOVAs with pairwise comparisons. Higher scores indicate more ADHD symptoms, higher levels of family functioning, and more social support by teachers and classmates. [†]ADHD symptoms were assessed using mean scores of the YSR and CBCL DSM-oriented ADHD symptom scales.

3.2. Mediation and interaction analyses

Mediation analyses (**Table 5.3**) revealed small direct effects of ADHD symptoms in childhood (around age 11) on lower educational level around age 14. Similarly, we found medium-sized direct effects of ADHD symptoms around age 14 and 16 on decreases in educational level by around age 16 and 19, respectively. Our models also revealed small associations between ADHD symptoms and lower family functioning, as well as less social support by classmates, and medium-sized to large associations between ADHD symptoms and less social support by teachers, throughout the whole study period. Unexpectedly, in none of our mediation models were family functioning and social support by teachers and classmates associated with (changes in) educational level. Accordingly, the interventional effects mediation models detected no significant indirect effects. Furthermore, we did not find any significant interactions between family functioning, and social support by classmates and teachers and ADHD symptoms.

Results regarding mediation in our sensitivity analysis allowing for ordinal outcomes were consistent with our main analysis (**Figures 5.S1-S3**). However, when one wave time lag (approximately 3 years) was allowed between the measurements of ADHD symptoms and family and school factors, we detected negligible to very small but significant associations between more social support by teachers and classmates around age 14 and a higher educational level around age 16, as well as between more social support by teachers around age 16 and increases in educational level by around age 19. Nevertheless, we found only a very small (proportion mediated = 0.05) indirect effect of ADHD symptoms around age 11 on lower education around age 16 via lower levels of social support by classmates around age 14 (**Figure 5.S4, Table 5.S3**).

In order to gain further insight into why we found no mediation when modelling ADHD symptoms and family and school factors contemporaneously, we conducted sequentially adjusted regression analyses of associations between family functioning and social support by teachers and classmates and subsequent (changes in) education (**Table 5.S4**). These analyses revealed some negligible to small associations between family and school factors and (changes in) education, all of which remained or became significant after adjustment for covariates, but none of which survived further adjustment for ADHD symptoms.

4. DISCUSSION

In this study, we investigated the role of family functioning and social support by classmates and teachers as mediators within associations between symptoms of ADHD and educational level, while also evaluating potential interactions between ADHD symptoms and these family and school factors. Significant direct effects revealed that ADHD symptoms were associated with being in a lower educational track in early adolescence, and that adolescents with high levels of ADHD symptoms more frequently decreased in their educational level over the course of adolescence, relative to their peers with low levels of ADHD symptoms, irrespective of family functioning and social support by teachers and peers. In addition, ADHD symptoms were associated with worse family functioning, as well as lower levels of social support by teachers and classmates throughout the whole study period. Yet, significant indirect effects were absent in all but one model in our sensitivity analyses, in which ADHD symptoms around age 11 were allowed to influence social support by classmates approximately three years later. Whenever family and school factors were modelled contemporaneously with ADHD symptoms (i.e., within the same social contexts), no mediation was found. This is likely due to the small magnitude of associations between family and school factors and subsequent (changes in) education after adjustment for covariates, which were even completely absent after also adjusting for concurrent measures of ADHD symptoms. Lastly, we observed no interactions between ADHD symptoms and family and school factors, suggesting that associations between ADHD symptoms and education do not differ in the presence of varying levels of family functioning and social support by teachers and peers. Overall, our results highlight the robustness of the detrimental associations of ADHD symptoms with educational level throughout adolescence, which persist even in the presence of positive social relationships within the family and with teachers and peers.

4.1. Interpretation of findings

4.1.1. Family and school factors largely do not mediate the association between ADHD symptoms and educational level

Despite robust and consistent relations of ADHD symptoms with both educational level and family and school factors, contrary to our hypotheses, we found little empirical support that these factors contribute much to the association between ADHD symptoms and lower education as mediators. Mediation was largely absent given that we only observed negligible to small associations between family functioning and social support by teachers and classmates and (changes in) education, which were no longer significant following adjustment for covariates and ADHD symptoms, if those symptoms were assessed within the same social contexts as family and school factors. One exception was an extremely

TABLE 5.3 Direct and indirect effects of ADHD symptoms on (changes in) educational level in each subsequent wave, as well as selected estimates from mediator and outcome models in the TRAILS study (wave 1 – 4, the Netherlands, 2000–2010, N = 2,229); potential mediators were measured concurrently with ADHD symptoms and evaluated in separate models; linear regression (standardized beta-coefficient, 95% Confidence Interval, p-value)

	Family functioning		
	IE	DE	PM
Direct and indirect effects			
Educational level age 14	0.00 (-0.01 – 0.01), 0.818	**-0.14 (-0.17 – -0.11), <0.001**	0.01
Changes in educational level between around age 14 and 16	0.00 (0.00 – 0.01), 0.652	**-0.09 (-0.12 – -0.07), <0.001**	0.01
Changes in educational level between around age 16 and 19	0.00 (-0.01 – 0.00), 0.491	**-0.08 (-0.11 – -0.05), <0.001**	0.01
	Family functioning		
Mediator model estimates *(hypothesized mediators were measured concurrently with ADHD symptoms)*			
ADHD symptoms around age 11	**-0.17 (-0.21 – -0.12), <0.001**		
ADHD symptoms around age 14	**-0.18 (-0.23 – -0.14), <0.001**		
ADHD symptoms around age 16	**-0.18 (-0.23 – -0.13), <0.001**		
	Educational level around age 14		
Outcome model estimates *(outcomes were measured one wave after ADHD symptoms and the hypothesized mediators)*			
Family functioning	0.02 (-0.01 – 0.05), 0.248		
Social support by teachers	0.02 (-0.01 – 0.06), 0.186		
Social support by classmates	0.01 (-0.02 – 0.05), 0.421		
ADHD × family functioning	-0.02 (-0.06 – 0.01), 0.118		
ADHD × social support by teachers	0.01 (-0.02 – 0.04), 0.366		
ADHD × social support by classmates	0.02 (-0.01 – 0.05), 0.110		

IE = indirect effect; DE = direct effect; PM = proportion mediated. ***Boldface*** *denotes statistical significance at p<0.05. All models are adjusted for time-stable covariates measured at baseline (i.e., gender, ethnicity, IQ, parental SES) and age assessed in the same wave ADHD symptoms and potential mediators were measured. Past education, which is a potential exposure-induced mediator-outcome confounder (Chan & Leung, 2022; VanderWeele et al., 2014; Vansteelandt & Daniel, 2017), was treated as additional mediator.*

Social support by teachers			**Social support by classmates**		
IE	DE	PM	IE	DE	PM
-0.01 (-0.02 – 0.00), 0.088	**-0.14 (-0.17 – -0.10), <0.001**	0.05	-0.01 (-0.01 – 0.00), 0.079	**-0.14 (-0.17 – -0.11), <0.001**	0.04
-0.01 (-0.01 – 0.00), 0.232	**-0.09 (-0.11 – -0.06), <0.001**	0.03	0.00 (-0.01 – 0.00), 0.514	**-0.09 (-0.11 – -0.06), <0.001**	0.01
-0.01 (-0.02 – 0.00), 0.179	**-0.07 (-0.10 – -0.04), <0.001**	0.04	0.00 (0.00 – 0.01), 0.696	**-0.08 (-0.11 – -0.05), <0.001**	0.01

Social support by teachers	Social support by classmates
-0.21 (-0.25 – -0.16), <0.001	**-0.16 (-0.20 – -0.11), <0.001**
-0.25 (-0.29 – -0.21), <0.001	**-0.18 (-0.23 – -0.14), <0.001**
-0.30 (-0.35 – -0.25), <0.001	**-0.14 (-0.19 – -0.09), <0.001**

Changes in educational level between around age 14 and 16	Changes in educational level between around age 16 and 19
-0.01 (-0.04 – 0.01), 0.314	0.01 (-0.02 – 0.04), 0.574
0.02 (-0.01 – 0.04), 0.232	0.02 (-0.01 – 0.05), 0.150
0.01 (-0.01 – 0.03), 0.446	-0.01 (-0.04 – 0.02), 0.722
0.01 (-0.02 – 0.03), 0.653	0.01 (-0.02 – 0.03), 0.689
0.00 (-0.02 – 0.03), 0.746	0.00 (-0.02 – 0.03), 0.810
0.00 (-0.02 – 0.02), 0.911	0.00 (-0.03 – 0.02), 0.846

small indirect effect via social support by classmates in the sensitivity analysis in which family and school factors were measured approximately three years after ADHD symptoms. This was unexpected, given that we deemed mediation more likely with ADHD symptoms and family and school factors measured at the same time. Our findings contrast past research highlighting the importance of the social environment at home and at school for adolescents' educational attainment (Blackson, 1995; Lin et al., 2019; Robertson & Reynolds, 2010; Roorda et al., 2017; Roy et al., 2017; Tao et al., 2022; Wentzel et al., 2021).

There are several potential explanations for the discrepancy between our results and past research. First, our measures of family functioning and peer/teacher support may not have sufficiently tapped into the aspects of social relationships contributing most strongly to the association between ADHD symptoms and educational level. Family functioning, for instance, is a rather general measure assessing family climate but may not exactly capture the specific aspects of parenting (e.g., parental involvement in school) important for the education of adolescents (Castro et al., 2015; Masud et al., 2015; Song et al., 2015). Similarly, adolescents may perceive much affection and behavioural confirmation from peers and teachers, which would be reflected in high scores on the SPF scales used here, but may lack more academic support from their classmates (e.g., through note sharing) (Dvorsky et al., 2018) or teachers (e.g., accommodations) (Harrison et al., 2020). Future studies may benefit from including measures of social support that more specifically relate to adolescents' academic development.

Second, most previous studies have used GPA-based measures (Gallardo et al., 2016; Goguen et al., 2010; Scales et al., 2020; Sebanc et al., 2014; Wentzel & Caldwell, 1997) or standardized testing (Blackson, 1995; Lee, 2012; Li et al., 2020; Liem & Martin, 2011; Lin et al., 2019; Phan & Ngu, 2018; Song et al., 2015) as measures of education. Family and school factors may strongly affect day-to-day fluctuations in GPA, yet these fluctuations might not be substantial enough to cause moving to a lower school type for most adolescents. Still, two studies which, similar to ours, used measures of long-term educational outcomes (mean number of school certificate passes (Woodward and Fergusson, 2000), and degree attainment in young adulthood (Roy et al., 2017)) did find associations. Nevertheless, family and school factors might be more strongly and consistently associated with GPA than long-term educational outcomes.

Lastly, differences between our results and past findings could be explained by divergent approaches to covariate adjustment. For example, most previous studies on social factors and education had a non-clinical focus and therefore did not adjust for, for instance, adolescents' psychological problems, including ADHD symptoms. We were able to find only two studies controlling for similar covariates to our study, which, in line with our results, both found substantial reductions in associations between family and school factors and educational outcomes after statistical adjustment. Yet, unlike most associations in our study, associations in these studies remained significant. In Sun et al. (2021), the

association between family functioning and school disruption was strongly attenuated after adjusting for adolescents' psychological problems, including ADHD symptoms. Similar results were found by Woodward and Fergusson (2000), who besides ADHD symptoms, like in our study also adjusted for parental SES and IQ, concerning the association between peer relationship problems and later educational attainment (Woodward & Fergusson, 2000). Overall, these results highlight the importance of careful covariate selection when evaluating associations between social factors and educational outcomes.

4.1.2. Family and school factors and ADHD symptoms do not interact

We found no interactions of family functioning and social support by teachers and classmates with ADHD symptoms, suggesting that ADHD symptoms are similarly related to education across varying levels of these family and school factors. A potential explanation for this finding is that the current structure of the Dutch educational system might not be able to provide enough support to adolescents with high levels of ADHD symptoms, even if teachers and parents are willing to provide such assistance. For example, classes in most schools might be too large to optimally support students with special needs, including ADHD, for whom small classes of maximally 8 to 15 students may be optimal (Loe & Feldman, 2007). In 2016, in secondary schools in the Netherlands, the academic and higher vocational tracks had an average class size of 27 pupils in the first year, and the intermediate and lower vocational tracks 19 and 12 pupils, respectively (Dutch House of Representatives, 2017). In primary school, the average class size was at 23 pupils (Dutch Ministry of Education, Culture and Science, 2018). It is thus possible that, particularly in primary school and the higher educational tracks, the Dutch system currently cannot sufficiently accommodate the academic needs of adolescents grappling with ADHD symptoms. The effect of insufficient facilitation may be strong, such that educational outcomes do not change for the better even if these adolescents perceive positive relationships with teachers and peers and grow in up in households with a positive family climate. Furthermore, the smaller number of pupils in intermediate vocational and lower vocational tracks may be late in view of reaching higher educational outcomes, and insufficient facilitation in primary school may have already set lower educational outcomes in motion.

4.2. Strengths and limitations

Key strengths of our study are a high response rate, its long follow-up, and the consistency of measures over time, allowing to capture multiple developmental periods simultaneously (Oldehinkel et al., 2015). By incorporating both parent- and self-reported ADHD symptoms, we were able to avoid mono-informant bias (Martel et al., 2021). Furthermore, we incorporated factors both in the school and home context, which has rarely been done in previous studies. Another strength of our study is our measure of educational level, which is consistent throughout adolescence and young adulthood. The selective educational

system of the Netherlands provides an age-appropriate measure of educational attainment, as proxy for developing SES over the course of adolescence. That is, the selection into educational tracks as early as at age 11-12 years means that Dutch adolescents grow up in distinct educational environments that are characterized by different social norms, future expectations, cognitive resources, and occupational prospects — characteristics that are closely related to conceptualizations of SES in adulthood (Schmengler et al., 2021). One could therefore argue that in selective educational systems, such as in the Netherlands, youngsters move into 'their own' SES at a much earlier age than in comprehensive systems, such as in Finland or the USA. TRAILS provides a unique opportunity to investigate both the antecedents and consequences, in terms of health-related characteristics, of this differentiation and subsequent intragenerational social mobility in adolescents and young adults (Schmengler et al., 2021).

Some limitations of this study may have affected our results and conclusions. First, we used data from a relatively low-risk population-based sample of adolescents, which means that findings may not extend to high-risk or clinical samples of adolescents diagnosed with ADHD. As we did not use clinical diagnoses, we could not distinguish between the impact of symptoms above and below clinical thresholds. Second, informants might differ in how they judge social relationships. Future studies may take a multi-informant approach when assessing family functioning and social relationships of adolescents with teachers and peers, to take into account differing perspectives on these relationships. Third, attrition might have influenced the results of our study. Although we implemented multiple imputations to manage missing data, higher dropout of adolescents with less favourable conditions (e.g., lower education, parental SES, IQ) may still have affected our results. As these characteristics are also important determinants of adverse outcomes in young adulthood, further research on at-risk groups is necessary (Caspi et al., 2016). Lastly, future research may also consider the reverse-causal path in the association between ADHD and family functioning. For example, past studies have found associations between early-life adversity, including indicators of family dysfunction, and later neurodevelopmental outcomes, including symptoms of ADHD (Xu et al., 2022). Particularly early and severe emotional deprivation may adversely affect children's neurodevelopment, potentially through epigenetic mechanisms (Sonuga-Barke et al., 2017), yet familial confounding may also contribute to associations between childhood adversity and ADHD symptoms (Carlsson et al., 2021). Future research may employ genetically informed designs to investigate to what extent ADHD symptoms contribute to the association between childhood adversity, including severe family dysfunction, and later educational attainment.

5. CONCLUSION AND IMPLICATIONS

Our findings suggest that ADHD symptoms are robustly associated with lower educational attainment over the course of adolescence. Yet, this association was not mediated by general measures of family functioning and social support by teachers and classmates. Furthermore, we found no evidence that these measures amplify the association between ADHD symptoms and lower educational level. General social support and family functioning may still substantially contribute to associations between ADHD symptoms and other functional outcomes, such as mental health (Dvorsky & Langberg, 2016; Karawekpanyawong et al., 2021; Meinzer et al., 2021). For educational attainment, specific aspects of social support proximally related to academic functioning may be more important (e.g., parental involvement in school, note sharing with classmates, accommodations at school). Crucially, research on the role of social factors in associations between ADHD symptoms and functional outcomes is still in its infancy (Dvorsky & Langberg, 2016). Therefore, replication studies are necessary to explore the extent to which our results extend across dimensions of social support and to other, including high-risk, populations. Lastly, our findings may differ from studies on interventions targeting adolescents' social context, as interventions addressing multiple systems (family, teachers, peers) have shown promise and should not be ignored based on our results (DuPaul et al., 2020; Sibley et al., 2016; Sibley et al., 2020). These studies typically have a relatively short follow-up (DuPaul et al., 2020) and may therefore also capture more subtle effects of the social context on academic functioning, which may be missed in studies with long follow-up and focus on long-term educational attainment, like TRAILS.

DATA AVAILABILITY

Under the General Data Protection Regulation (GDPR), our dataset is considered pseudonymized rather than anonymized, and is still regarded as personal data. When participants were invited to the cohort more than 20 years ago, they were not asked to give informed consent to make their personal data publicly available in pseudonymized form. As a result of this, legal and ethical restrictions prevent the authors from making data from the TRAILS study publicly available. For more information about accessing data from the TRAILS study, please see https://www.trails.nl/en/hoofdmenu/data/data-use. The syntax for our analyses can be obtained from: https://github.com/hschmengler/ADHD-symptoms-and-educational-level-in-adolescents-the-role-of-the-family-teachers-and-peers

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CONFLICT OF INTEREST

The authors declare that they have no conflicts of interest**.**

ETHICAL APPROVAL

Ethical approval for TRAILS was obtained from the Dutch national ethics committee Central Committee on Research Involving Human Subjects (#NL38237.042.11).

INFORMED CONSENT

Written informed consent was obtained from both adolescents (all waves) and their parents (first three waves) prior to each assessment wave.

OPEN ACCESS

AUTHOR CONTRIBUTIONS

Heiko Schmengler: Conceptualization, Methodology, Formal analysis, Visualization, Writing - Original Draft, Writing - Review & Editing, Margot Peeters: Conceptualization, Supervision, Project administration, Writing - Review & Editing, Gonneke W. J. M. Stevens: Writing - Review & Editing, Catharina A. Hartman: Writing - Review & Editing, Albertine J. Oldehinkel: Supervision, Writing - Review & Editing, Wilma A. M. Vollebergh: Conceptualization, Supervision, Project administration, Funding acquisition, Writing - Review & Editing.

CHAPTER 6

SOCIOECONOMIC INEQUALITIES IN ADOLESCENT HEALTH BEHAVIOURS ACROSS 32 DIFFERENT COUNTRIES – THE ROLE OF COUNTRY-LEVEL SOCIAL MOBILITY

Based on: **Schmengler, H.**, Peeters, M., Stevens, G. W. J. M., Kunst, A. E., Delaruelle, K., Dierckens, M., Charrier, L., Weinberg, D., Oldehinkel, A. J., & Vollebergh, W. A. M. (2022). Socioeconomic inequalities in adolescent health behaviours across 32 different countries – the role of country-level social mobility. Social Science & Medicine, 310, 115289.

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ABSTRACT

Higher family affluence is associated with healthier behaviours in adolescents, but the strength of this association varies across countries. Differences in social mobility at the country-level, i.e. the extent to which adolescents develop a different socioeconomic status (SES) than their parents, may partially explain why the association between family affluence and adolescent health behaviours is stronger in some countries than in others. Using data from adolescents aged 11–15 years from 32 countries, participating in the 2017/2018 wave of the Health Behaviour in School-aged Children (HBSC) study (N = 185,086), we employed multilevel regression models with cross-level interactions to examine whether country-level social mobility moderates the association between family affluence and adolescent health behaviours (i.e. moderate-to-vigorous physical activity, vigorous physical activity, healthy and unhealthy foods consumption, having breakfast regularly, and weekly smoking). Higher family affluence was more strongly associated with higher levels of adolescent physical activity in countries characterized by high levels of social mobility. No cross-level interactions were found for any of the other health behaviours. Differences in social mobility at the country-level may contribute to cross-national variations in socioeconomic inequalities in adolescent physical activity. Further research can shed light on the mechanisms linking country-level social mobility to inequalities in adolescent physical activity to identify targets for policy and interventions.

1. BACKGROUND

Health behaviours affect health and mortality over the life course, and are influenced by both individual dispositions and the social environment (Mackenbach, 2012; Marmot et al., 2008; Viner et al., 2012). Several important health behaviours deteriorate during the transition from childhood to adolescence. For example, average levels of physical activity (Farooq et al., 2018), fruit and vegetable consumption (Albani et al., 2017), and the frequency of having breakfast (Alexy et al., 2010) tend to decrease, while the consumption of some unhealthy foods, such as soft drinks, tends to increase (Inchley et al., 2020). Meanwhile, substance use, such as tobacco smoking, often has its onset in adolescence (Moor et al., 2015). Importantly, health behaviours established during adolescence frequently continue into adulthood (Wiium et al., 2015), increasing the significance of targeting unhealthy behaviours in this phase of life.

In most high-income countries, higher family socioeconomic status (SES) is associated with more favourable health behaviours in adolescents, but the strength of this association varies considerably across countries (Pförtner et al., 2015; Sigmundová et al., 2019; Zaborskis et al., 2020). In recent years, there have been substantial efforts to identify country-level macroeconomic (e.g., GDP, income inequality), policy (e.g., educational system stratification), and sociocultural factors (e.g., meritocratic attitudes) that could explain why socioeconomic inequalities in adolescent health and health behaviours are larger in some countries than in others (Currie & Morgan, 2020; Dierckens et al., 2020; Weinberg et al., 2021). Most of these studies primarily focussed on inequalities in well-being and physical and mental health (Dierckens et al., 2020; Elgar et al., 2015; Högberg et al., 2019; Weinberg et al., 2021), whilst a smaller number examined the social gradient in health behaviours (Elgar et al., 2015; Pförtner et al., 2015; Rathmann et al., 2016). Overall, findings were mixed and depended on the health outcome or behaviour and country-level factor under consideration. For example, Elgar et al. (2015) reported that higher income inequality was associated with steeper inequalities in psychological and physical symptoms, but not in physical activity.

An area that, to our knowledge, has not been investigated is the contribution of country-level social mobility to cross-national differences in socioeconomic inequalities in adolescent health behaviours. Country-level social mobility refers to the extent to which adolescents develop a different SES to that of their parents within a given society. In countries with low levels of social mobility, children's socioeconomic chances are more strongly related to their parents' socioeconomic resources. If country-level social mobility is high, young people's own SES is less dependent on their parents' SES and more dependent on individual factors, such as skills and talent (Mackenbach, 2012; Simons et al., 2013). One might expect that countries with high levels of social mobility, which are often characterized by equitable social policies, also feature lower health inequalities. Yet,

extensive research on adults has found that socioeconomic health inequalities are often equally strong or even stronger in countries with extensive welfare regimes, such as the Nordic countries, which also have particularly high levels of social mobility (Mackenbach, 2012). Only two studies have focussed specifically on the role of contextual-level social mobility. One study found larger inequalities in mortality in countries with higher levels of social mobility, and the other smaller inequalities in mortality in counties in the USA with more social mobility (Simons et al., 2013; Venkataramani et al., 2020). We were unable to identify any studies on contextual-level social mobility and socioeconomic inequalities in health behaviours (or health) among adolescents.

The inconsistent results of the limited research available may hint at the complexity of the relationship between contextual-level social mobility and health inequalities, yet also give reasons to hypothesize that country-level social mobility may contribute to cross-national variations in the associations between family SES and adolescent health behaviours. There are reasons to think that inequalities in adolescent health behaviours may be larger in countries with either low or high social mobility, without presenting a compelling case as to the direction of this hypothesized interaction. The competing lines of reasoning are outlined below.

*1.) Why the association between family SES and adolescent health behaviours may be larger in countries with **low** social mobility*

In countries characterized by low social mobility, adolescents are less likely to escape the socioeconomic circumstances of their parents. Once low-SES adolescents from such countries realize how unequal their chances are, they may increasingly feel entrapped in the circumstances of their parents and start to believe that they cannot attain long-term socioeconomic goals valued by society, such as esteemed, well-paid employment. This may lead to a decreased orientation towards the future, increased short-term gratification, and seeking alternative means to gain status, such as substance use and delinquent behaviour (Bak & Yi, 2020; Elstad, 2010; Van Houtte & Stevens, 2008). Accordingly, in a Mexican study, adolescents from poor families had worse health behaviours and higher risks of delinquency if they perceived fewer opportunities for upward social mobility for themselves (Ritterman Weintraub et al., 2015).

A second explanation relates to differences in the role of parental resources in less versus more socially mobile countries. A main predictor of adolescents' health behaviours is their own educational level (Kuntz & Lampert, 2013), which may also be thought of as an important mechanism (mediator) connecting parental SES to adolescent health behaviours. In less socially mobile countries, parental SES is a relatively stronger predictor of adolescent educational level. If the association between parental SES and adolescent educational level is stronger in a given country, then also the associations between family SES and adolescent health behaviours should be larger in this country. Conversely, if the association

between parental SES and adolescent educational level is weaker, as expected in high-mobility countries, then also the associations between parental SES and adolescent health behaviours should be weaker.

*2.) Why the association between family SES and adolescent health behaviours may be larger in countries with **high** social mobility*

Higher country-level social mobility over the past generations may, in contrast, have led to widening socioeconomic inequalities in adolescent health behaviours, due to a potential accumulation of individuals with poor health, low cognitive ability, and vulnerable psychological characteristics amongst the lower socioeconomic strata resulting from an increasing salience of health-related selection mechanisms (Mackenbach, 2012; Simons et al., 2013). During the post-war period, social mobility rose substantially in many high-income countries, facilitated by egalitarian policies (Breen, 2010; Mackenbach, 2012). For example, Sweden removed tuition fees, made books, teaching aids, school meals, and school-based health care freely available, and increased the tracking age within the educational system (Breen, 2010; Jonsson & Erikson, 2000; OECD, 1981). Such policies partially offset disadvantages faced by students from low-SES families, allowing individuals with high cognitive ability and resilient psychological characteristics to attain a higher SES than their family of origin (Breen, 2010). This could have led to a homogenization of an increasingly smaller low-SES group in terms of vulnerable psychological characteristics in highly socially mobile countries. Vulnerable psychological characteristics tend to be associated with decreased chances for upward social mobility, as well as poorer health behaviours (Mackenbach, 2012).

A second explanation of why socioeconomic differences in adolescent health behaviours might be larger in more socially mobile countries could relate to stronger collective beliefs in equality of opportunity and meritocracy (i.e., the idea that people get what they deserve) in these countries. These beliefs may lead to a higher tolerance for inequalities (Heiserman et al., 2020; Shariff et al., 2016) and stigmatization of low-SES individuals, who are considered responsible for their life circumstances (Destin, 2020; Simons et al., 2018). In adults, SES-related stigmatization has been associated with poorer mental health (Chan et al., 2022; Simons et al., 2017) and, somewhat less consistently, with substance use (Ahuja et al., 2022; Sartor et al., 2021; Simons et al., 2017). Accordingly, stronger country-level meritocratic beliefs predicted steeper associations of family affluence with life satisfaction and psychosomatic complaints in adolescents (Weinberg et al., 2021). Stronger collective meritocratic beliefs may thus be a potential mechanism linking higher country-level social mobility to more pronounced socioeconomic inequalities in adolescent health behaviours.

1.1. Aim

To the best of our knowledge, this is the first study investigating whether associations between parental SES and adolescent health behaviours differ depending on country-level social mobility. Depending on the line of reasoning used, these associations might be either larger or smaller in countries characterized by high levels of social mobility. Making use of a large cross-national sample of adolescents from 32 countries with varying levels of social mobility, we evaluated health behaviours for which socioeconomic inequalities have most consistently been found in adolescents: physical activity, consumption of healthy (i.e., fruit and vegetables) and unhealthy (i.e., sweets and soft drinks) foods, having breakfast regularly, and smoking (Elgar et al., 2015; Moor et al., 2015; Zaborskis et al., 2020).

2. METHODS

2.1. Sample

We used data from the Health Behaviour in School-aged Children (HBSC) study, an international cross-sectional study investigating health behaviours of adolescents aged 10-16 years in collaboration with the World Health Organization (WHO), which is carried out every four years in a network of countries in the WHO European Zone and North America (Inchley et al., 2018). For our analysis, the 2017/2018 wave of HBSC was used, which included nationally representative samples of adolescents from 44 different countries, who were recruited using cluster sampling with classes within schools as initial sampling units (N = 242,581; mean age = 13.50). The response rates at the individual level exceeded 60% for most participating countries (HBSC Network, 2020). Data were collected using self-report questionnaires, which were completed in the classroom under the supervision of a teacher or trained interviewer. The same standardized protocol was used in all countries, ensuring consistency in measures, sampling methods, and implementation (Inchley et al., 2018). Passive or active consent was obtained from school administrators, parents, and adolescents prior to participation, in line with the regulations in each participating country. We restricted our analysis to 32 countries for which country-level social mobility, as defined below, could be calculated based on the European Social Survey (ESS), yielding a final sample of 185,086 adolescents (76% of the original sample).

2.2. Individual-level variables

2.2.1. Family affluence

Parental socioeconomic status (SES) was assessed using the Family Affluence Scale (FAS) III. The FAS III is a validated adolescent-report questionnaire with six items, which has shown good validity and reliability, as well as higher response rates than other adolescent-report

SES indicators (Torsheim et al., 2016). The FAS III captures different aspects of the family's material resources: car ownership, having one's own bedroom, holidays abroad, computer ownership, dishwasher ownership, and number of bathrooms. The Cronbach's alpha for the FAS III in our study was 0.56. The HBSC countries vary widely in terms of economic conditions, which needs to be accounted for when evaluating family affluence. Therefore, we ridit-transformed the FAS sum scores (range 0-13) separately for each country, gender, and age group (i.e., <12.5 years, 12.5-14.5 years, and >14.5 years) using the 'egenmore' package for Stata (Cox, 2000; Elgar et al., 2017), eliminating endogeneity due to differences on these variables. This yielded a score ranging from 0 (lowest SES) to 1 (highest SES) with a mean of 0.5 for each country. In regression analyses, coefficients can be interpreted as the differences in the outcome between the highest and lowest SES groups (Elgar et al., 2017).

2.2.2. Health behaviours

Only health behaviours for which socioeconomic inequalities (i.e., higher FAS = better health behaviours) have been found in most of the HBSC countries were included in this study as outcomes: physical activity, eating behaviours, and smoking (Elgar et al., 2015; Moor et al., 2015; Voráčová et al., 2016; Zaborskis et al., 2020).

1.) Physical activity

We used two indicators of physical activity: moderate-to-vigorous physical activity (MVPA), and vigorous physical activity (VPA) (Inchley et al., 2018). MVPA was measured using the following item: "Over the past 7 days, on how many days were you physically active for a total of at least 60 minutes per day?". Response options ranged from 0 to 7 days. VPA was assessed with the following question: "Outside school hours: how often do you usually exercise in your free time so much that you get out of breath or sweat? The response options were 'never' = 0, 'less than once a month' = 1, 'once a month' = 2, 'once a week' = 3, '2 to 3 times a week' = 4, '4 to 6 times a week' = 5, and 'every day' = 6.

2.) Eating behaviours

Four questions on foods consumed were asked: "How many times a week do you consume fruit/vegetables/sweetened soft drinks/sweets?" (Inchley et al., 2018). Response options were 'never' = 0, 'less than once a week' = 1, 'once a week' = 2, '2-4 days a week' = 3, '5-6 days a week' = 4, 'once daily' = 5, 'more than once daily' = 6. Two sum scores were created (range 0–12), capturing the consumption of healthy (fruit/vegetables) and unhealthy foods (sweets/soft drinks).

Breakfast consumption was assessed as follows: "How often do you usually have breakfast (more than a glass of milk or fruit juice)?" Response options ranged from 'never' = 0 to 'five days' = 5 for weekdays, and from 'never' = 0 to 'both days' = 2 for weekend days. Responses for weekdays and weekend days were summed to generate a

score representing the number of days per week on which breakfast was consumed. As breakfast on weekends was not assessed in Slovakia, Slovak participants (N = 4,785) were excluded from analyses investigating breakfast consumption.

3.) Tobacco smoking

Smoking was assessed with the following question: "On how many days (if any) did you smoke cigarettes?" Adolescents could respond with the following options with respect to the last 30 days: 'never' = 0, '1-2 days' = 1, '3-5 days' = 2, '6-9 days' = 3, '10-19 days' = 4, '20-29 days' = 5, and '30 days (or more)' = 6. Adolescents' answers were dichotomized into a binary variable indicating weekly smoking (i.e., '3-5 days or more' = 1 vs. '1-2 days or less' = 0).

2.2.3. Individual-level covariates

Furthermore, we included age (range 11–16.5) and gender ('female' = 0, 'male' = 1) as individual-level covariates in the analysis.

2.3. Country-level variables

2.3.1. Social mobility

We used data from the 2018 (Round 9) edition of the European Social Survey (ESS) to calculate country-level social mobility (ESS Data Team, 2021). For five countries, data from previous editions of the ESS were used since they were not available in the 2018 version: Russia and Israel (ESS Round 8, 2016), Albania and Ukraine (ESS Round 6, 2012), and Greece (ESS Round 5, 2010). ESS participants were asked to self-report their own and their parents' highest level of education. To compare countries, educational attainment was recoded into a 7-category variable specifically developed for the ESS (ES-ISCED), which is based on the International Standard Classification of Education (ISCED) scale (**Table 6.S1**) (Schneider, 2020). As no Polish participants were assigned to category I (i.e., less than lower secondary education), and no Finnish, Bulgarian, Portuguese, Ukrainian, and Russian participants to category IIIb (i.e., lower tier upper secondary education), we collapsed category I with II, and IIIb with IIIa, respectively, generating a 5-category measure of educational attainment which is consistent across all 32 countries. In case mothers and fathers differed in terms of their highest educational attainment, we used whichever was higher (dominance method). The sample of ESS participants was restricted to those aged 25-65, as the majority in this age range have already completed their education and not reached retirement, yielding a sample size of N = 37,653 participants (Gugushvili et al., 2019).

Country-level social mobility was operationalized using a relative educational mobility measure (Bukodi et al., 2020), as this was expected to be the most salient measure for adolescents, who are likely to be more aware of their prospects in terms of educational attainment than occupational status or income. Relative measures of educational mobility

compare individuals' positions on the educational ladder (relative to their peers) to their parents' educational position (relative to the parents' own peers). Hence, these measures take into account changes across cohorts in the prevalence of educational credentials (e.g., as consequence of educational expansion) (Gugushvili et al., 2019). A social mobility score was created for each country using uniform difference (UNIDIFF) models and the 'udiff' package for Stata (Breen, 2004; Jann & Seiler, 2019). On their original scale, higher UNIDIFF parameters indicate stronger associations between parents' and children's educational attainment and therefore lower relative educational mobility (Präg & Gugushvili, 2020). To enhance interpretability, we multiplied the scores by -1, such that higher scores indicate more social mobility in a given country.

2.3.2. Country-level covariates

Gross national income (GNI) and national income inequality, as measured with the GINI index for disposable (post-tax, post-transfer) income, were included as country-level covariates in the analysis. GINI coefficients for each country were obtained from the 9th version of the Standardized World Income Inequality Database (Solt, 2019). We used GINI coefficients for 2018 for all countries, except for Iceland, for which only 2017 scores were available. GNI per capita for each country in 2018 was obtained from the World Bank DataBank (World Bank, 2021). The Atlas Method was used to convert GNI scores in national currencies to USD, to facilitate cross-national comparisons.

2.4. Analysis

We conducted multilevel analyses to account for clustering within countries, with individual-level variables at level one and country-level variables at level two. We used linear regression for all continuous outcomes (i.e., MVPA and VPA, healthy foods consumption, unhealthy foods consumption, breakfast consumption), and logistic regression for weekly smoking. All individual-level variables were group mean centred, and all country-level variables were grand mean centred (social mobility and GINI scores) or standardized over the grand mean (GNI) prior to inclusion in multilevel models. Standardization of GNI scores was necessary to facilitate the convergence of models. Maximum likelihood with robust standard errors (MLR) was used as an estimation method to account for the non-normality of the data.

First, we computed null models (for linear regression only) to determine the proportions of variance in health behaviours explained by between-country differences, which is given by the Intraclass Correlation (ICC) (**Tables 6.S2 – S6, Model 0**). Second, we added the individual-level predictors (i.e., family affluence, age, and gender) (**Tables 6.S2 – S7, Model 1**). Third, we added the country-level predictors (i.e., social mobility score, GINI, and GNI) (**Tables 6.S2 – S7, Model 2**). Fourth, we included a random slope for family affluence, to test whether there were significant differences in the associations between family

affluence and health behaviours across countries (**Tables 6.S2 – S7, Model 3**). Finally, we added cross-level interactions between all country-level variables and family affluence (in case random slopes were significant) (**Tables 6.S2 – S7, Model 4**), to test whether the association between family affluence and health behaviours differed depending on country-level social mobility, GINI, and GNI.

As the proportion of missing observations was low for all variables (**Table 6.S8**), missing data were handled using listwise deletion. All analyses were conducted in Stata 16.1 (data processing and UNIDIFF models) and Mplus 8.8 (multilevel models).

3. RESULTS

Table 6.1 shows differences across countries for all health behaviours, mean levels of family affluence, as well as GNI, income inequality, and country-level social mobility.

Table 6.2 shows the results of our final multilevel models. The ICCs were rather small, suggesting that only a small proportion of the variance in the five health behaviours was explained by between-country differences (3.9% for MVPA, 2.4% for VPA, 2.2% for healthy food consumption, 6.3% for unhealthy food consumption, and 4.0% for breakfast consumption). The variance of the random slope for family affluence was significant for all six health behaviours, indicating that the strength of the associations between family affluence and health behaviours differed across countries. Higher family affluence was consistently associated with better health behaviours: adolescents from more affluent families tended to be more physically active, eat more healthily, had breakfast on more days per week, and smoked less frequently than youths from less affluent families. Adolescents from more socially mobile countries on average consumed less unhealthy foods, had breakfast more often, and had lower odds of smoking than adolescents from less socially mobile countries.

We only found significant cross-level interactions between country-level social mobility and family affluence for MVPA and VPA (Beta-coefficient: 0.34; SE 0.13; p=0.009, and 0.31; 0.10; p=0.002, respectively), indicating that the association between higher family affluence and more physical activity was stronger in countries with higher social mobility. To shed further light on these interactions, we plotted them using the Johnson-Neyman technique (**Figure 6.1**) (Bauer & Curran, 2005; Lin, 2020; Muthén & Muthén, 2022). These plots show the associations between family affluence and MVPA and VPA at different levels of country-level social mobility, as predicted by the multilevel models. For both outcomes, the associations were stronger at higher levels of social mobility, yet the confidence intervals never crossed zero within the observed range of social mobility scores (i.e., -0.58 to 0.79). This suggests that, despite the interaction, there was still an association between family affluence and MVPA/VPA in countries with relatively low social mobility, such as Hungary.

These observations are largely confirmed by country-wise linear regression models of the associations of family affluence with MVPA and VPA, of which the resulting beta-coefficients are presented in **Figure 6.2** (in order of the social mobility score of each country).

FIGURE 6.1 Johnson–Neyman plots depicting the conditional associations of family affluence with moderate-to-vigorous physical activity (MVPA) and vigorous physical activity (VPA) at different levels of country-level social mobility; 95% confidence intervals (CIs) are represented by dotted lines

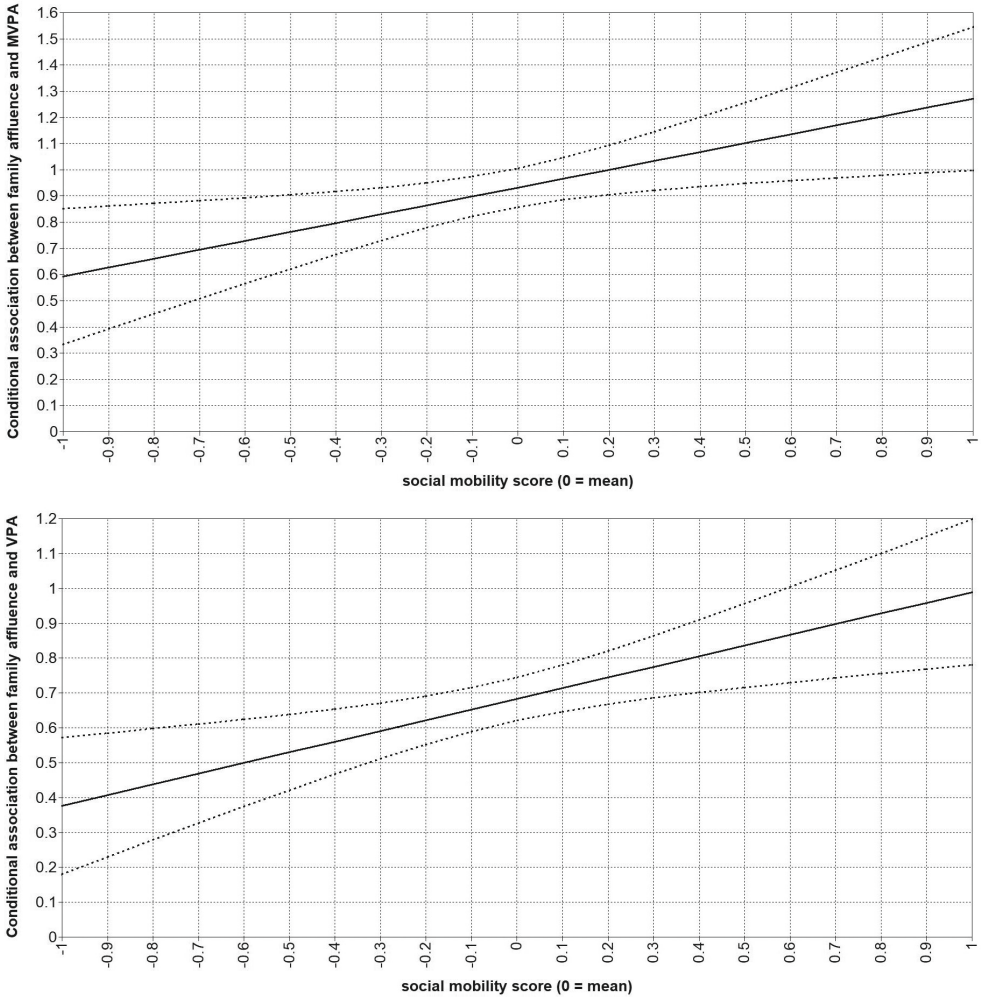


TABLE 6.1 Characteristics of adolescents participating in the HBSC study (2018, N = 185,086) and their countries (N=32)

			Country-level characteristics			Individual-level characteristics		
			Social mobility[c]	GNI	GINI	Family affluence sum score	MVPA	VPA
Country		N				Mean (SD)	Mean (SD)	Mean (SD)
All countries[a]	-	185,086	0.00	33,544	0.30	8.36 (2.48)	4.08 (2.06)	4.00 (1.59)
Albania	AL	1,765	-0.02	4,860	0.38	6.08 (2.83)	4.11 (2.07)	4.14 (1.65)
Austria	AT	4,129	-0.29	48,950	0.28	9.25 (1.97)	4.30 (2.01)	4.34 (1.33)
Belgium	BE	9,911	0.08	46,010	0.26	8.83 (2.20)	3.98 (2.08)	3.85 (1.58)
Bulgaria	BG	4,548	-0.30	8,530	0.38	7.25 (2.29)	4.19 (2.21)	4.06 (1.79)
Croatia	HR	5,169	-0.12	14,280	0.29	7.60 (2.35)	4.39 (2.02)	3.73 (1.73)
Czech Rep.	CZ	11,564	-0.33	20,560	0.24	8.05 (2.36)	4.18 (1.97)	4.01 (1.51)
Denmark	DK	3,181	0.20	61,260	0.27	9.61 (1.86)	3.55 (1.98)	3.87 (1.42)
Estonia	EE	4,725	0.27	21,300	0.31	8.20 (2.27)	4.00 (1.99)	3.95 (1.48)
Finland	FI	3,146	0.21	48,160	0.26	8.84 (1.92)	4.80 (1.93)	4.43 (1.35)
France	FR	9,170	0.10	41,150	0.30	8.52 (2.10)	3.41 (2.00)	3.75 (1.65)
Germany	DE	4,347	-0.05	47,410	0.30	9.32 (2.07)	3.81 (1.93)	4.10 (1.44)
Greece	GR	3,863	-0.22	19,060	0.32	6.76 (2.22)	4.03 (2.03)	4.30 (1.49)
Hungary	HU	3,789	-0.58	14,980	0.28	7.26 (2.55)	4.05 (2.12)	4.11 (1.60)
Iceland	IS	6,996	0.79	67,760	0.25	9.20 (1.80)	4.45 (2.04)	4.25 (1.53)
Ireland	IE	3,833	-0.07	59,280	0.30	9.22 (2.19)	4.68 (1.99)	4.42 (1.44)
Israel	IL	7,712	-0.09	41,320	0.34	8.47 (2.69)	2.98 (2.22)	3.49 (1.91)
Italy	IT	4,144	-0.11	33,810	0.34	7.83 (2.27)	3.36 (1.96)	3.68 (1.62)
Latvia	LV	4,412	0.16	16,530	0.35	7.20 (2.49)	3.99 (2.09)	4.09 (1.48)
Lithuania	LT	3,797	0.08	17,450	0.36	6.95 (2.50)	4.29 (1.96)	3.76 (1.73)
Netherlands	NL	4,698	0.25	51,250	0.27	8.98 (1.84)	4.30 (1.96)	4.24 (1.31)
Norway	NO	3,127	0.22	80,320	0.26	9.88 (1.72)	4.30 (1.87)	4.24 (1.30)
Poland	PL	5,224	-0.04	14,150	0.29	7.76 (2.31)	4.22 (1.97)	3.71 (1.66)
Portugal	PT	6,126	-0.14	22,030	0.32	8.09 (2.30)	3.49 (1.87)	3.53 (1.82)
Russia	RU	4,281	0.18	10,250	0.32	6.44 (2.36)	3.67 (2.07)	3.95 (1.67)
Serbia	RS	3,933	-0.38	6,410	0.33	7.26 (2.50)	4.72 (2.16)	4.20 (1.76)
Slovakia[b]	SK	4,785	-0.35	18,320	0.23	7.72 (2.47)	4.38 (2.06)	4.30 (1.58)
Slovenia	SI	5,667	-0.25	24,610	0.25	9.38 (2.09)	4.53 (1.97)	4.28 (1.43)
Spain	ES	4,320	0.33	29,280	0.33	8.57 (2.27)	4.39 (1.97)	3.94 (1.58)
Sweden	SE	4,185	0.14	55,640	0.26	9.35 (1.98)	3.93 (2.02)	3.81 (1.54)
Switzerland	CH	7,510	-0.10	83,280	0.30	9.81 (1.97)	4.16 (1.87)	4.05 (1.32)
UK	GB	24,369	0.34	42,410	0.32	9.16 (2.35)	4.23 (1.99)	4.03 (1.56)
Ukraine	UA	6,660	0.08	2,800	0.27	5.64 (2.34)	4.28 (2.15)	4.22 (1.57)

SD = standard deviation; MVPA = moderate-to-vigorous physical activity; VPA = vigorous physical activity.[a] for the country-level variables, the mean of all 32 countries is given;[b] data on breakfast consumption is missing for Slovakia;[c] grand mean centred.

Healthy foods consumed	Unhealthy foods consumed	Days per week with breakfast	Weekly smoking	Male gender	Age
Mean (SD)	Mean (SD)	Mean (SD)	N (%)	N (%)	Mean (SD)
7.63 (2.87)	5.55 (2.80)	5.25 (2.27)	8,476 (4.82)	91,277 (49.32)	13.51 (1.63)
8.90 (2.75)	6.62 (3.20)	4.76 (2.43)	65 (3.81)	803 (45.50)	13.55 (1.52)
7.54 (2.75)	5.71 (2.76)	4.33 (2.53)	182 (4.51)	2,036 (49.31)	13.28 (1.62)
8.17 (2.84)	6.18 (3.02)	5.42 (2.21)	291 (3.04)	4,935 (49.79)	13.33 (1.70)
7.58 (3.01)	6.40 (3.19)	5.08 (2.11)	576 (12.66)	2,200 (48.37)	13.53 (1.65)
7.14 (2.70)	5.75 (2.82)	5.08 (2.21)	456 (9.19)	2,635 (50.98)	13.80 (1.70)
7.94 (2.80)	5.42 (2.71)	5.04 (2.33)	608 (5.33)	5,821 (50.34)	13.37 (1.66)
7.97 (2.76)	4.69 (2.18)	5.86 (1.92)	104 (3.31)	1,545 (48.57)	13.33 (1.61)
7.80 (2.70)	5.21 (2.32)	5.38 (2.17)	260 (5.64)	2,369 (50.14)	13.78 (1.64)
6.94 (2.71)	4.48 (1.91)	5.62 (2.06)	168 (5.43)	1,562 (49.65)	13.92 (1.61)
7.27 (3.01)	5.66 (3.07)	5.41 (2.25)	352 (3.91)	4,539 (49.50)	13.30 (1.46)
7.33 (2.78)	5.57 (2.61)	5.00 (2.40)	174 (4.06)	2,041 (46.95)	13.41 (1.68)
7.23 (2.50)	4.76 (2.30)	4.71 (2.37)	196 (5.14)	1,927 (49.88)	13.82 (1.66)
7.11 (2.86)	6.13 (2.98)	4.57 (2.41)	178 (6.66)	1,788 (47.19)	13.52 (1.63)
7.62 (2.87)	3.63 (2.19)	5.49 (2.16)	136 (2.00)	3,510 (50.17)	13.60 (1.63)
8.03 (2.82)	4.86 (2.36)	5.89 (1.92)	95 (2.52)	1,940 (50.61)	13.41 (1.56)
8.10 (3.44)	6.45 (3.33)	4.69 (2.43)	218 (4.10)	3,482 (45.15)	13.63 (1.59)
6.95 (2.89)	5.63 (2.71)	5.02 (2.48)	317 (7.74)	1,998 (48.21)	13.68 (1.62)
7.13 (2.53)	5.28 (2.37)	5.35 (2.17)	275 (6.35)	2,188 (49.59)	13.47 (1.65)
7.52 (2.81)	5.40 (2.63)	4.93 (2.37)	373 (10.05)	1,914 (50.41)	13.70 (1.65)
7.96 (2.18)	6.19 (2.60)	6.24 (1.58)	151 (3.23)	2,287 (48.68)	13.51 (1.60)
7.63 (2.68)	4.65 (2.10)	5.91 (1.82)	42 (3.02)	1,517 (48.51)	13.02 (1.61)
7.61 (2.78)	5.76 (2.83)	5.44 (2.16)	265 (5.12)	2,570 (49.20)	13.59 (1.66)
7.37 (3.06)	4.97 (2.78)	5.85 (1.87)	188 (3.19)	2,926 (47.76)	13.31 (1.53)
7.59 (2.77)	5.51 (2.74)	5.19 (2.22)	157 (4.08)	2,043 (47.72)	13.82 (1.66)
7.94 (2.90)	6.32 (3.18)	5.41 (2.06)	284 (7.38)	1,945 (49.45)	13.98 (1.68)
7.63 (2.92)	6.31 (2.97)	-	302 (6.71)	2,455 (51.31)	13.32 (1.52)
7.83 (2.78)	4.57 (2.46)	4.65 (2.37)	241 (4.28)	2,879 (50.80)	13.59 (1.63)
7.25 (2.76)	5.01 (2.68)	5.54 (2.10)	186 (4.38)	2,088 (48.33)	13.62 (1.62)
7.43 (2.80)	4.73 (2.17)	5.75 (2.00)	146 (3.64)	2,081 (49.73)	13.63 (1.64)
8.07 (2.70)	6.19 (2.66)	4.96 (2.39)	290 (3.89)	3,785 (50.40)	13.42 (1.60)
7.39 (3.03)	5.87 (2.70)	5.01 (2.44)	891 (3.75)	12,221 (50.15)	13.50 (1.59)
8.35 (2.75)	6.02 (2.81)	5.77 (1.97)	309 (4.76)	3,247 (48.75)	13.40 (1.63)

TABLE 6.2 Multilevel regression models for adolescent health behaviours with country-level predictors and cross-level interaction terms between country-level predictors and individual-level family affluence in the HBSC study (2017/2018, N participants = 185,086, N countries = 32)

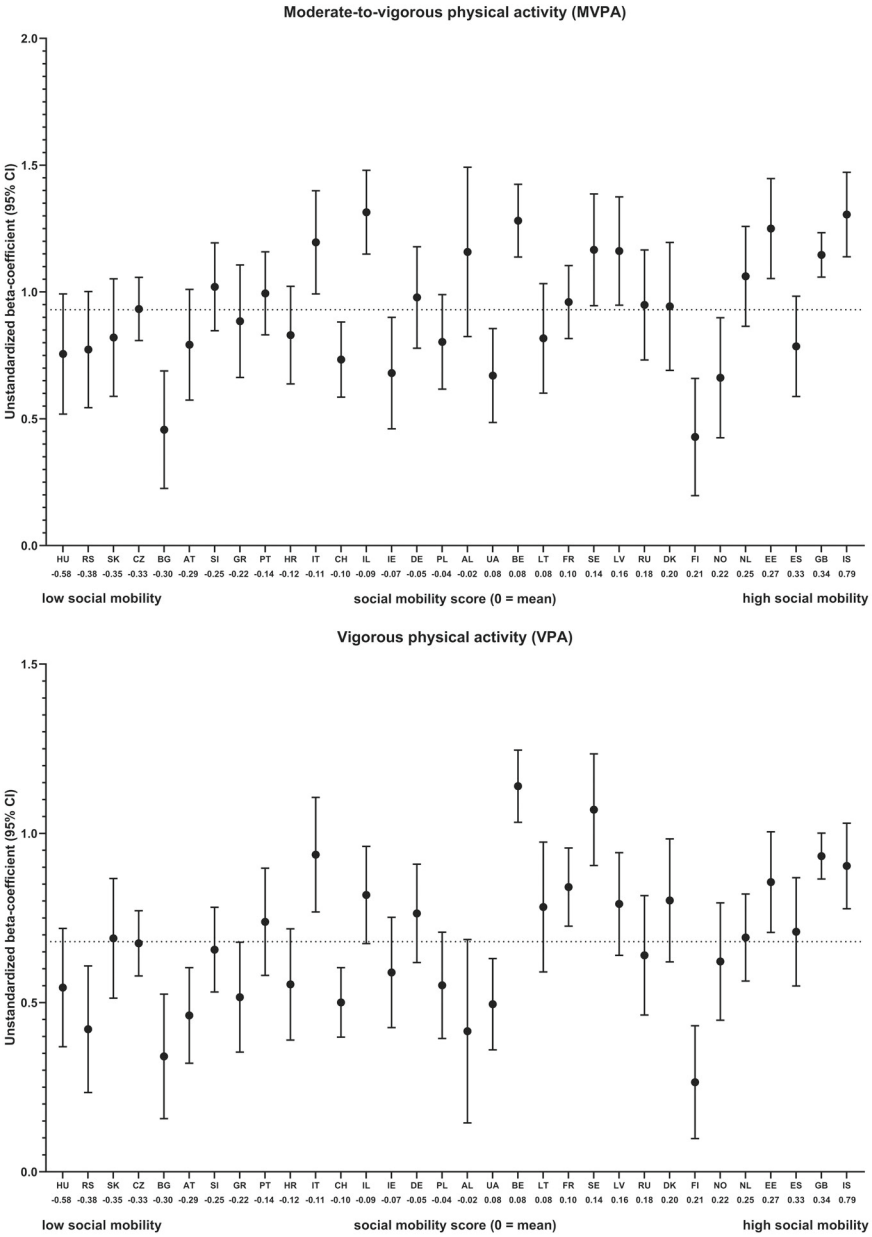
	MVPA			**VPA**			**Healthy foods consumed**		
	N = 173,580			**N = 174,119**			**N = 172,240**		
	B	**(SE)**	**p-value**	**B**	**(SE)**	**P-value**	**B**	**(SE)**	**p-value**
Individual-level									
Male gender	**0.50**	**(0.03)**	**<0.001**	**0.44**	**(0.04)**	**<0.001**	**-0.54**	**(0.03)**	**<0.001**
Age	**-0.17**	**(0.02)**	**<0.001**	**-0.12**	**(0.01)**	**<0.001**	**-0.16**	**(0.01)**	**<0.001**
Family affluence	**0.93**	**(0.04)**	**<0.001**	**0.68**	**(0.03)**	**<0.001**	**1.25**	**(0.06)**	**<0.001**
Residual variance of the outcome at the individual level	**3.83**	**(0.07)**	**<0.001**	**2.33**	**(0.08)**	**<0.001**	**7.75**	**(0.21)**	**<0.001**
Country-level									
Social mobility	0.10	(0.23)	0.670	-0.06	(0.12)	0.628	-0.01	(0.26)	0.957
GINI	**-3.87**	**(1.75)**	**0.027**	-2.08	(1.07)	0.053	-0.12	(2.57)	0.964
GNI	-0.08	(0.07)	0.298	0.01	(0.04)	0.829	0.00	(0.09)	0.999
Intercept	**4.10**	**(0.07)**	**<0.001**	**4.03**	**(0.04)**	**<0.001**	**7.64**	**(0.08)**	**<0.001**
Residual variance of the outcome at the country level	**0.15**	**(0.03)**	**<0.001**	**0.05**	**(0.01)**	**<0.001**	**0.18**	**(0.05)**	**<0.001**
Residual variance of the slope of family affluence	**0.04**	**(0.01)**	**0.002**	**0.03**	**(0.01)**	**0.004**	**0.08**	**(0.03)**	**0.005**
Cross-level interactions									
Family affluence × social mobility	**0.34**	**(0.13)**	**0.009**	**0.31**	**(0.10)**	**0.002**	0.17	(0.25)	0.481
Family affluence × GINI	0.12	(1.22)	0.921	-0.44	(0.89)	0.621	1.04	(1.37)	0.446
Family affluence × GNI	-0.01	(0.05)	0.892	0.02	(0.04)	0.586	-0.03	(0.05)	0.549
Intraclass correlation coefficient	3.9%			2.4%			2.2%		

TABLE 6.2 Continued

	Unhealthy foods consumed			Days per week with breakfast[a]			Weekly smoking			
	N = 172,224			N = 163,658			N = 168,271			
	B	(SE)	p-value	B	(SE)	P-value	B	(SE)	OR	p-value
Individual-level										
Male gender	**0.30**	**(0.03)**	**<0.001**	**0.31**	**(0.05)**	**<0.001**	0.05	(0.07)	1.05	0.525
Age	**0.12**	**(0.01)**	**<0.001**	**-0.21**	**(0.02)**	**<0.001**	**0.70**	**(0.03)**	**2.01**	**<0.001**
Family affluence	**-0.19**	**(0.07)**	**0.006**	**0.64**	**(0.06)**	**<0.001**	**-0.29**	**(0.06)**	**0.75**	**<0.001**
Residual variance of the outcome at the individual level	**7.24**	**(0.28)**	**<0.001**	**4.77**	**(0.16)**	**<0.001**	-	-	-	-
Country-level										
Social mobility	**-1.16**	**(0.37)**	**0.002**	**0.85**	**(0.32)**	**0.008**	**-0.57**	**(0.15)**	**0.56**	**<0.001**
GINI	5.47	(2.86)	0.056	-3.00	(1.70)	0.076	2.87	(1.74)	17.67	0.100
GNI	-0.06	(0.12)	0.626	-0.01	(0.07)	0.892	**-0.17**	**(0.05)**	**0.84**	**0.001**
Intercept/threshold	**5.49**	**(0.10)**	**<0.001**	**5.28**	**(0.07)**	**<0.001**	**3.53**	**(0.07)**	**34.02**	**<0.001**
Residual variance of the outcome at the country level	**0.31**	**(0.07)**	**<0.001**	**0.14**	**(0.03)**	**<0.001**	**0.09**	**(0.03)**	**1.10**	**0.001**
Residual variance of the slope of family affluence	**0.13**	**(0.03)**	**<0.001**	**0.09**	**(0.02)**	**<0.001**	**0.04**	**(0.02)**	**1.04**	**0.023**
Cross-level interactions										
Family affluence × social mobility	0.47	(0.31)	0.134	0.09	(0.18)	0.619	-0.31	(0.25)	0.74	0.219
Family affluence × GINI	-1.87	(1.94)	0.335	-0.75	(1.79)	0.673	1.89	(1.50)	6.64	0.208
Family affluence × GNI	-0.22	(0.12)	0.061	0.06	(0.06)	0.322	-0.03	(0.08)	0.97	0.714
Intraclass correlation coefficient	6.3%			4.0%			-			

B = unstandardized regression coefficient; SD = standard deviation; OR = odds ratio; MVPA = moderate-to-vigorous physical activity; VPA = vigorous physical activity. Unstandardized linear regressions were used for continuous outcomes (i.e., MVPA, VPA, healthy/unhealthy foods consumption, breakfast consumption). Logistic regression was used for weekly smoking. [a] Data on breakfast consumption is missing for Slovakia. Therefore, this analysis is based on 31 countries.

FIGURE 6.2 Country-wise adjusted[a] unstandardized linear regressions of the associations between family affluence and moderate-to-vigorous (MVPA) and vigorous physical activity (VPA) according to the level of country-level social mobility, in 32 countries participating in the HBSC study (2017/2018, N participants = 185,086, N countries = 32)



[a] adjusted for gender and age; CI = confidence interval; the dotted lines represent the unstandardized linear regression coefficients for family affluence for all 185,086 participants, after controlling for clustering within countries, individual- and country-level variables, as well as cross-level interactions, as reported in ***Table 6.2****.*

4. DISCUSSION

In line with past research, higher family affluence was significantly associated with better health behaviours (i.e., higher levels of moderate-to-vigorous and vigorous physical activity, increased consumption of healthy foods, lower consumption of unhealthy foods, having breakfast more frequently, lower odds of weekly smoking). As expected, we found that associations between family affluence and health behaviours varied across countries (Elgar et al., 2015; Pförtner et al., 2015; Rathmann et al., 2016). Furthermore, we found that higher levels of country-level social mobility were associated with somewhat better health behaviours in the adolescent population: on average, adolescents from more socially mobile countries tended to consume less unhealthy foods, had breakfast more frequently and tended to smoke less. Finally, the positive association between family affluence and physical activity (both MVPA and VPA) was stronger in more socially mobile countries. No cross-level interactions with country-level social mobility were found for any of the other health behaviours.

4.1. Interpretation of findings

In this study, we investigated whether associations between family affluence and adolescent health behaviours differ depending on country-level social mobility. Two competing lines of reasoning suggest that socioeconomic inequalities in adolescent health behaviours are either smaller or larger in countries with higher levels of social mobility.

The first line of reasoning was not supported by our results, as for none of the health-behaviours investigated socioeconomic differences based on parental SES were larger in countries with low levels of social mobility. It is possible that adolescents from low-SES backgrounds living in these countries may not be aware of the structural inequalities of opportunity in their societies and may instead be more strongly affected by factors in their immediate social environment, for example at school. It has been found that socioeconomic inequalities in adolescent smoking and mental health tend to be smaller in highly stratified educational systems, which more commonly characterize countries with low social mobility (Hanushek & Wößmann, 2006; Högberg et al., 2019; Pekkarinen, 2018; Rathmann et al., 2016; Van de Werfhorst & Mijs, 2010). A potential explanation for this surprising result could be that socially disadvantaged adolescents may experience less pressure from social comparison in their immediate social environment if grouped together with adolescents from similar backgrounds at school (Högberg et al., 2019), and this could at least temporarily offset the negative psychosocial consequences of a lack of social mobility in a given society. The differences in the educational systems between high- and low mobility countries also raise the possibility of variations in the associations between adolescents' own educational level and their health behaviours, depending on country-level social mobility. This could also explain why putatively stronger associations

between parental SES and adolescents' own educational level in low-mobility countries did not lead to wider socioeconomic inequalities in these countries. Future studies may attempt to delineate potential differences in associations between measures of adolescents' own educational level and health behaviours between countries with high and low levels of social mobility.

We found only partial support for the second line of reasoning, as we detected larger inequalities in adolescent physical activity (both MVPA and VPA) in countries characterized by more social mobility. Country-level social mobility was not associated with inequalities in any of the other health behaviours we have assessed. These results corroborate previous international findings using HBSC data, which also found that interactions between country-level characteristics and family affluence are not comparable across all dimensions of adolescent health (behaviours) (Elgar et al., 2015). Since we only found cross-level interactions for two out of six outcomes, our findings are not fully compatible with the two explanations for potentially larger inequalities in more socially mobile countries explained above (i.e., increased health-related selection, stronger country-level meritocratic beliefs). Psychological characteristics relevant to health-related selection, such as behavioural control (Schmengler et al., 2022), predict multiple health behaviours simultaneously, rather than only physical activity specifically (Audiffren & André, 2019; Padin et al., 2017). Similarly, country-level meritocratic beliefs, which are potentially more prevalent in socially mobile societies, are unlikely to explain why we found a significant interaction for physical activity only, as SES-related stigmatization, partly by affecting mental health (Simons et al., 2018), would be expected to be associated with multiple health behaviours, including diet and smoking.

Explanations of why inequalities in physical activity are larger in socially mobile countries might instead be sought in factors specifically related to physical activity, but not necessarily other health behaviours. Future studies might, for example, focus on differences in the educational systems between high- and low mobility countries (Hanushek & Wößmann, 2006; Pekkarinen, 2018), as stated above, specifically in relation to adolescents' participation in physical activity, as well as cultural differences in beliefs on the importance of sports for health and personal/group identity (Bann et al., 2019). Educational systems vary widely across countries in terms of curriculum time allocated to physical activity and the extent to which participation in sports is enforced at school (Bann et al., 2019). This could have implications for differences across countries in the engagement of low-SES adolescents in physical activity both as part of the formal curriculum, and during extracurricular activities. Furthermore, future studies might seek to gain understanding of the role of sports as source of a positive social identity (Hughson, 2009), sense of community, and belonging for low-SES adolescents in high and low social mobility countries (Eriksen & Stefansen, 2021). For example, it is possible that in low-mobility countries, which are characterized more strongly by traditional class structures and their transmission across

generations, sports may be important for forming a positive "working class" social identity in low-SES adolescents (Hughson, 2009). Crucially, it is likely that educational, policy, and sociocultural factors interact in complex ways in shaping inequalities in adolescent physical activity. A complex system approach might therefore help understand why we find larger inequalities in physical activity in countries with high levels of social mobility (Diez Roux, 2011; Holdsworth et al., 2017).

An additional finding that warrants reflection is the association between higher country-level social mobility and better health behaviours, which was found for three out of six behaviours (i.e., less consumption of unhealthy foods, having breakfast more regularly, lower prevalence of smoking). This echoes previous studies showing that more egalitarian societies (e.g., in terms of social mobility (Gugushvili & Kaiser, 2020), and income equality (Pickett & Wilkinson, 2015)) often feature better overall population health, even whilst health inequalities in such countries are not necessarily smaller (Mackenbach, 2012; Simons et al., 2013). Country-level social mobility therefore also seems to be related to health behaviours in the whole population of adolescents rather than specifically those from low-affluent families. Socially mobile countries are frequently characterized by more generous welfare regimes, and higher educational spending (OECD, 2018) which, to a certain extent, may benefit adolescents from all socioeconomic groups similarly (Pickett & Wilkinson, 2015).

4.2 Strengths and limitations

Key strengths of this study are its large sample size and its cross-national approach, reflecting a wide range of socio-cultural, economic, and political contexts, whilst including identical measures of health behaviours and family affluence across 32 different countries (Inchley et al., 2018). However, our study also has several limitations. First, associations of the FAS with more conventional measures of family SES (parental income, educational level, occupational level) are often moderate at best (Corell et al., 2021), suggesting that we might not have tapped into all aspects of families' socioeconomic circumstances. Unfortunately, information on parental educational attainment, occupational status, and income are difficult to collect in exclusively adolescent-report surveys like HBSC, as adolescents often do not know detailed information on these variables from their parents (Hartley et al., 2016). Nevertheless, the FAS III is one of the most reliable and valid self-report measures of family SES of adolescents, with high agreement between parent and child-report scores (Andersen et al., 2008; Hartley et al., 2016). Information on other important dimensions of adolescents' SES, such as own educational level, were also not included in our study. Previous research suggests that different aspects of SES have distinct associations with health behaviours (Kuntz & Lampert, 2013; Schmengler et al., 2022) and may also interact differently with country-level factors, as compared to family affluence (Weinberg et al., 2021). Future studies may include more detailed assessments of SES, including adolescents'

educational level, as well as parent-report questionnaires to additionally collect information on parents' educational level, income, and occupational status.

Second, our approach to measuring social mobility cannot distinguish between the extent of upward and downward social mobility in a given society, which may differ substantially between countries with similar overall social mobility (Bukodi et al., 2017), yet contextual levels of upward and downward mobility may have distinct associations with inequalities in adolescent health behaviours. Future research may study the extent of upward and downward social mobility in relation to health inequalities. Third, we cannot rule out residual confounding by country-level variables we have not assessed, and which are associated with both inequalities in health behaviours and country-level social mobility. While we have controlled for income inequality and GNI, we have not accounted for differences in policy factors that characterize countries with high vs. low social mobility, such as those related to the educational system. Further studies could evaluate such factors as a potential explanation of the wider inequalities in physical activity we found in highly socially mobile countries. Fourth, caution must be applied when interpreting our results in light of the 'ecological fallacy', as the units of analysis for social mobility were at the aggregate/country-level (Carneiro & Howard, 2011). We therefore do not know about the extent of social mobility individual HBSC participants were exposed to in their specific social context (e.g., school, town, family, etc.). Finally, limitations apply to the external validity of our study. Particularly adolescents from very low-SES families tend to be underrepresented in epidemiologic studies (Fakkel et al., 2020), which could lead to an underestimation of the associations between family affluence and health behaviours. Luckily, response rates in school-based surveys like HBSC tend to be higher than in studies where adolescents are approached outside the educational context (Dey et al., 2021).

5. CONCLUSION

Few studies have focussed on the role of country-level social mobility as potential explanation for cross-national differences in socioeconomic health inequalities. This study contributes to the literature by investigating the role of country-level social mobility in socioeconomic inequalities in adolescent health behaviours. While higher country-level social mobility predicted more inequalities in physical activity only, it was generally associated with somewhat better health behaviours (i.e., less tobacco use, lower consumption of unhealthy foods, more frequent breakfast) in adolescents. To identify targets for intervention, future research should focus on identifying socio-cultural and policy factors specifically related to inequalities in adolescent physical activity, which characterize countries with low and high levels of social mobility.

DATA AVAILABILITY

Data from the HBSC study can be obtained from the HBSC Data Management Centre in accordance with the HBSC data access policy. Further information on accessing HBSC data is available from: https://www.uib.no/en/hbscdata. Data from the European Social Survey are freely available from: https://www.europeansocialsurvey.org/. The syntax for our analyses can be obtained from: https://github.com/hschmengler/Socioeconomic-inequalities-in-adolescent-health-behaviours-across-32-different-countries.

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(Olga Balakireva and Dasha Pavlova). For details, see http://www.hbsc.org. Some of the data in this publication are based on the European Social Survey (ESS). The data are prepared and made available by NSD Norwegian Centre for Research Data. NSD is not responsible for the analyses/interpretation of the data presented here. More information here: https://www.europeansocialsurvey.org/data/conditions_of_use.html.

DECLARATION OF COMPETING INTEREST

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

ETHICAL STANDARD STATEMENT

Passive or active consent for participation in the HBSC Study was obtained from school administrators, parents, and adolescents prior to participation, in line with the regulations in each participating country. Institutional ethical approval was obtained in each participating country.

OPEN ACCESS

AUTHOR CONTRIBUTIONS

Heiko Schmengler: Conceptualization, Methodology, Formal analysis, Visualization, Writing - Original Draft, Writing - Review & Editing, Margot Peeters: Supervision, Project administration, Writing - Review & Editing, Gonneke W. J. M. Stevens: Project administration, Data Curation, Resources, Funding acquisition, Writing - Review & Editing, Anton E. Kunst: Writing - Review & Editing, Katrijn Delaruelle: Writing - Review & Editing, Maxim Dierckens: Writing - Review & Editing, Lorena Charrier: Writing - Review & Editing, Dom Weinberg: Writing - Review & Editing, Albertine J. Oldehinkel: Supervision, Writing - Review & Editing, Wilma A. M. Vollebergh: Conceptualization, Supervision, Project administration, Funding acquisition, Writing - Review & Editing.

6

CHAPTER

7

SUMMARY AND GENERAL DISCUSSION

This dissertation explored the mechanisms underlying the development of socioeconomic inequalities in mental health and health behaviours in adolescence and young adulthood. First, we investigated the role of social causation and health related-selection mechanisms in the development of educational inequalities in adolescent and young adult substance use (specifically, alcohol use and smoking) (**Chapters 2 and 4**), as well as mental health (specifically, externalizing behaviours and attention problems) (**Chapter 3**). To better understand the role of the immediate social context (i.e., the microsystem) in direct health-related selection processes, we subsequently investigated whether associations between ADHD symptoms and lower education are mediated by lower family functioning and receiving less social support from teachers and peers, and whether these associations differ between adolescents who experience high levels of family functioning and social support and those who do not (**Chapter 5**). Lastly, this dissertation explored whether country-level social mobility in the previous generations is associated with the extent of inequalities in adolescent health behaviours by family affluence (**Chapter 6**). This chapter (**Chapter 7**) first summarizes the main findings of each of the five empirical studies of this dissertation (see also **Table 7.1**), and then discusses the scientific and practical relevance of these findings, whilst also providing directions for future research.

1. SUMMARY OF RESULTS

1.1. The interplay of social causation and health-related selection mechanisms in the development of educational inequalities in substance use and mental health

The role of social causation and health-related selection mechanisms (both direct and indirect) differed depending on the developmental period and on which health behaviour or mental health problem was considered.

1.1.1. Direct health-related selection for attention problems/ADHD symptoms

Direct health-related selection was mainly found regarding attention problems (**Chapter 3**), and relatedly ADHD symptoms (**Chapter 5**), which consistently predicted decreases in education over the whole course of adolescence and young adulthood. In addition, we found that associations between ADHD symptoms and lower education were not mediated by key features of adolescents' social context (i.e., family functioning, social support by teachers, and social support by classmates). Furthermore, we did not find differences in these associations depending on the level of family functioning and social support (**Chapter 5**). Also externalizing behaviour was associated with decreases in education from early adolescence to young adulthood, but these associations disappeared after adjusting for attention problems (**Chapter 3**).

1.1.2. Largely no indirect health-related selection related to childhood cognitive skills

We did not find much evidence of indirect health-related selection related to cognitive skills in childhood. As expected, both higher IQ and effortful control strongly predicted selection into a higher educational trajectory. However, in most analyses, a high IQ was not directly associated with less externalizing behaviour (**Chapter 3**), attention problems (**Chapter 3**), alcohol use (**Chapter 2**), and smoking (**Chapter 4**), and, similarly, high effortful control was not directly associated with less drinking (**Chapter 2**), and tobacco use (**Chapter 4**). Instead, we found that both IQ and effortful control were related to substance use indirectly through their associations with adolescents' and young adults' educational trajectories, which in turn predicted drinking (**Chapter 2**) and smoking behaviours (**Chapter 4)**. Our analyses using polygenic scores (PGSs) were suggestive of some indirect health-related selection related to genetic factors when considering educational inequalities in smoking (**Chapter 4**). Genetic dispositions for smoking and lower educational attainment were significantly associated with smoking behaviours, as well as being in a lower educational trajectory, indicating that genetic factors serve as shared predictors of both phenotypes. However, we observed only small reductions in associations between educational level and smoking after adjusting for genetic risk factors, which may suggest that the impact of indirect health-related selection due to these factors is only minor. Nevertheless, it is important to note that currently PGSs capture only part of the genetic variance associated with smoking and educational attainment (Pingault et al., 2021), which means that statistical adjustment for PGSs could underestimate the extent of indirect health-related selection related to genetics.

1.1.3. Social causation for alcohol use and smoking, as well as externalizing behaviour

When considering educational differences in adolescent and young adult substance use, social causation processes appeared to be present. Young adolescents in the lower educational trajectories increased in their drinking behaviour more strongly three years later than their peers in the higher tracks (**Chapter 2**). In young adulthood, influences of the educational context were in the opposite direction, increasing the drinking behaviour of those in the higher educational trajectories. Associations between educational level and smoking did not reduce much after controlling for genetic risk factors, cognitive skills, and other important covariates, suggesting that these associations may indeed reflect the role of educational differences in the social context (**Chapter 4**). Further in line with social causation, higher parental SES predicted selection into a higher educational trajectory and less drinking in early adolescence, but stronger increases in alcohol use in young adulthood (**Chapter 2**). When considering mental health, parental SES seemed more important than youngsters' educational level in social causation processes. After controlling for potential

confounders, educational level was neither associated with changes in attention problems nor externalizing behaviour over the course of adolescence and young adulthood. Instead, we found that lower parental SES predicted more attention problems and externalizing behaviour in early adolescence, and additionally contributed to a bivariate association between a lower educational level in early adolescence and increases in externalizing behaviour in mid-adolescence (**Chapter 3**).

1.2. Country-level social mobility only of importance for the association between family affluence and physical activity

Lastly, we found that adolescents from low-affluent families living in countries with high levels of social mobility in the previous generations tended to be less physically active than their peers from similar socioeconomic backgrounds in countries with lower levels of social mobility (**Chapter 6**). Country-level social mobility did not moderate inequalities in any other of the health behaviours studied (i.e., healthy and unhealthy foods consumed, having breakfast regularly, and smoking). Taken together, our findings highlight the complexity of the relationship between contextual-level social mobility and health inequalities.

2. REFLECTION ON THE MAJOR FINDINGS AND THEIR THEORETICAL AND PRACTICAL IMPLICATIONS

Below, I discuss the theoretical and practical implications of the key findings of this dissertation on the mechanisms studied (i.e., direct and indirect health-related selection and social causation), as well as on the role of country-level social mobility, in the emergence of socioeconomic inequalities in adolescents' and young adults' mental health and health behaviours.

2.1. The interplay of social causation and health-related selection mechanisms in the development of educational inequalities in substance use and mental health

2.1.1. Direct health-related selection for attention problems/ADHD symptoms

Attention problems (**Chapter 3**), and relatedly ADHD symptoms (**Chapter 5**), were consistently associated with decreases in educational level throughout adolescence and young adulthood. Our findings are in line with studies using clinical diagnoses of ADHD, which found that ADHD is strongly associated with adverse long-term educational outcomes, such as ineligibility for upper secondary education (Bortes et al., 2022) or dropout from secondary education (Mikkonen et al., 2018). Recent findings show that also subclinical levels of ADHD symptoms are associated with academic problems (Zendarski

et al., 2022). These results are consistent with our findings, which were based on the whole spectrum of attention problems (or ADHD symptoms), including those in the subclinical range. Taken together, findings suggest that it may be critical to support the education of adolescents grappling with attention problems (or ADHD symptoms), even if they may not meet the formal diagnostic criteria of ADHD (Zendarski et al., 2022).

Unexpectedly, in our study, externalizing behaviour was not associated with decreases in educational level, above and beyond attention problems (**Chapter 3**). Our findings may suggest that decreases in educational level in adolescence are not predicted by delinquent or aggressive behaviour, but by co-occurring attention problems. Alternatively, it is possible that associations between externalizing behaviour and educational outcomes are non-linear, such that only high levels of externalizing problems are independently associated with lower long-term educational outcomes. Likewise, whilst we did not find prospective associations between quantity-frequency scores for alcohol use and decreases in education (**Chapter 2**), it may be premature to conclude that alcohol use cannot adversely affect adolescents' long-term educational outcomes. It is still possible that mainly very heavy alcohol use is associated with decreases in education (Dalsgaard et al., 2020; Van Hoof et al., 2018; Vergunst et al., 2021). Such non-linear associations may have been missed in our analytic approach based on a relatively small adolescent cohort study, linear models, and continuous measures of alcohol use and mental health problems. Adolescents and young adults with severe mental health and substance use problems are less likely to participate in cohort studies, and if they do, may have higher risks of attrition (Fröjd et al., 2010; Zhao et al., 2009). Furthermore, severe forms of problems are relatively rare in the general population, which means that it may be easiest to investigate them either using samples recruited in clinical settings, or large population registers like those in the Scandinavian countries, which do not require active involvement of participants, and can draw on the whole population of adolescents and young adults in their catchment area. Indeed, in Scandinavian registers, clinical diagnoses of externalizing disorders (e.g., conduct disorder) were associated with adverse long-term educational outcomes, such as dropout from upper-secondary education, even after accounting for comorbidities (Mikkonen et al., 2018). Accordingly, Danish and Norwegian adolescents diagnosed with substance use disorders (SUDs) were less likely to finish their secondary education on time (Dalsgaard et al., 2020; Jensen et al., 2023). Currently little is known about the long-term educational outcomes of adolescents and young adults with severe psychiatric problems in the context of selective educational systems like in the Netherlands, which means that future research using larger and clinical samples in countries with such systems would be valuable.

Health-related selection in the educational system may not only be a consequence of limitations inherent to mental health problems, but could also depend on adolescents' social context at home and at school, which are key components of the microsystem in bioecological systems models (Bronfenbrenner & Morris, 2007). Parents, teachers, and

peers can respond to mental health problems by offering or withdrawing support. The amount of social support received may then determine how strongly a given mental health problem affects educational performance. In line with this idea, a recent study found variations in the association between ADHD symptoms and educational achievement across schools, suggesting that differences in the school context may indeed influence how strongly ADHD symptoms relate to educational outcomes (Cheesman et al., 2022). Surprisingly, in our sample three key features of adolescents' immediate social context (family functioning, social support by teachers, and social support by classmates) did not contribute substantially to associations between ADHD symptoms and lower education as mediators. Furthermore, associations did not differ between adolescents who experienced good family functioning and high levels of social support and those who did not (**Chapter 5**). This result could point at the robustness of the association between ADHD symptoms and lower educational attainment, but could also mean that our general measures of social support and family functioning may not have sufficiently tapped into aspects of social support most important for academic development (e.g., note sharing with classmates, help with homework by parents, accommodations by teachers). That specifically factors related to academic support could be critical is also highlighted by recent preliminary results suggesting that associations between ADHD symptoms and lower academic achievement may vary depending on the provision and quality of special educational support (Cheesman et al., 2022). However, it must be noted that research on the role of social factors in the context of ADHD and education is still in its infancy (Dvorsky & Langberg, 2016; Dvorsky et al., 2018). There is a profound lack of research on the extent to which key features of the school context, such as classroom size, affect the educational outcomes of adolescents with ADHD. Nevertheless, it seems reasonable to hypothesize that providing sufficient support to students with high levels of ADHD symptoms may be rather difficult within the large classrooms common particularly in the higher educational tracks of secondary school and in elementary education in the Netherlands (Dutch House of Representatives, 2017; Dutch Ministry of Education, Culture and Science, 2018).

The potential role of social factors as mediators in associations between mental health problems and long-term educational outcomes is also supported by a recent study suggesting that school performance mediates only about a third of the association between mental disorders and secondary school non-completion, and about half of the association between mental disorders and choosing a lower educational track (Mikkonen et al., 2021). Therefore, other factors, such as stigma and reduced educational expectations by adolescents' social environment may also be important (Chatzitheochari & Platt, 2019), and their role in associations between ADHD symptoms and education should be subject to future research. From a strictly sociological perspective, health-related selection can be viewed as a response of the opportunity structures and (educational) institutions that control access to the higher socioeconomic classes to individual characteristics that are

seen as problematic or less valued (West, 1991). If health-related selection within the educational system is indeed attributable to insufficient educational support (e.g., due to too large classrooms, lack of accommodations), stigma, or lower expectations towards adolescents with health issues, it may well be considered as form of disability discrimination (West, 1991).

2.1.2. Largely no indirect health-related selection related to childhood cognitive skills

Besides direct health-related selection (i.e., health problems or health-risk behaviours affecting educational level), it is also possible that health-related individual differences, including differences in cognitive skills in childhood (e.g., childhood IQ and effortful control), serve as shared predictors of educational level, as well as mental health and health behaviours. These cognitive skills themselves are influenced by both genetic dispositions (Deary et al., 2006; Yamagata et al., 2005) and differences in the early environment (Farah, 2017; Ng-Knight & Schoon, 2017; Sturge-Apple et al., 2017). This mechanism is also referred to as indirect health-related selection (Mackenbach, 2012).

We indeed found that genetic dispositions for smoking and lower educational attainment were associated with lower effortful control, and genetic dispositions for lower educational attainment also with having a lower IQ. Lower IQ and effortful control, in turn, strongly predicted being selected into a lower educational track in early adolescence (**Chapter 4**). Conversely, we observed that direct associations between cognitive skills and the health behaviours and mental health characteristics we assessed were largely absent, which is inconsistent with indirect health-related selection related to cognitive skills. Lower IQ and effortful control were not directly associated with smoking behaviour after controlling for PGSs and covariates (**Chapter 4**). Similarly, lower IQ was not associated with more attention problems and externalizing behaviour in early adolescence, and neither with increases in these symptoms throughout adolescence and young adulthood (**Chapter 3**). Only regarding alcohol use, we found a weak association of lower IQ with drinking in early adolescence, yet this association lost significance after also controlling for educational level (**Chapter 2**). Accordingly, associations of educational level with attention problems, externalizing behaviour, alcohol use, and smoking did not change much after controlling for cognitive skills. These associations would be expected to decrease substantially if strong indirect health-related selection effects related to cognitive skills were present.

Nevertheless, we found some evidence for indirect health-related selection related to genetic factors when considering educational inequalities in smoking. We found significant associations of genetic dispositions for smoking and lower educational attainment with both smoking behaviours and being in a lower educational trajectory, suggesting that genetic factors serve as shared predictors of both phenotypes. Accordingly, we observed very small reductions in associations between educational level and smoking in response

to adjusting for genetic risk factors. It is possible that genetic variants influence both smoking and educational attainment through separate phenotypic mechanisms, which is known as horizontal pleiotropy, or through shared phenotypic predictors associated with both smoking and education we have not measured, consistent with confounding pleiotropy (Davies et al., 2019).

Importantly, the lack of indirect health-related selection related to cognitive skills does not mean that IQ and effortful control play no critical role in educational health inequalities. Both cognitive skills strongly predicted adolescents' and young adults' educational trajectories, which were in turn associated with both smoking and alcohol use, consistent with social causation mechanisms (**Chapters 2 and 4**). These findings are in line with previous studies, which suggest that associations of cognitive ability with health and health behaviours are at least to a substantial part mediated by educational level or other socioeconomic factors, and hereby the social environment (Calvin et al., 2011; Link et al., 2008; Wraw et al., 2016; Wrulich et al., 2013).

2.1.3. Social causation for alcohol use and smoking, as well as externalizing behaviour

Lower educational level in early adolescence was associated with increases in alcohol use in mid adolescence, whilst in young adulthood there was a tendency towards opposite associations, with higher education predicting increases in alcohol use (**Chapter 2**). Our findings corroborate past research, which also found an earlier escalation in drinking amongst the lower educational tracks, and a reversal of the association between educational level and alcohol use in young adulthood (de Looze et al., 2013b; Jang et al., 2019; Latvala et al., 2014; Slutske, 2005). Conversely, lower education was consistently associated with smoking over the course of development, and these associations increased slightly as adolescents became young adults (**Chapter 4**). Associations between lower education and smoking did not change much after controlling for differences in cognitive skills and genetic predictors of both education and smoking, as mentioned above. Our findings are in line with some previous research also showing that the social gradient in smoking widens during the transition from adolescence to young adulthood (Alves et al., 2023; Pampel et al., 2017; Widome et al., 2013).

Educational differences in substance use could be explained by differences in social norms and peer group composition between educational tracks. In the Netherlands, classrooms in the lower (vocational) educational tracks are more strongly characterized by popularity norms endorsing smoking and alcohol use in early adolescence, and these norms in turn predicted adolescents' substance use within classrooms (Peeters et al., 2021). Accordingly, adolescents in the lower tracks more frequently perceived substance use as 'adult-like' behaviour, which may be used to gain popularity with peers (de Looze et al., 2013b). Changes in social norms, in combination with occupational and family demands,

may explain why in young adulthood the association between education and alcohol use reversed. In university culture, intensive alcohol use is strongly encouraged, potentially contributing to an escalation in drinking in young adults attending higher education (Fenton et al., 2023; Robertson & Tustin, 2018). Meanwhile, young adults who have followed the vocational tracks are frequently already in paid employment and often transition to adult family roles earlier, which may be difficult to combine with intensive alcohol use (Green et al., 2017; Staff et al., 2010). Nevertheless, even though from young adulthood onwards, lower educational groups often no longer consume more alcohol overall, they still tend to experience higher levels of alcohol-related harms over the life course (Boyd et al., 2022). Further research should explore the extent to which an earlier initiation of drinking could contribute to this so-called 'alcohol harm paradox', as early alcohol use has been found to be an important predictor of later problematic use, as well as alcohol use disorder (Grant & Dawson, 1997).

To address educational differences in adolescent and young adult substance use it is critical to understand the mechanisms underlying the divergence of substance use-related social norms across educational tracks. A promising avenue of future research could be to focus on the role of the psychological consequences of educational stratification in adolescent substance use. In Dutch culture, theoretical professions enjoy much higher prestige than practical or blue-collar work, resulting in societal norms that prefer academic above vocational education (van de Weerd, 2022). Therefore, the vocational tracks are often not chosen because of something an adolescent is good at (e.g., cooking, mechanics), but because the adolescent did not meet the requirements of the higher tracks (van de Weerd, 2022; Van Houtte, 2016). In addition, particularly discourses around the lower vocational track in the Netherlands are frequently characterized by a focus on perceived deficits, and its students are often seen as unintelligent, insubordinate, difficult, or lazy (van de Weerd, 2022). The loss of status, accompanied by prospects of a more insecure working life, may lead students in the lower educational tracks to believe that they have little control over their educational future and that putting effort into their education is futile (Spruyt et al., 2015; Van Houtte & Stevens, 2008). Feelings of futility have contributed to less school engagement amongst students in the lower educational tracks (Van Houtte & Stevens, 2010), as well as higher prevalences of school misconduct (Van Houtte & Stevens, 2008). Feelings of futility concerning education accordingly also strongly predicted intentions to leave school early amongst students in the lower educational tracks (Van Houtte & Demanet, 2016). The psychosocial consequences of educational stratification for students in the lower educational tracks have previously been suggested as explanations for emerging educational differences in adolescent substance use by Elstad (2010), but so far, to my knowledge, this hypothesis has not been empirically tested. This is surprising, given that sense of control and upward social comparison have been implicated as psychological mechanisms driving inequalities in health and health behaviours in adults (Hounkpatin

et al., 2016; Marmot, 2004; Peacock et al., 2014a, 2014b; Whitehead et al., 2016; Wolff et al., 2010).

We found that lower parental SES was a shared predictor of having a lower educational level, more externalizing behaviour, and more attention problems in early adolescence, and additionally contributed to an association between lower education in early adolescence and increases in externalizing behaviour in mid-adolescence (**Chapter 3**). Our results are in line with previous research from the Netherlands showing that lower parental SES strongly predicts teachers' recommendations to follow a lower educational track after primary school, even after controlling for children's performance in cognitive tests (van Spijker et al., 2017; Weinberg et al., 2019). Primary school teachers' recommendations for educational tracks can also be viewed as expression of the teachers' expectations regarding a student's ability to perform in secondary education (Timmermans et al., 2015). Past studies have found that teachers, perhaps unconsciously, tend to have lower educational expectations towards adolescents from lower SES families, possibly because they expect them to receive less educational support at home (Timmermans et al., 2015; Weinberg et al., 2019). At the same time, higher SES parents are often more effective at vouching for their children at school, raising teachers' educational expectations (Seghers et al., 2021; Weinberg et al., 2019).

Our findings are also consistent with previous results showing that socioeconomic disadvantage is associated with adolescents' mental health, including externalizing behaviour and ADHD symptoms (Piotrowska et al., 2015; Reiss, 2013; Russell et al., 2016). This may be explained by higher prevalences of risk factors related to mental health, such as early life stressors, neighbourhood disadvantage, stressful life events, and unhealthy family functioning in lower SES families (Barnhart et al., 2022; Markham & Spencer, 2022; Piotrowska et al., 2019; Sharp et al., 2021). In addition to more attention problems and externalizing behaviour, lower parental SES was associated with more alcohol use in early adolescence, but less alcohol use in young adulthood in our study (**Chapter 2**). A possible explanation for this finding is that young adults from higher SES families are less likely to live with their parents whilst attending higher education (van den Berg, 2020). Previous research has found that young adults living with parents whilst attending college tend to consume less alcohol than their peers living independently, possibly because of higher social control in the parental home (Benz et al., 2017). While research on the time-varying association between parental SES and adolescent substance use is very limited, a previous study also found a negative correlation between parental SES and underage drinking, and a positive correlation with drinking in young adulthood (Pedersen & von Soest, 2013).

2.1. Country-level social mobility only of importance for the association between family affluence and physical activity

We found that adolescents from low-affluent families living in countries characterized by higher social mobility in the previous generations were less likely to engage in physical activity than adolescents with similar socioeconomic backgrounds relative to their peers in countries that had relatively low levels of social mobility in the previous generations. Our measure of country-level social mobility was not associated with variations in socioeconomic inequalities in any of the other health behaviours assessed (i.e., having breakfast regularly, healthy and unhealthy foods consumed, smoking) (**Chapter 6**). It is unlikely that this result is explained by increased indirect health-related selection in socially mobile countries over the past generations and subsequent intergenerational transmission of cognitive skills and other individual differences related to health behaviours. Such individual differences should predict multiple health behaviours, rather than physical activity specifically. Future studies could focus on national-level characteristics associated with country-level social mobility in the past generations that may have an impact specifically on inequalities in physical activity. For example, country-level social mobility is strongly related to properties of the educational system, including the degree of educational tracking (Hanushek & Wößmann, 2006; Pekkarinen, 2018). Educational systems differ substantially in terms of curriculum time spent on physical education, and the extent to which participation in physical activity is required at school (Bann et al., 2019). This could have consequences for differences across countries in the engagement of low-SES adolescents in sports both as part of the formal curriculum, and during extracurricular activities.

It may also be worthwhile to study the moderating role of country-level social mobility in inequalities in health behaviours between young peoples' educational trajectories, as key components of their developing SES. If there is increased health-related selection in socially mobile countries in the current generation of adolescents, we would expect steeper inequalities by adolescents' educational level, as adolescents' educational trajectories should be more strongly predicted by individual differences potentially related to health behaviours, rather than their socioeconomic background. Unfortunately, we could not investigate this question, as most HBSC participants attended comprehensive education and therefore had not yet been subjected to educational tracking. Furthermore, we also do not yet have data on the extent of social mobility experienced by the current generation of adolescents. Opportunities for social mobility for the current generation of adolescents may differ from those experienced by previous generations, as social mobility appears to have been declining in several affluent countries (Connolly et al., 2021; Ludwinek et al., 2017). Future studies may also take into account differences in the extent of upward and downward social mobility, which may differ considerably between countries with similar overall social mobility (Apouey et al., 2022; Bukodi et al., 2017).

Lastly, future cross-national research could focus specifically on the experience of the most disadvantaged adolescents and young adults. Previous studies on country-level factors and adolescent health inequalities have mostly compared associations between SES and health across countries (e.g., Dierckens et al., 2022; Elgar et al., 2015; Weinberg et al., 2021). However, countries differ widely not only in socioeconomic health inequalities (and relatedly the variance in population health), but also in the average levels of population health around which inequalities are found (Kelly-Irving et al., 2023). Therefore, adolescents at the lower end of the socioeconomic spectrum in one country may face different health risks than adolescents with a similar socioeconomic position relative to their peers in another country, even if the extent of health inequalities is identical in both countries. The limited research available focussing specifically on the most deprived populations suggests that these groups are often the most affected by (changes in) country-level factors, including social policies and macroeconomic conditions. For example, Scotland has seen sharp increases in premature mortality in the most deprived socioeconomic groups over the past decade, while improvements in mortality in the other groups have largely stalled (Walsh & McCartney, 2023). This finding may be explained by several changes in macro-level conditions that could have disproportionately affected the health of the most deprived populations, including extensive reductions in public spending, and most recently the COVID-19 pandemic and very high levels of inflation (Walsh & McCartney, 2023). How differences across countries in (changes in) macro-level conditions are related to the health of the most disadvantaged adolescents is currently largely unknown and should be subject to future investigation.

3. STRENGTH AND LIMITATIONS

A key strength of this dissertation lies in the high quality of the datasets on which it is based. **Chapters 2-5** are based on data from the TRAILS study, which is characterized by a high response rate, a long follow-up (16 years), and the consistency of measures over time, allowing to investigate multiple developmental periods simultaneously. TRAILS provides rich data, capturing both characteristics of the individual (e.g., genetic data, cognitive test results, standardized questionnaires on health and behaviours), as well as the social context (e.g., social support at school, family functioning, parental SES, educational level) over the course of development. TRAILS is one of the few studies featuring a consistent and age-appropriate measure of educational level, as key component of developing SES, over the course of adolescence and young adulthood. **Chapter 6** benefits from the large sample size of HBSC, as well as its cross-national approach, reflecting a wide range of socio-cultural, economic, and political contexts, whilst including identical measures of health behaviours and family affluence across as many as 32 different countries (Inchley et al., 2018).

A further strength of this dissertation lies in the innovative statistical techniques used. For example, cross-lagged panel models with fixed effects allowed to disentangle the temporal directions in associations between alcohol use, mental health, and educational level, whilst also addressing unmeasured time-stable confounding (Allison et al., 2017). Mediation analyses based on 'interventional effects' permitted to estimate potential indirect effects of ADHD symptoms on educational level via social support at school and family functioning, whilst simultaneously allowing for interactions between ADHD symptoms and these hypothesized mediators (Vansteelandt & Daniel, 2017). Lastly, a particular strength of this dissertation is its interdisciplinary approach, drawing on a wide range of disciplines, including social epidemiology, public health, psychology, sociology, and behavioural genetics. This approach may indeed be best suited to study the complex web of influences that underlies the development of health inequalities (Lundberg, 2020).

However, this dissertation also has several limitations, which should be acknowledged. First, although we implemented multiple imputations or full information maximum likelihood to manage missing data, attrition and non-response may still have influenced our conclusions. Adolescents with more (mental) health problems and health-risk behaviours, those with a lower educational level, and those growing up in lower SES families tend to be underrepresented in adolescent cohorts, as they are more difficult to recruit and to retain in studies (Cheung et al., 2017; Fakkel et al., 2020; Fröjd et al., 2010; Nederhof et al., 2012). Additionally, those adolescents from low SES backgrounds and/or with low educational level who participated in TRAILS/HBSC may differ to some extent from adolescents with the same backgrounds who did not (e.g., in terms of having better health, being more resilient, coming from better functioning families, etc.). If that is the case, our results could overestimate the health status of adolescents with a relatively low educational level and/or family SES, and hereby underestimate the extent of health inequalities in the population. Reassuringly, both HBSC and TRAILS have been comparably good at including participants from hard-to-reach backgrounds. HBSC relies on school-based surveys, which tend to have higher response rates than studies where adolescents are approached outside the educational context, particularly amongst underrepresented groups (Dey et al., 2021). TRAILS has been substantially better at including participants from lower SES backgrounds than other Dutch adolescent cohorts (Fakkel et al., 2020), which is thanks to extensive efforts to recruit hard-to-reach adolescents (e.g., home visits and call-backs) (de Winter et al., 2005; Nederhof et al., 2012). Nevertheless, in particular adolescents scoring low on multiple SES indicators (e.g., both parents low educated and low income) were poorly represented even in TRAILS (Fakkel et al., 2020). Although our approach might not have been ideal in studying adolescents at the highest levels of socioeconomic risk, our results can still give important insights on much of the social gradient in adolescent health, which typically covers the entire socioeconomic spectrum. This means that health inequalities often exist even between the highest socioeconomic groups (Marmot et al., 1997).

Second, this dissertation mainly took a non-clinical approach, focussing on common manifestations of health behaviours and mental health problems. We did not have sufficient sample sizes in the TRAILS population cohort to study severe forms of health problems, which are relatively rare in the general population of adolescents, yet may be most strongly associated with adverse educational and socioeconomic outcomes (Mikkonen, 2021). Such conditions can be studied with population registers, which are common in the Scandinavian countries, but to the best of my knowledge, are currently unavailable in any country with selective educational systems. More research on high-risk groups, in terms of both socioeconomic deprivation and health, is crucial. Past research has shown that particularly adolescents with low scores on multiple SES indicators are at high risk of impairments in development (Fakkel et al., 2020), and that adolescents with severe mental health problems, such as diagnosed SUD, conduct disorder, or psychosis, as well as those with a history of psychiatric hospitalization, are at especially high risk of adverse educational outcomes (Dalsgaard et al., 2020; Holttinen et al., 2022; Mikkonen et al., 2018). Accordingly, past research has found that a relatively small proportion of children, who often come from backgrounds characterized by economic deprivation and maltreatment and tend to have relatively low cognitive skills, carry a disproportionate burden of adverse health and socioeconomic outcomes in adulthood (Caspi et al., 2016). Lastly, this dissertation did not comprehensively address differences by gender, ethnicity, and sexual identity. Larger samples than TRAILS are needed to have sufficient power to identify potential variations in associations across subgroups of adolescents and young adults. Future research on these questions is critical, as adolescents experience membership in multiple groups simultaneously (e.g., being in a lower educational track and having non-Dutch ethnicity). Different combinations of group memberships may interact and, in this way, strengthen or weaken each other's association with health outcomes (Duinhof, 2020).

4. GENERAL CONCLUSION

The beginning of the third decade of the 21st century has been characterized by a syndemic of multiple crises, including the COVID-19 pandemic, accelerating global warming, a new war in Europe, and rampant inflation in many countries, including the Netherlands. These crises have all been or likely will be associated with further increases in health inequalities (Castro et al., 2022; Bambra et al., 2020; Limb, 2022), and are known to disproportionately affect the health of adolescents and young adults (Kadir et al., 2019; McGushin et al., 2022; Panchal et al., 2021; Scharpf et al., 2021; Skinner et al., 2023; UK Youth, 2023). Furthermore, substantial changes have taken place in the labour market, including increasing workplace automation and offshoring, and associated declines in lower skilled jobs and wages, as well as a proliferation of precarious employment (Gray et al., 2021; O'Brien et al., 2022). Studies

suggest that these changes may have contributed to a widening of health inequalities (Gray et al., 2021; LaMontagne, 2010; O'Brien et al., 2022). Simultaneously, many affluent countries have decreased spending on social protection systems over the past decades, and there is accumulating evidence that this has adversely affected the health of disadvantaged populations (Labonté & Stuckler, 2016; Stuckler et al., 2017; Walsh & McCartney, 2023; Wickham et al., 2020). Research and action on health inequalities have therefore perhaps never been more urgent than now.

I hope that this dissertation can make a small contribution towards a better understanding of the mechanisms underpinning the development of socioeconomic health inequalities in adolescence and young adulthood. Our findings support the idea that a complex interplay of individual differences and the social context underlies the formation of socioeconomic health inequalities. Our results highlighted the robust detrimental effects of ADHD symptoms on educational attainment (i.e., direct health-related selection). Regarding educational differences in adolescents' and young adults' smoking and alcohol use, social causation mechanisms seemed to predominate. Inconsistent results were found regarding the question whether country-level social mobility moderates associations between family affluence and adolescent health behaviours, highlighting the complexity of the relationship between macro-level contextual factors and health inequalities. In future research, complex systems approaches may be employed to provide further insight into the dynamic interplay of characteristics of the individual, the home and school context, and the wider sociocultural, policy, and economic context in shaping inequalities over the course of development (i.e., the micro- and macrosystem) (Diez Roux, 2011; Holdsworth et al., 2017). These approaches could also address the impact of ongoing rapid changes in macro-level conditions (i.e., the chronosystem), including shocks and crises, on health inequalities, and in particular the experience of the most disadvantaged adolescents and young adults.

TABLE 7.1 Summary per chapter

	Chapter 2	Chapter 3	Chapter 4
Gaps of knowledge	There is a lack of research on social causation and health-related selection mechanisms in educational inequalities in alcohol use in the selective educational systems common in Western Europe.	There is a lack of research on social causation and health-related selection mechanisms in educational inequalities in mental health in the selective educational systems common in Western Europe.	Recent studies suggest that smoking and lower educational attainment may have genetic influences in common. However, little is known about the developmental mechanisms through which genetic influences contribute to educational inequalities in adolescent and young adult smoking.
Aims	1. Evaluate associations between educational level and changes in subsequent alcohol use between around age 14 and 26 (i.e., social causation), whilst controlling for reverse-causation, as well as measured and unmeasured time-stable characteristics 2. Evaluate associations between alcohol use and changes in subsequent educational level between around age 14 and 26 (i.e., direct health-related selection) whilst controlling for reverse-causation, as well as measured and unmeasured time-stable characteristics 3. Evaluate the role of characteristics in childhood (i.e., IQ, effortful control, parental SES) in early adolescent educational level and alcohol use, as well as their contribution to later bidirectional associations between educational level and alcohol use	1. Evaluate associations of educational level with changes in subsequent attention problems and externalizing behaviour between around age 14 and 26 (i.e., social causation), whilst controlling for reverse-causation, as well as measured and unmeasured time-stable characteristics 2. Evaluate associations of attention problems and externalizing behaviour with changes in subsequent educational level between around age 14 and 26 (i.e., direct health-related selection) whilst controlling for reverse-causation, as well as measured and unmeasured time-stable characteristics 3. Evaluate the role of characteristics in childhood (i.e., IQ, effortful control, parental SES) in early adolescent educational level, attention problems, and externalizing behaviour, as well as their contribution to later bidirectional associations between educational level and externalizing behaviour and attention problems	1. Evaluate whether cross-phenotype associations exist between a PGS for smoking (PGS_{SMOK}) and educational attainment, and a PGS for educational attainment (PGS_{EDU}) and smoking 2. Evaluate the extent to which both PGSs predict IQ and effortful control measured in childhood (11 years), and whether these cognitive skills in turn act as shared predictors of both educational level and smoking behaviour in adolescence and young adulthood (from around age 16 to 26 years) (i.e., indirect health-related selection) 3. Examine the extent to which adolescents' educational level, and hereby educational differences in the social context (i.e., social causation), mediate associations between PGSs and smoking (consistent with social causation explanations)
Data	TRAILS study general population cohort (N = 2,229), waves 1 – 6 (measurements around age 11, 14, 16, 19, 22, and 26)	TRAILS study general population cohort (N = 2,229), waves 1 – 6 (measurements around age 11, 14, 16, 19, 22, and 26)	TRAILS study general population cohort and clinical cohort, only participants with genetic information (N = 1,581), waves 1, and 3 – 6 (measurements around age 11, 16, 19, 22, and 26)

	Chapter 5	Chapter 6
	ADHD symptoms are robustly associated with lower educational attainment across studies. Yet, little is known about the role of social factors in the home and school context, which may mediate or moderate this association.	So far, no studies have investigated the role of contextual-level social mobility in socioeconomic inequalities in adolescent health behaviours.
	1. Evaluate whether family functioning, as well as social support by teachers and classmates mediate the association of ADHD symptoms with educational level in early adolescence, as well as with subsequent declines in educational level over the course of adolescence 2. Evaluate whether family functioning, as well as social support by teachers and classmates interact with ADHD symptoms, such that the association between ADHD symptoms and lower education is weaker in the presence of good family functioning and high levels of social support by teachers and classmates	1. Investigate whether associations between family affluence and adolescent health behaviours differ between countries with high and low levels of social mobility, and, if so, determine the direction of these potential interactions
	TRAILS study general population cohort (N = 2,229), waves 1 – 4 (measurements around age 11, 14, 16, and 19)	2017/2018 HBSC study, data from 32 countries (N = 185,086) European Social Survey (ESS)

TABLE 7.1 Continued

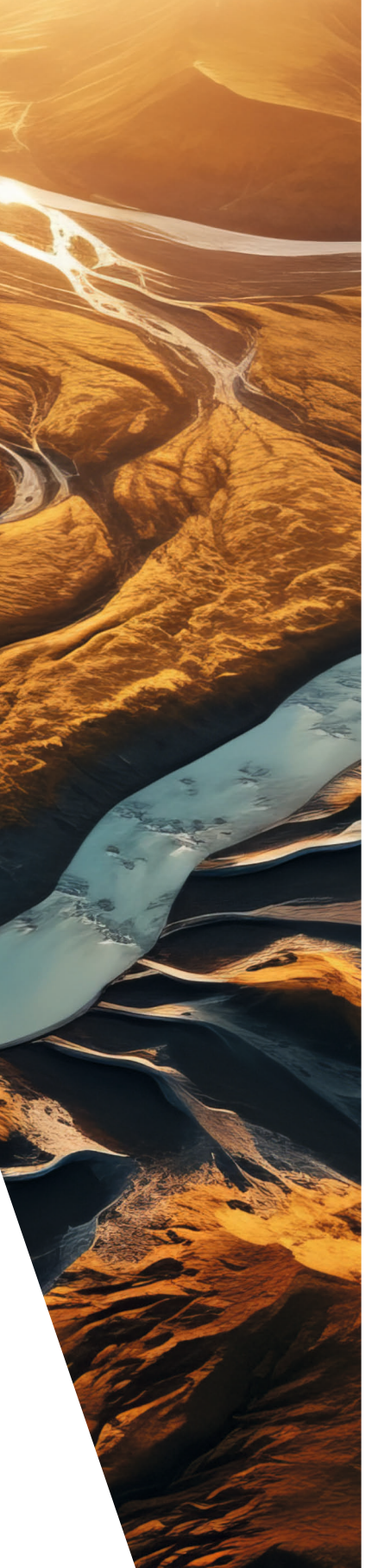
	Chapter 2	Chapter 3	Chapter 4
Measures	- alcohol use quantity-frequency measure (self-report; waves 2 – 5) - AUDIT-C (self-report; wave 6) - educational level (self-report; waves 2 – 6) - IQ (wave 1; Block Design and Vocabulary subtests of the WISC-R) - effortful control (parental report; wave 1; EATQ-R) - parental SES (parental report; wave 1)	- externalizing behaviour and attention problems scales from the ASEBA YSR (waves 2 – 3), and ASR (waves 4 – 6) (self-report) - educational level (self-report; waves 2 – 6) - IQ (wave 1; Block Design and Vocabulary subtests of the WISC-R) - parental SES (parental report; wave 1)	- past month smoking (self-report) - educational level (self-report; waves 2 – 6) - polygenic scores (PGSs) for ever smoking regularly and years of education - IQ (wave 1; Block Design and Vocabulary subtests of the WISC-R) - effortful control (parental report; wave 1; EATQ-R) - parental educational level (parental report; wave 1) - parental smoking (parental report; wave 1)
Main findings	1. Lower education around age 14 predicted increases in drinking around 16. From age 19 onward, we found a tendency towards opposite associations, with higher education predicting increases in alcohol use. 2. Alcohol use was not associated with subsequent changes in education. 3. Childhood characteristics (i.e., IQ, effortful control, parental SES) strongly predicted education around age 14 and to a lesser extent early drinking.	1. Attention problems predicted decreases in education throughout all of adolescence and young adulthood. 2. Differences in parental SES contributed to increases in externalizing behaviour amongst the lower educational tracks in mid-adolescence. 3. Higher childhood IQ and parental SES strongly predicted a higher educational level around age 14. Higher parental SES also predicted less early adolescent attention problems and externalizing behaviour.	1. Genetic vulnerability for smoking (PGS_{SMOK}) was associated with having a lower educational level throughout adolescence and young adulthood. Similarly, a PGS for having a lower educational attainment (PGS_{EDU}) was associated with smoking. 2. Whereas PGS_{SMOK} and PGS_{EDU} were both significantly associated with lower effortful control, and PGS_{EDU} also with lower IQ, these cognitive skills were largely not direct predictors of smoking and lower education (i.e., no indirect health-related selection related to cognitive skills). 3. Partly through their associations with lower cognitive skills, PGS_{EDU} and PGS_{SMOK} predicted selection into a lower educational track, which in turn predicted increased smoking behaviour.

	Chapter 5	Chapter 6
	- DSM-oriented ADHD symptom scales from the ASEBA YRS and CBCL (waves 1 – 3; both parental and self-report) - educational level (self-report; waves 2 – 4) - family functioning (parental report; waves 1 – 3; McMaster Family Assessment Device) - social support by teachers and classmates (self-report; waves 1 – 3; affection and behavioural confirmation subscales adapted from the Social Production Functions Questionnaire) - IQ (wave 1; Block Design and Vocabulary subtests of the WISC-R) - parental SES (parental report; wave 1)	- moderate-to-vigorous and vigorous physical activity, healthy foods consumed (fruit/vegetables), unhealthy foods consumed (sweets/soft drinks), having breakfast regularly, and smoking (all self-report) - family affluence (self- report; FAS-III) - country-level social mobility, computed based on educational data in the ESS using UNIDIFF models
	1. Family functioning and social support by teachers and classmates did not mediate associations between ADHD symptoms and (decreases in) educational level. 2. Associations between ADHD symptoms and lower education did not differ depending on the level of family functioning, or social support by teachers and classmates.	1. Socioeconomic inequalities in both moderate-to-vigorous and vigorous physical activity are larger in countries with higher levels of social mobility. 2. No cross-level interactions were found regarding any of the other health behaviours studied (i.e., healthy and unhealthy foods consumed, having breakfast regularly, and smoking.

TABLE 7.1 Continued

	Chapter 2	Chapter 3	Chapter 4
Conclusions	We mainly found evidence for social causation in early adolescence, when lower education predicted increases in subsequent alcohol use. We found no evidence in support of the direct health-related selection hypothesis with respect to alcohol use. By determining initial educational level, childhood characteristics also predict subsequent trajectories in alcohol use. Our findings emphasize the need for interventions to delay the early escalation of alcohol use amongst adolescents in the lower educational tracks, as early drinking is an important predictor of later problematic use and alcohol use disorders.	In line with the direct health-related selection hypothesis, our findings suggest that attention problems pose a risk for decreases in educational level during all phases of adolescence and young adulthood. In line with social causation, lower parental SES strongly predicted selection into a lower educational trajectory and to a lesser extent more attention problems and externalizing behaviour in early adolescence. Our results emphasize the need for interventions to address the negative impact of attention problems on educational attainment. Further, our results highlight the role of social causation from parental SES in determining adolescent educational level, attention problems, and externalizing behaviour.	Our results suggest that educational differences in the social context contribute to associations between genetic liabilities and educational inequalities in smoking, consistent with social causation explanations. Our findings shed further light on how social conditions, such as educational differences in the classroom context, add to the genetic relationship between smoking and lower educational attainment. The social contexts in the lower educational tracks (e.g., social norms, peer group composition, social stressors) may therefore be an important target for interventions.

	Chapter 5	Chapter 6
	Results indicate that the association between ADHD symptoms and lower education is insensitive to key aspects of adolescents' social context.	Contextual-level social mobility may contribute to international differences in the extent of socioeconomic inequalities in adolescent physical activity.
	Conclusions might differ if measures more proximally related to academic functioning are used (e.g., parental involvement in school, note sharing with classmates, accommodations at school). This could be tested in future studies.	Future studies on socio-cultural and policy factors related to inequalities in adolescent physical activity, as well as country-level social mobility, are warranted.



APPENDICES

A

SAMENVATTING
(summary in Dutch)

SUPPLEMENTARY MATERIALS

REFERENCES

ACKNOWLEDGEMENTS

CURRICULUM VITAE

SAMENVATTING (SUMMARY IN DUTCH)

1. INLEIDING

De mechanismes die ten grondslag liggen aan de ontwikkeling van sociaaleconomische ongelijkheden in de mentale gezondheid en het gezondheidsgedrag van jongeren en jongvolwassenen zijn niet goed onderzocht. Het is belangrijk om deze mechanismes beter te begrijpen om beleid en interventies te ontwikkelen om deze gezondheidsverschillen al zo vroeg mogelijk aan te pakken. Het hoofddoel van dit proefschrift is om meer inzicht te krijgen in het samenspel van twee processen die mogelijk bijdragen aan het ontstaan van sociaaleconomische gezondheidsverschillen: sociale causatie en gezondheidsgerelateerde selectie (Mackenbach, 2012). Sociale causatie benadrukt de rol van de sociaaleconomische achtergrond van de familie en van verschillen in de sociale omgeving die samenhangen met onderwijsniveaus in het ontstaan van gezondheidsverschillen (Mackenbach, 2012). Gezondheidsgerelateerde selectie kan verwijzen naar twee soorten processen: directe en indirecte gezondheidsgerelateerde selectie (Mackenbach, 2012). Directe gezondheidsgerelateerde selectie verwijst naar processen waarbij gezondheidsproblemen (waaronder psychische klachten zoals aandachtsproblemen en externaliserend gedrag) en bepaalde soorten gezondheidsgedrag (bijvoorbeeld intensief alcoholgebruik) de schoolcarrière van jongeren en jongvolwassenen beïnvloeden (Jensen et al., 2023; Mackenbach, 2012; Van Hoof et al., 2018; Veldman et al., 2014). Indirecte gezondheidsgerelateerde selectie verwijst naar de rol van individuele kenmerken die al in de kindertijd aanwezig zijn. Deze individuele kenmerken voorspellen het latere opleidingsniveau alsook gezondheidskenmerken of -gedrag, en dragen hierdoor als 'derde variabelen' bij aan de verbanden tussen opleidingsniveau, gezondheidskenmerken en gezondheidsgedrag (Mackenbach, 2012). Deze variabelen kunnen genetische factoren en genetisch beïnvloede kenmerken van het individu omvatten die het latere opleidingsniveau voorspellen, zoals zelfcontrole en IQ in de kindertijd (Brody, 1997; Deary et al., 2006; Veronneau et al., 2014; Yamagata et al., 2005), evenals de mentale gezondheid en het gezondheidsgedrag in de adolescentie en jongvolwassenheid (Daly & Egan, 2017; Daly et al., 2016; Deary et al., 2021; Kubička et al., 2001; Moffitt, 1993; Peeters et al., 2017; Wedow et al., 2018).

In selectieve onderwijssystemen zoals in Nederland is de rol van sociale causatie en gezondheidsgerelateerde selectie in de ontwikkeling van verschillen in mentale problemen en middelengebruik tussen onderwijsniveaus beperkt bestudeerd. Daarom onderzochten we bidirectionele verbanden (d.w.z. directe gezondheidsgerelateerde selectie en sociale causatie) tussen alcoholgebruik en opleidingsniveau (**Hoofdstuk**

2), evenals tussen externaliserend gedrag/aandachtsproblemen en opleidingsniveau (**Hoofdstuk 3**) bij Nederlandse adolescenten en jongvolwassenen van ongeveer 11 tot ongeveer 26 jaar oud. Daarbij hielden we rekening met sociale causatie gerelateerd aan de ouderlijke sociaaleconomische status en met indirecte gezondheidsgerelateerde selectie gerelateerd aan IQ en zelfcontrole in de kindertijd. Van alle mentale problemen laten aandachtsproblemen (en daaraan verwante gedragsvertoningen, zoals ADHD-symptomen) en externaliserend gedrag de sterkste verbanden zien met een lager opleidingsniveau bij adolescenten (Evensen et al., 2016; Meißner et al., 2022; Van Houtte & Stevens, 2008; Veldman et al., 2014). In **Hoofdstuk 4** onderzochten we mechanismes die genetische factoren koppelen aan verschillen in roken tussen onderwijsniveaus in de adolescentie en jongvolwassenheid. We evalueerden met name of genetische varianten die roken of een lager onderwijsniveau voorspellen samenhangen met een lager IQ en een lagere zelfcontrole in de kindertijd, en of deze kenmerken vervolgens zowel later roken als opleidingsniveau voorspellen (d.w.z. indirecte gezondheidsgerelateerde selectie met betrekking tot cognitieve kenmerken). Daarnaast hebben we onderzocht of genetische factoren indirect samenhangen met opleidingsniveau-gerelateerde ongelijkheden in roken door de onderwijstrajecten van jongeren te voorspellen, en hierdoor verschillen in de sociale context (in lijn met sociale causatie). Vervolgens onderzochten wij in **Hoofdstuk 5** de rol van drie cruciale factoren in de sociale omgeving van adolescenten – namelijk het gezinsfunctioneren en de hoeveelheid aan sociale steun van klasgenoten en van leraren – in het verband tussen ADHD-symptomen en een lager onderwijsniveau. Mogelijk leiden ADHD-symptomen tot een slechter gezinsfunctioneren en minder sociale steun op school en vervolgens tot een lager opleidingsniveau (d.w.z. mediatie). Bovendien zouden de negatieve effecten van ADHD-symptomen op het onderwijsniveau sterker kunnen zijn bij adolescenten met een slechter functionerend gezin of minder sociale steun (d.w.z. interactie).

De bredere sociale en culturele context in een land kan van invloed zijn op de mechanismes die bijdragen aan de ontwikkeling van sociaaleconomische gezondheidsverschillen. Een kenmerk op landelijk niveau waarvan gedacht wordt dat het een rol speelt bij het voortbestaan van sociaaleconomische gezondheidsverschillen, zelfs in landen met uitgebreide sociale voorzieningen, is sociale mobiliteit (Mackenbach, 2012). In landen met veel sociale mobiliteit is de eigen sociaaleconomische status niet sterk afhankelijk van de ouderlijke sociaaleconomische status. De eigen status hangt daarom mogelijk sterker af van, onder andere, cognitieve kenmerken zoals IQ en zelfcontrole die ook samenhangen met gezondheidsgedrag en een groot aantal andere gezondheidskenmerken (overeenkomend met indirecte gezondheidsgerelateerde selectie), zoals eerder vermeld. In **Hoofdstuk 6** hebben we daarom onderzocht of sociale mobiliteit op landelijk niveau gedurende de vorige generaties samenhangt met de sterkte van verbanden tussen de ouderlijke sociaaleconomische status en het gezondheidsgedrag van jongeren.

2. METHODOLOGISCHE BENADERING

Dit proefschrift maakt gebruik van de TRacking Adolescents' Individual Lives Survey (TRAILS), een studie die bestaat uit een populatiecohort (N = 2,229) en een klinisch cohort (N = 543) van adolescenten die werden gevolgd vanaf hun 11e levensjaar. Wij hebben metingen tussen 11 en 26 jaar gebruikt om mechanismes te onderzoeken die mogelijk bijdragen aan de ontwikkeling van gezondheidsverschillen tussen onderwijsniveaus in Nederland. Het bevolkingscohort werd geworven op 135 scholen in de provincies Groningen, Friesland en Drenthe, waarvan er 122 besloten om deel te nemen aan TRAILS (de Winter et al., 2005). Het klinische cohort bestaat uit adolescenten die op enig moment in hun leven werden verwezen naar de Universitaire Kinder- en Jeugdpsychiatrie in Groningen voor een consult of behandeling (20.8% ≤5 jaar, 66.1% 6 – 9 jaar, 13.1% 10 – 12 jaar). Een gedetailleerde beschrijving van TRAILS is elders te vinden (Oldehinkel et al., 2015). **Hoofdstukken 2 en 3** gebruiken de metingen 1 tot en met 6 (leeftijd circa 11 – 26 jaar), en **Hoofdstuk 5** de metingen 1 tot en met 4 (leeftijd circa 11 – 19 jaar) van het bevolkingscohort. **Hoofdstuk 4** combineert gegevens van het bevolkingscohort met die van het klinische cohort (meting 1 tot en met 6; leeftijd circa 11 – 26 jaar). **Hoofdstuk 6** gebruikt gegevens uit 32 landen die deelnamen aan de 2017/2018-editie van de Health Behavior in School-aged Children (HBSC) study (N = 185,086), een grootschalige internationale studie over het gezondheidsgedrag van adolescenten van 10 – 16 jaar (gemiddelde leeftijd 13.50 jaar) in samenwerking met de Wereldgezondheidsorganisatie (WHO), die om de vier jaar wordt uitgevoerd binnen een netwerk van landen in Europa en Noord-Amerika. In alle landen werd hetzelfde gestandaardiseerde protocol gebruikt, wat internationale vergelijkingen mogelijk maakt (Inchley et al., 2018). **Hoofdstuk 6** bevat bovendien gegevens op landniveau over sociale mobiliteit, die zijn berekend op basis van de European Social Survey (ESS) (ESS Data Team, 2021), evenals gegevens over het bruto nationaal inkomen (BNI) van de Wereldbank DataBank (World Bank, 2021), en inkomensongelijkheid, zoals bepaald door de GINI-index, uit de 9e versie van de Standardized World Income Inequality Database (Solt, 2019).

3. SAMENVATTING VAN DE RESULTATEN

3.1. Het samenspel van sociale causatie en gezondheidsgerelateerde selectie bij de ontwikkeling van opleidingsniveau-gerelateerde verschillen op het gebied van middelengebruik en mentale gezondheid

De rol van sociale causatie en zowel directe als indirecte gezondheidsgerelateerde selectie verschilde tussen ontwikkelingsperiodes, alsook tussen soorten gezondheidsgedrag of mentale problemen.

3.1.1. Directe gezondheidsgerelateerde selectie met betrekking tot aandachts-problemen/ADHD-symptomen

Directe gezondheidsgerelateerde selectie werd voornamelijk gevonden in samenhang met aandachtsproblemen (**Hoofdstuk 3**) en ADHD-symptomen (**Hoofdstuk 5**), die dalingen in het opleidingsniveau in alle fases van de adolescentie en jongvolwassenheid voorspelden. Bovendien ontdekten we dat verbanden tussen ADHD-symptomen en een lager onderwijsniveau niet werden gemedieerd door belangrijke kenmerken van de sociale context van adolescenten (d.w.z. gezinsfunctioneren, sociale steun van leraren en sociale steun van klasgenoten). Tevens vonden we geen verschillen in deze verbanden tussen adolescenten die in een goed functionerend gezin opgroeiden en veel sociale steun rapporteerden en diegenen die dat niet deden (**Hoofdstuk 5**). Ook externaliserend gedrag hing samen met dalingen in het opleidingsniveau in de vroege adolescentie en de jongvolwassenheid, maar deze verbanden verdwenen na correctie voor aandachtsproblemen (**Hoofdstuk 3**).

3.1.2. Weinig bewijs voor indirecte gezondheidsgerelateerde selectie met betrekking tot cognitieve kenmerken in de kindertijd

We vonden niet veel bewijs voor indirecte gezondheidsgerelateerde selectie met betrekking tot IQ en zelfcontrole in de kindertijd. Zoals verwacht waren zowel een hoger IQ als meer zelfcontrole sterke voorspellers van selectie in een hoger onderwijstraject. Echter vonden we in de meeste analyses geen directe verbanden tussen IQ en externaliserend gedrag (**Hoofdstuk 3**), aandachtsproblemen (**Hoofdstuk 3**), alcoholgebruik (**Hoofdstuk 2**) en roken (**Hoofdstuk 4**), en evenzo geen directe verbanden tussen zelfcontrole en drinken (**Hoofdstuk 2**) en tabaksgebruik (**Hoofdstuk 4**). In plaats daarvan ontdekten we dat zowel IQ als zelfcontrole indirect samenhingen met middelengebruik doordat deze variabelen de onderwijstrajecten van adolescenten en jongvolwassenen voorspelden, die op hun beurt samenhingen met de mate van alcoholgebruik (**Hoofdstuk 2**) en roken (**Hoofdstuk 4**). Met analyses gebaseerd op polygenetische scores vonden we enig bewijs van indirecte genetische gezondheidsgerelateerde selectie in de ontwikkeling van opleidingsgerelateerde verschillen in tabaksgebruik (**Hoofdstuk 4**). Genetische voorspellers van roken en een lager opleidingsniveau hingen significant samen met rookgedrag, alsook met een lager opleidingsniveau, en deze verbanden werden niet gemedieerd door IQ of zelfcontrole. We zagen echter slechts een kleine daling in het verband tussen opleidingsniveau en roken na correctie voor polygenetische scores, wat erop kan wijzen dat de impact van indirecte gezondheidsgerelateerde selectie door genetische factoren gering is. Aan de andere kant meten polygenetische scores momenteel slechts een deel van de genetische variantie die samenhangt met roken en opleidingsniveau (Pingault et al., 2021), hetgeen betekent dat correctie voor polygenetische scores de mate van indirecte genetische gezondheidsgerelateerde selectie vermoedelijk onderschat.

3.1.3. Sociale causatie met betrekking tot alcoholgebruik en roken, evenals externaliserend gedrag

Sociale causatie bleek een rol te spelen bij de ontwikkeling van verschillen in roken en drinken tussen opleidingsniveaus. Het drankgebruik van jonge adolescenten in de lagere onderwijstrajecten was drie jaar later sterker gestegen dan dat van hun leeftijdsgenoten in de hogere onderwijstrajecten (**Hoofdstuk 2**). In de jongvolwassenheid was de invloed van het onderwijsniveau in de tegenovergestelde richting: in die fase nam het drankgebruik van jongvolwassenen in het hoger onderwijs sterker toe dan dat van leeftijdsgenoten in lagere trajecten. De verbanden tussen opleidingsniveau en roken verzwakten niet veel na correctie voor polygenetische scores, cognitieve kenmerken en andere belangrijke covariaten, hetgeen suggereert dat opleidingsgerelateerde verschillen in tabaksgebruik vooral het gevolg zijn van verschillen in de sociale context (**Hoofdstuk 4**).

Eveneens in lijn met sociale causatie, voorspelde een hogere ouderlijke sociaaleconomische status selectie in een hoger onderwijstraject, minder alcoholgebruik in de vroege adolescentie en een sterkere toename van het drankgebruik in de jongvolwassenheid (**Hoofdstuk 2**). De ouderlijke sociaaleconomische status bleek een belangrijkere voorspeller van de mentale gezondheid van jongeren te zijn dan hun opleidingsniveau. Na correctie voor mogelijke confounders hing het opleidingsniveau van jongeren niet meer samen met veranderingen in aandachtsproblemen of externaliserend gedrag in de adolescentie en jongvolwassenheid. In plaats daarvan vonden we dat een lagere ouderlijke sociaaleconomische status meer aandachtsproblemen en externaliserend gedrag in de vroege adolescentie voorspelde. Bovendien droeg een lage ouderlijke sociaaleconomische status bij aan een bivariaat verband tussen een lager opleidingsniveau in de vroege adolescentie en een toename van externaliserend gedrag in de daaropvolgende jaren (**Hoofdstuk 3**).

3.2. Landelijke sociale mobiliteit alleen van belang voor het verband tussen gezinswelvaart en lichamelijke activiteit

Tenslotte ontdekten we dat adolescenten uit gezinnen met lage welvaart in landen met een hoge sociale mobiliteit gedurende de vorige generaties over het algemeen minder lichamelijk actief waren dan leeftijdsgenoten met vergelijkbare sociaaleconomische achtergronden in landen met een lagere sociale mobiliteit gedurende de vorige generaties (**Hoofdstuk 6**). Landelijke sociale mobiliteit gedurende de vorige generaties hing niet samen met sociaaleconomische verschillen in andere soorten gezondheidsgedrag (d.w.z. consumptie van gezond en ongezond voedsel, regelmatig ontbijten en roken). Samengevat benadrukken onze bevindingen de complexiteit van de relatie tussen sociale mobiliteit op landelijk niveau en gezondheidsverschillen in jongeren.

4. DISCUSSIE VAN DE BELANGRIJKSTE RESULTATEN EN HUN THEORETISCHE EN PRAKTISCHE IMPLICATIES

Hieronder bespreek ik de theoretische en praktische implicaties van de belangrijkste bevindingen van dit proefschrift over de bestudeerde mechanismes (d.w.z. directe en indirecte gezondheidsgerelateerde selectie en sociale causatie), evenals over de rol van de landelijke sociale mobiliteit, in het ontstaan van sociaaleconomische ongelijkheden in de geestelijke gezondheid en het gezondheidsgedrag van adolescenten en jongvolwassenen.

4.1. Het samenspel van sociale causatie en gezondheidsgerelateerde selectie bij de ontwikkeling van verschillen op het gebied van middelengebruik en mentale gezondheid tussen opleidingsniveaus

4.1.1. Directe gezondheidsgerelateerde selectie met betrekking tot aandachts-problemen/ADHD-symptomen

Aandachtsproblemen (**Hoofdstuk 3**) en ADHD-symptomen (**Hoofdstuk 5**) voorspelden dalingen in het onderwijsniveau tijdens de hele adolescentie en jongvolwassenheid. Onze resultaten bevestigen eerdere bevindingen dat jongeren met een ADHD-diagnose een hoger risico lopen op slechtere onderwijsresultaten en een lager opleidingsniveau op de lange termijn (Bortes et al., 2022; Mikkonen et al., 2018). We vonden geen verbanden tussen de hoeveelheid alcoholgebruik (**Hoofdstuk 2**) of externaliserend gedrag (na het controleren voor aandachtsproblemen) en dalingen in het onderwijsniveau (**Hoofdstuk 3**). Het is mogelijk dat dit komt omdat alleen zwaar alcoholgebruik en zware externaliserende gedragsproblemen tot dalingen in het onderwijsniveau leiden. Het is moeilijk om dergelijk ernstige problematieken te bestuderen met lineaire statistische modellen en in relatief kleinere cohortstudies zoals TRAILS. Eerdere studies gebaseerd op grotere steekproeven vonden dat externaliserende stoornissen, zoals anti-sociale gedragsstoornis, en zwaar middelenmisbruik of afhankelijkheid samenhingen met slechtere onderwijsuitkomsten op de lange termijn, zoals het verlaten van de middelbare school zonder diploma (Dalsgaard et al., 2020; Jensen et al., 2023; Mikkonen et al., 2018). Momenteel is er weinig bekend over de onderwijsresultaten van adolescenten en jongvolwassenen met ernstige psychiatrische problemen binnen selectieve onderwijssystemen zoals in Nederland. Daarom zou toekomstig onderzoek met grotere en klinische steekproeven in landen met dergelijke systemen waardevol zijn.

Gezondheidsgerelateerde selectie binnen het onderwijssysteem hoeft niet per se een gevolg te zijn van beperkingen die inherent zijn aan gezondheidsproblemen, maar kan ook afhangen van de sociale omstandigheden van jongeren thuis en op school. Ouders, leraren en klasgenoten kunnen reageren op mentale problemen van jongeren door steun aan te bieden of in te trekken en de hoeveelheid sociale steun zou een rol

kunnen spelen in hoe sterk een bepaald gezondheidsprobleem onderwijsprestaties beïnvloedt. In overeenstemming met dit idee vond een recente studie dat het verband tussen ADHD-symptomen en onderwijsprestaties varieerde tussen scholen, hetgeen suggereert dat verschillen in de schoolcontext inderdaad van belang zijn in de mate waarin ADHD-symptomen onderwijsresultaten beïnvloeden (Cheesman et al., 2022). Het verraste ons daarom dat drie belangrijke aspecten van de sociale omgeving van jongeren (gezinsfunctioneren, sociale steun van leraren, sociale steun van klasgenoten) niet als mediatoren bijdroegen aan verbanden tussen ADHD-symptomen en een lager onderwijsniveau. Bovendien verschilden deze verbanden niet tussen adolescenten die in een goed functionerend gezin opgroeiden en veel sociale steun rapporteerden en diegenen die dat niet deden (**Hoofdstuk 5**). Deze resultaten zouden kunnen betekenen dat verbanden tussen ADHD-symptomen en een lager onderwijsniveau erg robuust zijn en omgevingsfactoren er weinig invloed op hebben. Het is echter ook mogelijk dat onze maten van sociale steun en gezinsfunctioneren onvoldoende de aspecten van de sociale context die het belangrijkst voor de onderwijsresultaten van jongeren zijn hebben gemeten, zoals het delen van aantekeningen met klasgenoten, ouderlijke hulp bij het maken van huiswerk en praktische ondersteuning door leraren. Inderdaad zijn er eerste aanwijzingen dat de mate waarin aanpassingen in het onderwijs voor jongeren met ADHD mogelijk zijn bijdraagt aan verschillen tussen scholen in de relatie tussen ADHD-symptomen en onderwijsresultaten (Cheesman et al., 2022). Het is echter belangrijk om te benadrukken dat momenteel nog erg weinig bekend is over de rol van sociale factoren in de samenhang tussen ADHD-symptomen en onderwijsuitkomsten (Dvorsky & Langberg, 2016; Dvorsky et al., 2018). Desondanks lijkt het aannemelijk dat het niet eenvoudig zou zijn om voldoende steun te bieden aan leerlingen met ADHD in de grote klassen die met name het vwo, de havo en het primair onderwijs in Nederland kenmerken (Dutch House of Representatives, 2017; Dutch Ministry of Education, Culture and Science, 2018). Toekomstig onderzoek is hard nodig om te bekijken of dit daadwerkelijk het geval is.

4.1.2. Weinig bewijs voor indirecte gezondheidsgerelateerde selectie met betrekking tot cognitieve kenmerken in de kindertijd

Indirecte gezondheidsgerelateerde selectie verwijst naar de rol van kenmerken van het individu die zowel gezondheid en gezondheidsgedrag als het toekomstige onderwijsniveau zouden kunnen voorspellen, zoals IQ en zelfcontrole in de kindertijd (Brody, 1997; Daly & Egan, 2017; Daly et al., 2016; Deary et al., 2021; Kubička et al., 2001; Moffitt, 1993; Peeters et al., 2017; Veronneau et al., 2014; Wedow et al., 2018). Zelf worden deze kenmerken beïnvloed door de vroege omgeving (Farah, 2017; Ng-Knight & Schoon, 2017; Sturge-Apple et al., 2017) en genetische factoren (Deary et al., 2006; Yamagata et al., 2005). We stelden vast dat polygenetische scores voor roken inderdaad samenhingen met een lagere zelfcontrole, en polygenetische scores voor een lager onderwijsniveau

daarnaast ook met een lager IQ in de kindertijd. Een lagere zelfcontrole en een lager IQ voorspelden vervolgens selectie in een lager onderwijstraject. We vonden echter in de meeste analyses geen directe verbanden tussen cognitieve kenmerken in de kindertijd en het gezondheidsgedrag en de mentale gezondheid in de jeugd en jongvolwassenheid. Dienovereenkomstig veranderden de verbanden van opleidingsniveau met aandachtsproblemen, externaliserend gedrag, alcoholgebruik en roken nauwelijks na controle voor cognitieve kenmerken, terwijl deze verbanden aanzienlijk zouden afnemen wanneer er sterke indirecte gezondheidsgerelateerde selectie-effecten met betrekking tot cognitieve kenmerken aanwezig zouden zijn. Toch vonden we enig bewijs voor indirecte genetische gezondheidsgerelateerde selectie in de relatie tussen onderwijsniveau en roken. Het is mogelijk dat genetische varianten zowel roken als opleidingsniveau beïnvloeden via afzonderlijke fenotypische mechanismes, of via niet gemeten gedeelde fenotypische voorspellers van zowel roken als opleidingsniveau (Davies et al., 2019).

Het is belangrijk om te benadrukken dat het gebrek aan indirecte gezondheids-gerelateerde selectie met betrekking tot IQ en zelfcontrole niet betekent dat deze kenmerken geen cruciale rol spelen bij de ontwikkeling van gezondheidsverschillen tussen jongeren met verschillende opleidingsniveaus. Zowel IQ als zelfcontrole waren sterke voorspellers van de opleidingstrajecten van adolescenten en jongvolwassenen, die op hun beurt samenhingen met zowel roken als alcoholgebruik, overeenstemmend met sociale causatie-mechanismes (**Hoofdstukken 2 en 4**). Onze bevindingen bevestigen resultaten van eerdere studies, die aantonen dat verbanden van cognitieve vaardigheden met gezondheid en gezondheidsgedrag voor een substantieel deel worden gemedieerd door opleidingsniveau of andere aspecten van de sociaaleconomische status, en daarmee door de sociale omgeving (Calvin et al., 2011; Link et al., 2008; Wraw et al., 2016; Wrulich et al., 2013).

4.1.3. Sociale causatie met betrekking tot alcoholgebruik en roken, evenals externaliserend gedrag

Een lager opleidingsniveau in de vroege adolescentie hing samen met een toename van het alcoholgebruik in het midden van de adolescentie, terwijl er in de jongvolwassenheid een tendens was naar het tegenovergestelde verband, waarbij juist een hoger opleidingsniveau een toename van het alcoholgebruik voorspelde (**Hoofdstuk 2**). Onze bevindingen bevestigen resultaten uit het verleden, waarin ook een eerdere toename van het alcoholgebruik onder lager opgeleiden werd gevonden, en een omkering van de richting van het verband tussen opleidingsniveau en alcoholgebruik in de jongvolwassenheid (Jang et al., 2019; Latvala et al., 2014; Slutske, 2005). Het verband tussen een laag opleidingsniveau en roken veranderde niet van richting in de loop van de adolescentie en jongvolwassenheid (**Hoofdstuk 4**), wat overeenkomt met eerder onderzoek (Pampel et al., 2017).

Verschillen tussen onderwijsniveaus in middelengebruik zouden kunnen worden verklaard door verschillen in sociale normen en de samenstelling van peergroups. In Nederland worden klassen in de lagere onderwijstrajecten in de vroege adolescentie relatief sterk gekenmerkt door populariteitsnormen die roken en alcoholgebruik aanmoedigen, en deze normen voorspellen op hun beurt het middelengebruik van adolescenten (Peeters et al., 2021). Veranderingen in deze sociale normen, in combinatie met de eisen die een werkzaam leven en het stichten van een gezin aan iemand stellen, zouden kunnen verklaren waarom in de jongvolwassenheid het verband tussen opleidingsniveau en alcoholgebruik omkeerde in onze studie. De studentencultuur in Nederland wordt gekenmerkt door sociale normen die drinken sterk aanmoedigen, wat kan bijdragen aan een toename van het drankgebruik van jongvolwassenen binnen het hoger onderwijs (Fenton et al., 2023; Robertson & Tustin, 2018; Verhoog et al., 2019). Op dezelfde leeftijd hebben jongvolwassenen die het middelbaar beroepsonderwijs gevolgd hebben vaak al betaald werk en ze stichten ook vaak vroeger een gezin, twee situaties die moeilijk te combineren zijn met intensief alcoholgebruik (Green et al., 2017; Staff et al., 2010). Hoewel mensen met een lager opleidingsniveau vanaf de jonge volwassenheid in het algemeen niet meer drinken dan mensen met een hoger opleidingsniveau, komen problemen veroorzaakt door alcohol wel vaker voor bij mensen met een lager opleidingsniveau (Boyd et al., 2022). Toekomstige studies zouden kunnen onderzoeken tot hoeverre het eerdere begin van alcoholgebruik door laagopgeleiden zou kunnen bijdragen aan deze zogenaamde 'alcoholschade-paradox', aangezien vroeg alcoholgebruik een belangrijke voorspeller is van later problematisch gebruik en de ontwikkeling van een alcoholverslaving (Grant & Dawson, 1997). Aanvullend onderzoek is nodig om beter te begrijpen welke mechanismes ten grondslag liggen aan verschillen in sociale normen met betrekking tot middelengebruik tussen onderwijsniveaus binnen het Nederlandse onderwijsstelsel in de vroege adolescentie. Het zou kunnen dat de psychosociale consequenties van de buitengewoon vroege indeling in verschillende onderwijsniveaus hierbij een rol spelen (Elstad, 2010; Van Houtte & Stevens, 2008).

We vonden dat adolescenten met een lagere ouderlijke sociaaleconomische status vaker in de lagere opleidingstrajecten geselecteerd werden en ook vaker last hadden van externaliserende en aandachtsproblemen in de vroege adolescentie dan adolescenten uit hogere sociale klassen. Een lage ouderlijke sociaaleconomische status droeg ook bij aan het verband tussen een lager opleidingsniveau in de vroege adolescentie en een toename van externaliserend gedrag halverwege de adolescentie (**Hoofdstuk 3**). Onze resultaten komen overeen met eerder onderzoek uit Nederland waaruit blijkt dat een lagere ouderlijke sociaaleconomische status een sterke voorspeller is van selectie in een lager opleidingstraject, ook na correctie voor verschillen in cognitieve kenmerken (van Spijker et al., 2017; Weinberg et al., 2019). Eerdere studies hebben aangetoond dat leraren, misschien onbewust, lagere verwachtingen hebben van kinderen uit gezinnen met een

lagere sociaaleconomische status, mogelijk omdat ze aannemen dat deze kinderen thuis minder hulp bij school-gerelateerde zaken krijgen (Timmermans et al., 2015; Weinberg et al., 2019). Daarbij komen ouders met een hogere sociaaleconomische status vaak effectiever op voor hun kinderen op school en kunnen ze zo de verwachtingen van leraren tegenover de toekomstige schoolprestaties van hun kind verhogen (Seghers et al., 2021; Weinberg et al., 2019).

Onze bevindingen komen ook overeen met eerdere resultaten die een verband aantonen tussen een lagere ouderlijke sociaaleconomische status en meer psychiatrische problemen bij adolescenten, waaronder externaliserend gedrag en ADHD-symptomen (Piotrowska et al., 2015; Reiss, 2013; Russell et al., 2016). Dit kan worden verklaard door een hogere prevalentie van risicofactoren voor psychische problemen, zoals stress in de vroege kindertijd, wonen in een arme wijk, stressvolle levensgebeurtenissen en slecht gezinsfunctioneren in gezinnen met een lagere sociaaleconomische status (Barnhart et al., 2022; Markham & Spencer, 2022; Piotrowska et al., 2019; Sharp et al., 2021). Naast met meer aandachtsproblemen en externaliserend gedrag hing een lagere ouderlijke sociaaleconomische status in ons onderzoek ook samen met meer alcoholgebruik in de vroege adolescentie, maar met minder alcoholgebruik in de jongvolwassenheid (**Hoofdstuk 2**). Een mogelijke verklaring voor deze bevinding is dat jongvolwassenen uit gezinnen met een hogere sociaaleconomische status minder vaak bij hun ouders wonen terwijl ze het hoger onderwijs volgen (van den Berg, 2020). Uit eerder onderzoek is gebleken dat jongvolwassenen die bij hun ouders wonen tijdens hun studie minder drinken dan hun leeftijdsgenoten die zelfstandig wonen, mogelijk vanwege meer sociale controle in het ouderlijk huis (Benz et al., 2017).

4.2. Landelijke sociale mobiliteit alleen van belang voor het verband tussen gezinswelvaart en lichamelijke activiteit

We ontdekten dat adolescenten met een lage gezinswelvaart die in landen met een hoge mate van sociale mobiliteit gedurende de vorige generaties woonden minder geneigd waren om lichamelijk actief te zijn dan adolescenten met een vergelijkbare gezinswelvaart in landen met een relatief lage sociale mobiliteit. Sociale mobiliteit op landelijk niveau hing niet samen met internationale variaties in sociaaleconomische ongelijkheden in de andere soorten gezondheidsgedrag die we hebben bestudeerd (d.w.z. regelmatig ontbijten, consumptie van gezond en ongezond voedsel, roken) (**Hoofdstuk 6**). Het is onwaarschijnlijk dat dit resultaat wordt verklaard door een sterkere indirecte gezondheidsgerelateerde selectie in landen met een hoge mate van sociale mobiliteit gedurende de vorige generaties. Cognitieve kenmerken en andere gezondheidsgerelateerde individuele verschillen zouden samen moeten hangen met meerdere soorten gezondheidsgedrag, in plaats van specifiek met lichamelijke activiteit. Toekomstige studies zouden zich kunnen richten op kenmerken op landelijk niveau die verband houden met sociale mobiliteit en

die specifiek van invloed zouden kunnen zijn op ongelijkheden in lichamelijke activiteit. Sociale mobiliteit op landelijk niveau is bijvoorbeeld sterk geassocieerd met eigenschappen van het onderwijssysteem, waaronder de mate van stratificatie en selectiviteit (Hanushek & Wößmann, 2006; Pekkarinen, 2018). Onderwijssystemen verschillen aanzienlijk wat betreft de tijd die binnen het curriculum wordt besteed aan lichamelijke opvoeding en de mate waarin de deelname aan sport op school vereist is (Bann et al., 2019). Dit kan gevolgen hebben voor verschillen tussen landen in de betrokkenheid van jongeren met een lage sociaaleconomische status bij sport, zowel binnen het formele curriculum als tijdens buitenschoolse activiteiten.

Ten slotte zou toekomstig internationaal onderzoek zich specifiek kunnen richten op de gezondheid van de adolescenten en jongvolwassenen uit de allerarmste gezinnen. Het beperkte beschikbare onderzoek dat zich specifiek richt op de meest kwetsbare bevolkingsgroepen suggereert dat de gezondheid van deze groepen vaak het meest wordt beïnvloed door landelijke factoren, waaronder veranderingen in het sociale beleid en macro-economische omstandigheden. Zo was er in Schotland een sterke toename van voortijdige sterfgevallen in de allerlaagste sociaaleconomische groepen in de afgelopen tien jaar. Deze bevinding kan worden verklaard door veranderingen in landelijke omstandigheden die de gezondheid van de meest kwetsbare bevolkingsgroepen onevenredig hadden beïnvloed, waaronder sterke verlagingen van de overheidsuitgaven, en recentelijk de COVID-19-pandemie en oplopende inflatie (Walsh & McCartney, 2023). Over hoe verschillen tussen landen in (veranderingen in) omstandigheden op landelijk niveau verband houden met de gezondheid van de meest kwetsbare adolescenten is momenteel nog erg weinig bekend; dit zou onderwerp kunnen zijn van toekomstig onderzoek.

5. ALGEMENE CONCLUSIE

Het begin van het derde decennium van de 21e eeuw wordt gekenmerkt door een syndemie van meerdere crisissen, waaronder de COVID-19-pandemie, de toenemende klimaatverandering, een nieuwe oorlog in Europa en een hoge inflatie in veel landen, waaronder Nederland. Deze crisissen zullen allemaal bijdragen aan een verdere toename van gezondheidsverschillen (Castro et al., 2022; Bambra et al., 2020; Limb, 2022), en het is bekend dat crisissen de gezondheid van jongeren en jongvolwassenen sterker aantasten dan die van andere bevolkingsgroepen (Kadir et al., 2019; McGushin et al., 2022; Panchal et al., 2021; Scharpf et al., 2021; Skinner et al., 2023; UK Youth, 2023). Bovendien hebben zich belangrijke veranderingen op de arbeidsmarkt voorgedaan, waaronder een toegenomen automatisering van taken en offshoring, met de daarmee samenhangende afgenomen beschikbaarheid van banen en een relatieve daling van lonen voor lager

opgeleiden, evenals een toename van onzeker werk (Gray et al., 2021; O'Brien et al., 2022). Studies suggereren dat deze veranderingen mogelijk hebben bijgedragen aan een groei van gezondheidsverschillen (Gray et al., 2021; LaMontagne, 2010; O'Brien et al., 2022). Tegelijkertijd hebben veel welvarende landen de afgelopen decennia de uitgaven voor hun sociale beschermingsstelsels verlaagd. Er zijn steeds meer aanwijzingen dat dit negatieve gevolgen heeft gehad op de gezondheid van de meest kwetsbare bevolkingsgroepen (Labonté & Stuckler, 2016; Stuckler et al., 2017; Walsh & McCartney, 2023; Wickham et al., 2020). Onderzoek en actie om gezondheidsverschillen te verlagen zijn daarom misschien nog nooit zo urgent geweest als nu.

Ik hoop dat dit proefschrift een kleine bijdrage kan leveren om de mechanismes achter de ontwikkeling van gezondheidsverschillen in de adolescentie en jongvolwassenheid beter te begrijpen. Onze bevindingen steunen het idee dat er een complex samenspel van individuele kenmerken en de sociale context ten grondslag ligt aan de ontwikkeling van sociaaleconomische gezondheidsverschillen. Onze resultaten toonden robuuste nadelige effecten van ADHD-symptomen en aandachtsproblemen op het opleidingsniveau (een voorbeeld van directe gezondheidsgerelateerde selectie). Ten aanzien van opleidingsverschillen in het roken en het alcoholgebruik van adolescenten en jongvolwassenen leken sociale causatie-mechanismes te overheersen. Onze resultaten met betrekking tot de vraag of de landelijke sociale mobiliteit het verband tussen gezinswelvaart en het gezondheidsgedrag van adolescenten modereert waren inconsistent. In toekomstig onderzoek zouden complexe systeembenaderingen kunnen worden gebruikt om het dynamische samenspel van kenmerken van het individu, de thuis- en schoolcontext en de bredere landelijke context bij het ontstaan van gezondheidsverschillen in het verloop van de ontwikkeling inzichtelijk te maken (Diez Roux, 2011; Holdsworth et al., 2017). Deze benaderingen zouden ook rekening kunnen houden met snelle veranderingen in omstandigheden op macroniveau, inclusief schokken en crisissen, op gezondheidsverschillen, in het bijzonder met betrekking tot de meest kwetsbare adolescenten en jongvolwassenen.

SUPPLEMENTARY MATERIALS

SUPPLEMENTARY MATERIALS – CHAPTER 2

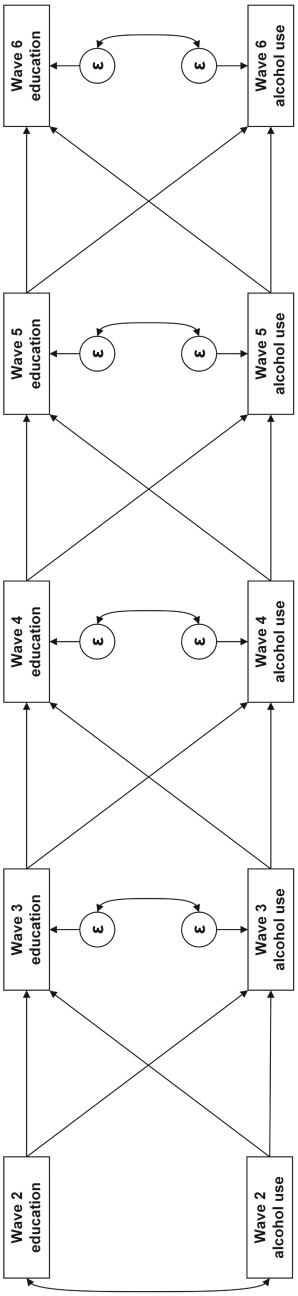


FIGURE 2.S1 Path diagram of a bivariate cross-lagged panel model

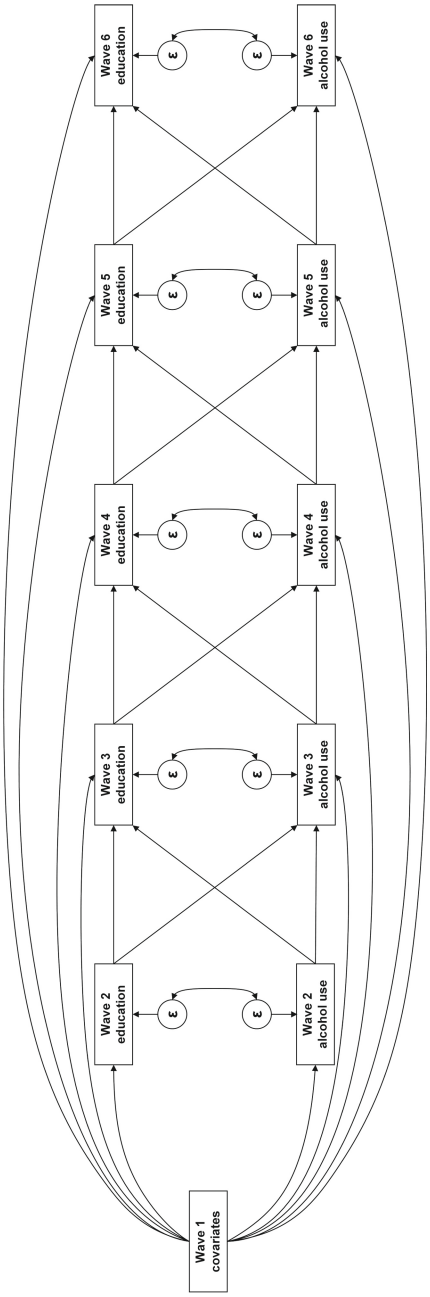


FIGURE 2.S2 Path diagram of a cross-lagged panel model adjusted for time-invariant baseline characteristics

FIGURE 2.S3 Path diagrams of one-sided cross-lagged panel models with fixed effects according to the specification by Allison et al. (2017); separate fixed effects models were fit to assess each of the two hypothesized causal directions between educational level and alcohol use

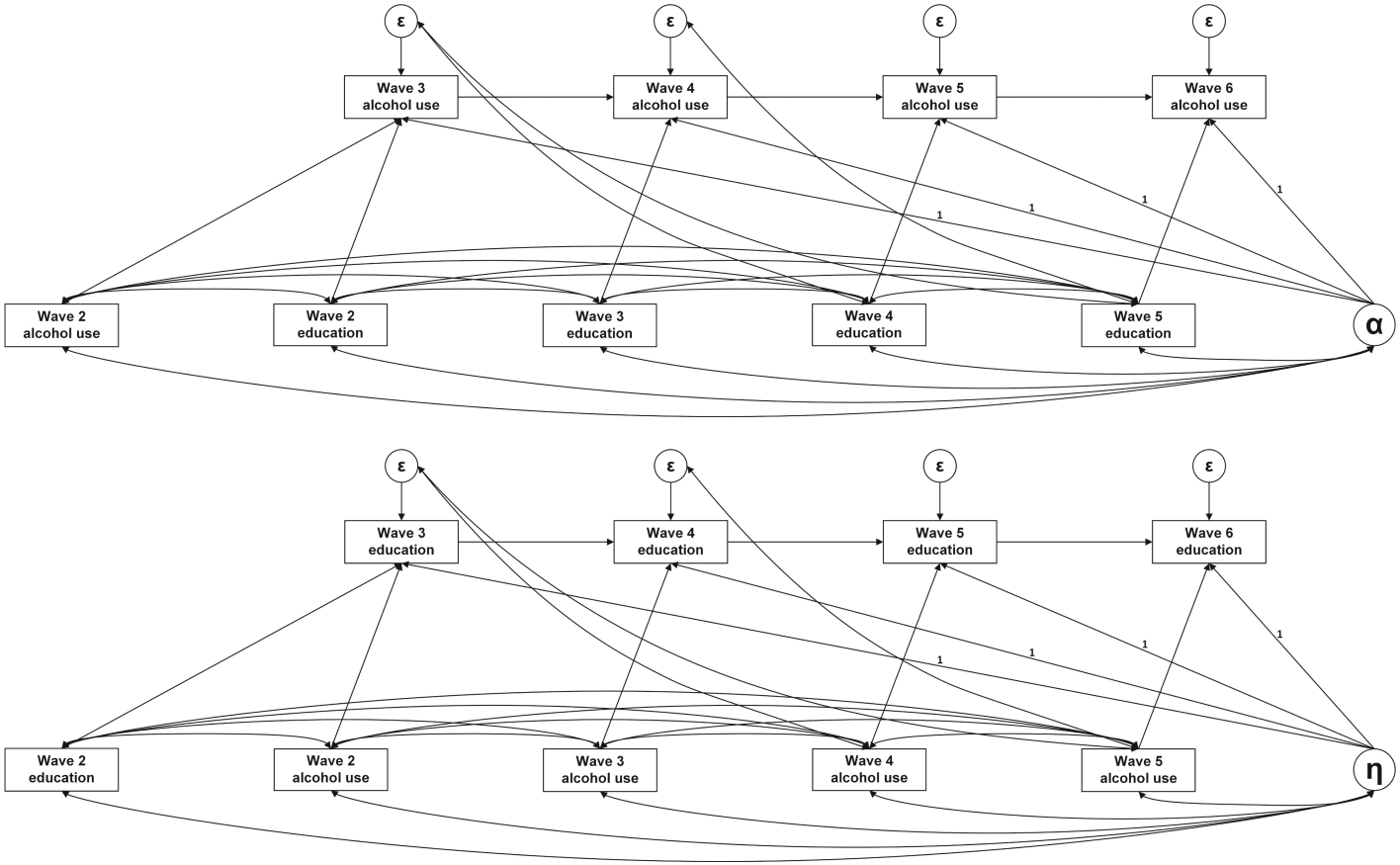


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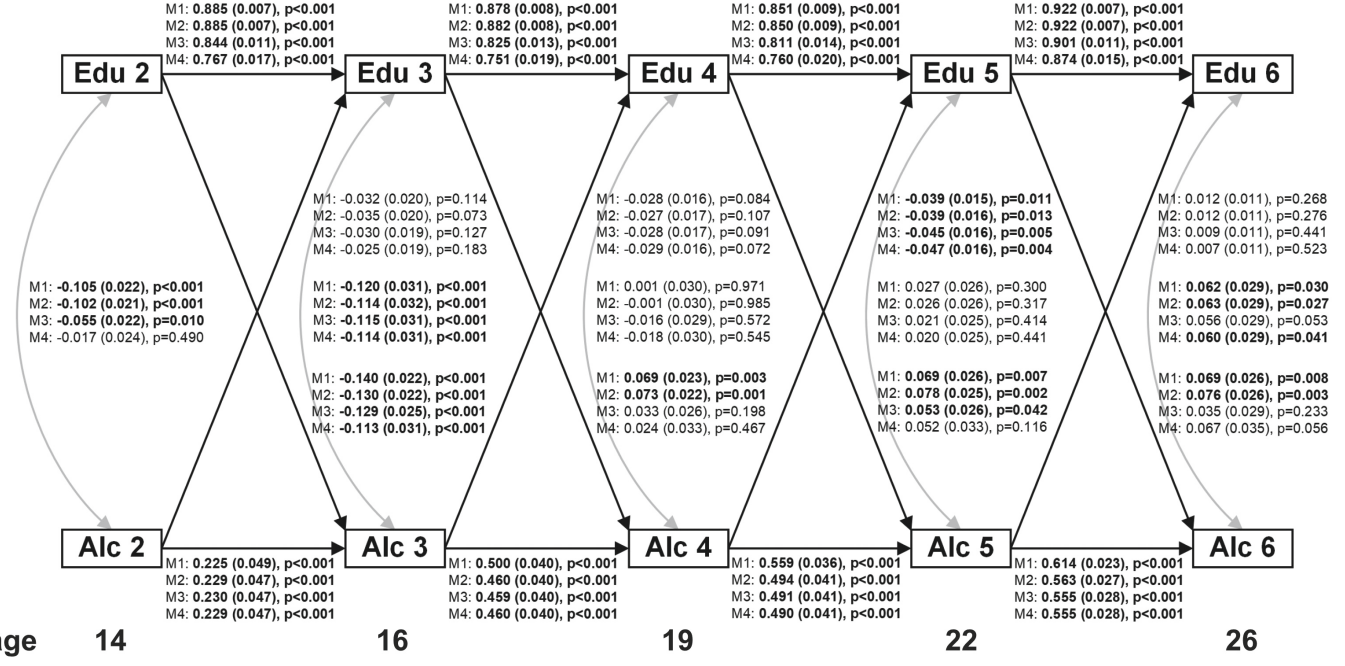
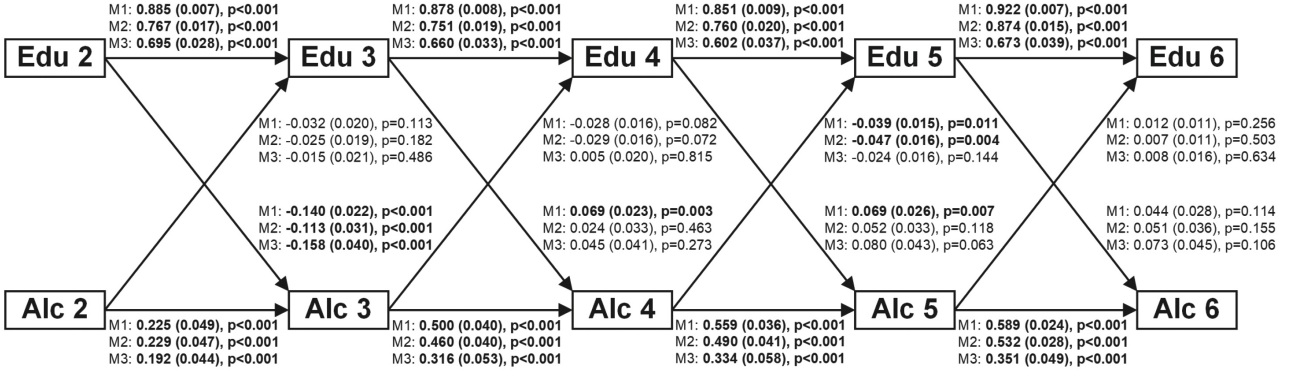
FIGURE 2.S4 Bidirectional associations between educational level and alcohol use in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); sequentially adjusted linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) for different sets of covariates



	CFI	TLI	SRMR	RMSEA
Model 1	0.984	0.969	0.032	0.045
Model 2	0.990	0.948	0.019	0.044
Model 3	0.992	0.956	0.017	0.039
Model 4	0.994	0.967	0.014	0.033

Model 1: bivariate cross-lagged panel model. Model 2: cross-lagged panel model adjusted for demographics (age, gender, area of residence, and ethnicity). Model 3: cross-lagged panel model adjusted for demographics, and parental socioeconomic status. Model 4: cross-lagged panel model adjusted for demographics, parental socioeconomic status, and adolescent psychological characteristics (IQ, effortful control). Edu = educational level; Alc = alcohol use. ***Boldface*** *denotes statistical significance at p < 0.05.*

FIGURE 2.S5 Bidirectional associations between educational level and alcohol use in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) from cross-lagged panel models without (Model 1 and 2) and with fixed effects (Model 3); the binge drinking item was removed from the AUDIT-C in these models



	CFI	TLI	SRMR	RMSEA
Model 1	0.985	0.972	0.030	0.043
Model 2	0.995	0.972	0.013	0.030
Model 3				
a) Education → Alcohol use	1.000	0.998	0.008	0.011
b) Alcohol use → Education	0.997	0.989	0.007	0.029

Model 1: bivariate cross-lagged panel model. Model 2: cross-lagged panel model adjusted for age, gender, area of residence, ethnicity, parental socioeconomic status, IQ, and effortful control at baseline (wave 1). Model 3: cross-lagged panel models with fixed effects – adjustment for time-invariant characteristics was performed by inclusion of a latent variable. Edu = educational level; Alc = alcohol use. ***Boldface*** *denotes statistical significance at p < 0.05.*

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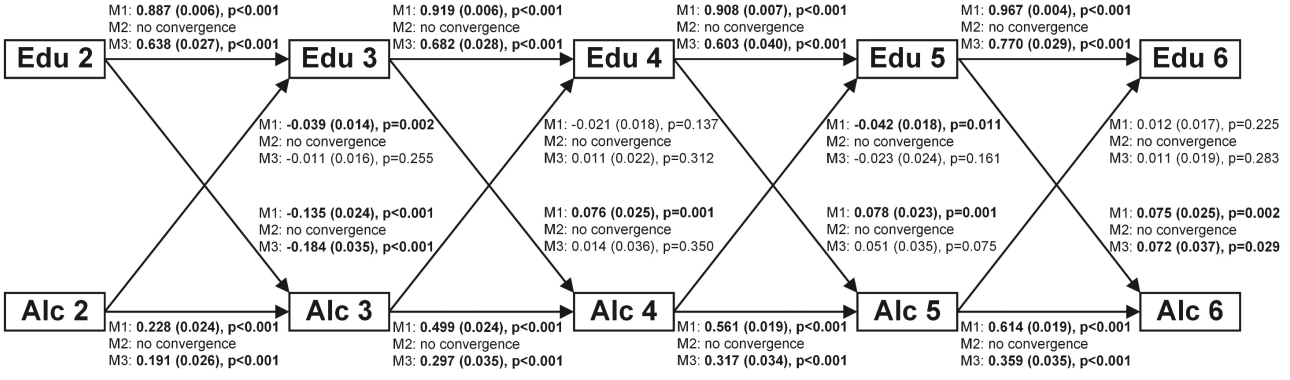
FIGURE 2.S6 Bidirectional associations between educational level and alcohol use in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) from cross-lagged panel models without (Model 1 and 2) and with fixed effects (Model 3), using the Bayes estimator; educational level from wave 3 to wave 5 was declared as "categorical" in Mplus



	Posterior predictive p-value	95% confidence interval for the difference between the observed and the replicated chi-square values
Model 1	0.229	-21.324 – 44.649
Model 2	no convergence	no convergence
Model 3		
a) Education → Alcohol use	0.398	-26.475 – 32.540
b) Alcohol use → Education	0.433	-27.114 – 31.927

Model 1: bivariate cross-lagged panel model. Model 2: cross-lagged panel model adjusted for age, gender, area of residence, ethnicity, parental socioeconomic status, IQ, and effortful control at baseline (wave 1). Model 3: cross-lagged panel models with fixed effects – adjustment for time-invariant characteristics was performed by inclusion of a latent variable. Edu = educational level; Alc = alcohol use. ***Boldface*** *denotes statistical significance at p < 0.05.*

TABLE 2.S1: Attrition analysis – characteristics of young adults remaining in the TRAILS study (the Netherlands, 2000–2017, N = 2,229) at wave 6, compared to participants who had dropped out of the cohort between wave 2 and wave 5

	Participants remaining in TRAILS by wave 6		**Drop-outs**		**P-value**
N participants, %	1,616	(72.50)	613	(27.50)	
Baseline characteristics					
Male gender, N (%)	735	(45.48)	363	(59.22)	<0.001
Non-Dutch ethnicity, N (%)	155	(9.59)	146	(23.82)	<0.001
Age, mean (SD)	11.09	(0.56)	11.16	(0.54)	0.006
Parental socioeconomic status (SES), mean (SD)	0.10	(0.76)	-0.44	(0.77)	<0.001
Wechsler Intelligence Deviation Quotient (IQ), mean (SD)	99.64	(14.54)	90.67	(14.24)	<0.001
Effortful control, mean (SD)	3.27	(0.68)	3.09	(0.67)	<0.001
Educational level, mean (SD)					
Wave 2	2.53	(1.14)	1.72	(0.97)	<0.001
Wave 3	2.64	(1.10)	1.95	(1.06)	<0.001
Wave 4	2.73	(0.95)	2.39	(1.02)	<0.001
Wave 5	2.83	(0.90)	2.29	(1.01)	<0.001
Alcohol use (quantity-frequency score), mean (SD)					
Wave 2	1.55	(4.61)	1.94	(4.37)	0.112
Wave 3	6.48	(8.76)	9.58	(12.87)	<0.001
Wave 4	10.20	(11.74)	10.02	(10.82)	0.841
Wave 5	10.09	(11.01)	11.95	(10.98)	0.153

SD = standard deviation. P-values were computed using chi-squared tests for categorical variables and two-sample t-tests for continuous variables.

TABLE 2.S2: Characteristics of participants with classifiable educational level compared to those with missing/unclassifiable educational level from wave 2 to wave 6 in the TRAILS study (the Netherlands, 2000–2017, N = 2,229)

	N participants per wave		**Male gender**		**Non-Dutch ethnicity**	
	N	(%)	N	(%)	N	(%)
Wave 2	2,148	(100)				
Education complete	1,927	(89.71)	924	(47.95)	237	(12.30)
Education unclassifiable/missing	221	(10.29)	130	(58.82)	35	(15.84)
P-value				0.002		0.134
Wave 3	1,818	(100)				
Education complete	1,529	(84.10)	704	(46.04)	175	(11.45)
Education unclassifiable/missing	289	(15.90)	163	(56.40)	36	(12.46)
P-value				0.001		0.623
Wave 4	1,880	(100)				
Education complete	1,507	(80.16)	671	(44.53)	160	(10.62)
Education unclassifiable/missing	373	(19.84)	227	(60.86)	52	(13.94)
P-value				<0.001		0.069
Wave 5	1,781	(100)				
Education complete	1,429	(80.24)	624	(43.67)	147	(10.29)
Education unclassifiable/missing	352	(19.76)	219	(62.22)	52	(14.77)
P-value				<0.001		0.017
Wave 6	1,616	(100)				
Education complete	1,192	(73.76)	474	(39.77)	106	(8.89)
Education unclassifiable/missing	424	(26.24)	261	(61.56)	49	(11.56)
P-value				<0.001		0.110

SD = standard deviation. [a]Alcohol use was measured using a quantity-frequency score from wave 2 to wave 5, and using the AUDIT-C at wave 6. P-values were computed using chi-squared tests for categorical variables and two-sample t-tests for continuous variables.

Age at baseline		Parental socioeconomic status (SES)		Wechsler Intelligence Deviation Quotient (IQ)		Effortful control		Concurrent alcohol use[a]	
Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
11.11	(0.56)	-0.03	(0.80)	97.58	(15.02)	3.22	(0.69)	1.63	(4.63)
11.07	(0.53)	-0.07	(0.75)	96.01	(13.44)	3.27	(0.64)	1.76	(3.83)
	0.344		0.531		0.138		0.315		0.703
11.09	(0.56)	0.08	(0.78)	99.75	(14.85)	3.26	(0.69)	6.59	(9.28)
11.19	(0.57)	-0.32	(0.78)	91.25	(13.22)	3.04	(0.63)	9.78	(11.12)
	0.004		<0.001		<0.001		<0.001		<0.001
11.09	(0.57)	0.13	(0.76)	100.34	(14.52)	3.30	(0.68)	9.99	(11.23)
11.10	(0.52)	-0.33	(0.74)	91.58	(13.13)	3.02	(0.63)	11.33	(13.87)
	0.693		<0.001		<0.001		0.001		0.110
11.10	(0.56)	0.12	(0.76)	100.13	(14.68)	3.30	(0.68)	10.24	(11.12)
11.11	(0.55)	-0.23	(0.76)	93.78	(13.88)	3.07	(0.66)	9.51	(9.69)
	0.608		<0.001		<0.001		<0.001		0.492
11.09	(0.56)	0.17	(0.75)	100.82	(14.47)	3.32	(0.67)	4.55	(2.37)
11.10	(0.56)	-0.09	(0.76)	96.31	(14.22)	3.11	(0.68)	4.97	(2.77)
	0.630		<0.001		<0.001		<0.001		0.078

TABLE 2.S3 The association between baseline characteristics (wave 1) and alcohol use and educational level from wave 2 to wave 6 in the TRAILS study (the Netherlands, 2000–2017, N = 2,229) in the multivariate-adjusted cross-lagged panel model (model 2) in Figure 2.2; linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value)

	Wave 2	**Wave 3**	**Wave 4**	**Wave 5**	**Wave 6**
	Educational level				
Male gender	-0.029 (0.015), p=0.060	-0.019 (0.012), p=0.107	0.003 (0.012), p=0.799	0.001 (0.014), p=0.935	0.002 (0.013), p=0.852
District					
City of Groningen	ref	ref	ref	ref	ref
Leeuwarden	**-0.037 (0.017), p=0.034**	**0.027 (0.013), p=0.040**	-0.014 (0.014), p=0.331	0.027 (0.015), p=0.075	-0.009 (0.012), p=0.454
Assen	**-0.039 (0.019), p=0.046**	**-0.043 (0.014), p=0.001**	-0.003 (0.015), p=0.852	-0.020 (0.017), p=0.237	0.000 (0.015), p=0.979
Other regions	**-0.057 (0.019), p=0.003**	-0.028 (0.015), p=0.059	0.018 (0.015), p=0.235	-0.010 (0.017), p=0.549	-0.011 (0.014), p=0.403
Non-Dutch ethnicity	0.009 (0.016), p=0.570	**0.033 (0.012), p=0.007**	**0.034 (0.014), p=0.014**	0.007 (0.017), p=0.668	**0.031 (0.013), p=0.018**
Age	0.001 (0.018), p=0.949	-0.015 (0.014), p=0.275	0.008 (0.014), p=0.566	**-0.033 (0.016), p=0.042**	-0.007 (0.013), p=0.613
Parental SES	**0.280 (0.017), p<0.001**	**0.081 (0.014), p<0.001**	**0.109 (0.015), p<0.001**	**0.073 (0.018), p<0.001**	**0.038 (0.014), p=0.007**
IQ	**0.462 (0.016), p<0.001**	**0.075 (0.015), p<0.001**	**0.081 (0.015), p<0.001**	**0.067 (0.018), p<0.001**	**0.039 (0.014), p=0.004**
Effortful control	**0.249 (0.017), p<0.001**	**0.079 (0.014), p<0.001**	**0.063 (0.015), p<0.001**	0.032 (0.017), p=0.055	0.021 (0.013), p=0.112
	Alcohol use				
	Quantity-frequency score				AUDIT-C score
Male gender	-0.024 (0.022), p=0.267	**0.174 (0.023), p<0.001**	**0.213 (0.023), p<0.001**	**0.215 (0.025), p<0.001**	**0.128 (0.027), p<0.001**
District					
City of Groningen	ref	ref	ref	ref	ref
Leeuwarden	0.004 (0.027), p=0.896	0.051 (0.027), p=0.057	-0.033 (0.026), p=0.208	-0.012 (0.023), p=0.610	-0.034 (0.026), p=0.197
Assen	**-0.052 (0.027), p=0.049**	-0.010 (0.025), p=0.697	-0.032 (0.025), p=0.204	-0.007 (0.025), p=0.772	-0.028 (0.028), p=0.330
Other regions	-0.008 (0.029), p=0.767	0.035 (0.032), p=0.286	-0.004 (0.025), p=0.861	-0.014 (0.026), p=0.584	-0.016 (0.028), p=0.577
Non-Dutch ethnicity	0.000 (0.030), p=0.995	**-0.058 (0.023), p=0.011**	**-0.044 (0.018), p=0.018**	-0.016 (0.023), p=0.494	-0.038 (0.032), p=0.243
Age	0.002 (0.029), p=0.956	0.030 (0.026), p=0.254	0.000 (0.025), p=0.994	-0.036 (0.026), p=0.173	-0.012 (0.027), p=0.665

TABLE 2.S3 Continued

	Wave 2	Wave 3	Wave 4	Wave 5	Wave 6
Parental SES	**-0.086 (0.021), p<0.001**	0.000 (0.026), p=0.987	**0.075 (0.027), p=0.005**	0.044 (0.027), p=0.107	**0.085 (0.029), p=0.004**
IQ	**-0.052 (0.026), p=0.045**	-0.020 (0.032), p=0.531	0.030 (0.028), p=0.293	0.021 (0.027), p=0.430	-0.021 (0.028), p=0.452
Effortful control	-0.042 (0.024), p=0.079	-0.011 (0.027), p=0.682	-0.016 (0.025), p=0.535	-0.024 (0.024), p=0.323	-0.051 (0.027), p=0.056

All predictors are mutually adjusted. From wave 3 to wave 6, all predictors are additionally adjusted for educational level and alcohol use in the preceding wave. ***Boldface*** *denotes statistical significance at p < 0.05.*

TABLE 2.S4 Post-hoc analysis – the association between baseline characteristics (wave 1) and alcohol use at wave 2 in the TRAILS study (the Netherlands, 2000–2017, N = 2,229) in the multivariate-adjusted cross-lagged panel model (Figure 2.2, Model 2) after additionally regressing wave 2 alcohol use on wave 2 education; linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value)

	Alcohol quantity-frequency score
Male gender	-0.025 (0.022), p=0.254
District	
City of Groningen	ref
Leeuwarden	0.003 (0.027), p=0.921
Assen	**-0.053 (0.026), p=0.042**
Other regions	-0.010 (0.028), p=0.727
Non-Dutch ethnicity	0.000 (0.030), p=0.989
Age	0.002 (0.029), p=0.955
Parental socioeconomic status (SES)	**-0.079 (0.024), p=0.001**
Wechsler Intelligence Deviation Quotient (IQ), mean (SD)	-0.041 (0.034), p=0.222
Effortful control	-0.036 (0.027), p=0.177
Wave 2 educational level	-0.024 (0.035), p=0.490
Fit measures	
CFI	0.994
TLI	0.967
SRMR	0.014
RMSEA	0.033

All predictors are mutually adjusted. ***Boldface*** *denotes statistical significance at p < 0.05.*

SUPPLEMENTARY MATERIALS – CHAPTER 3

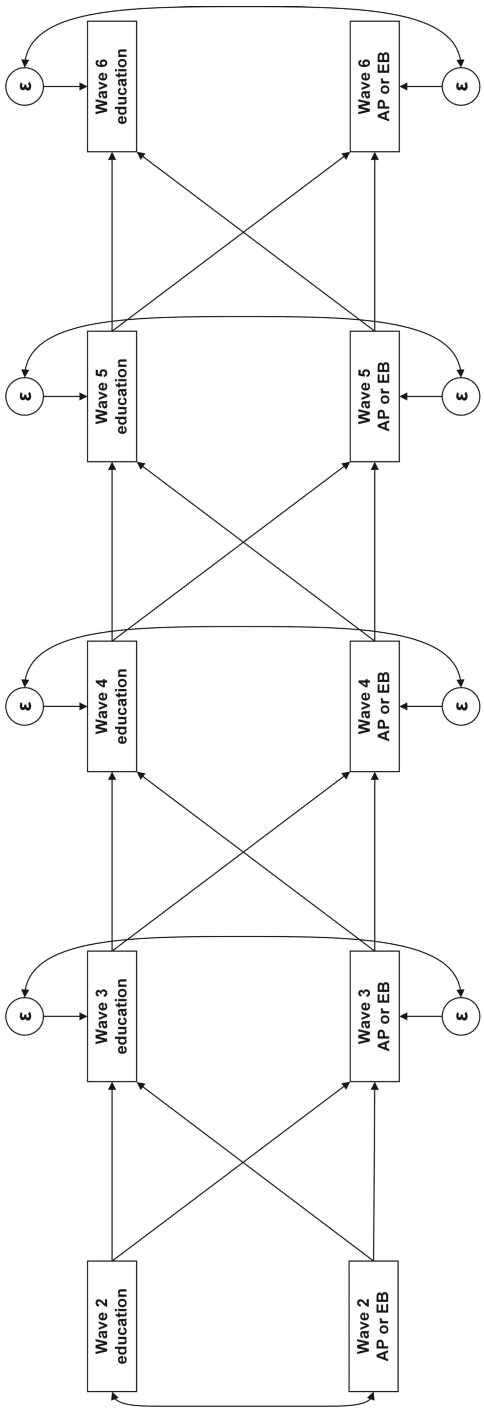
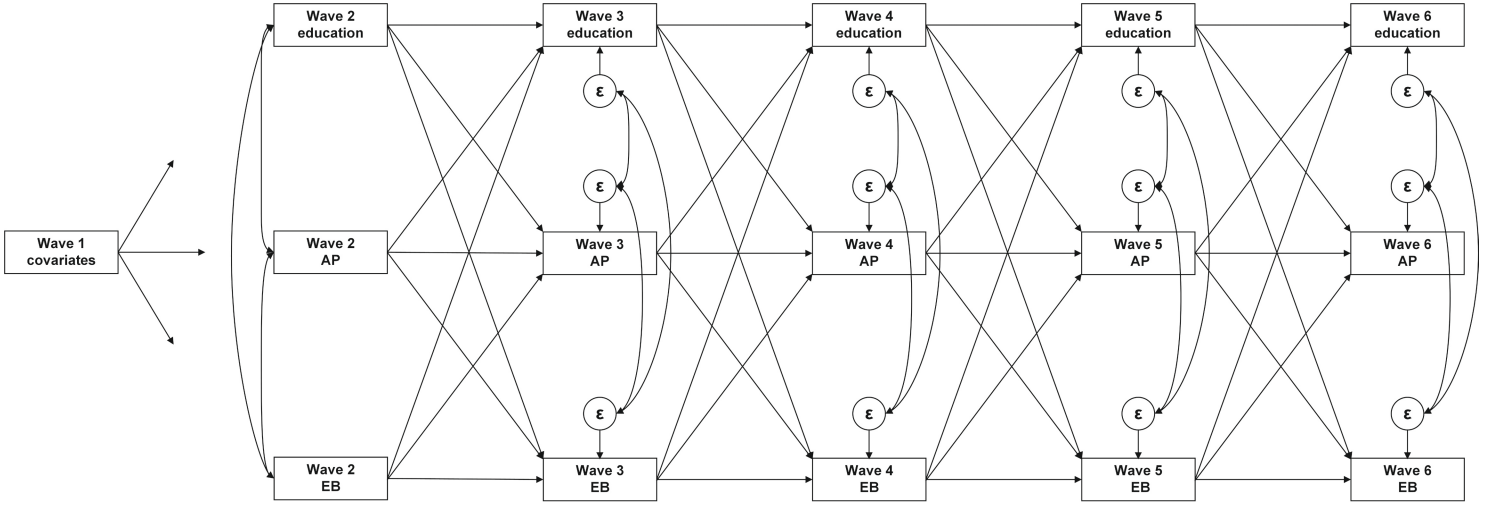


FIGURE 3.S1 Path diagram of a bivariate cross-lagged panel model

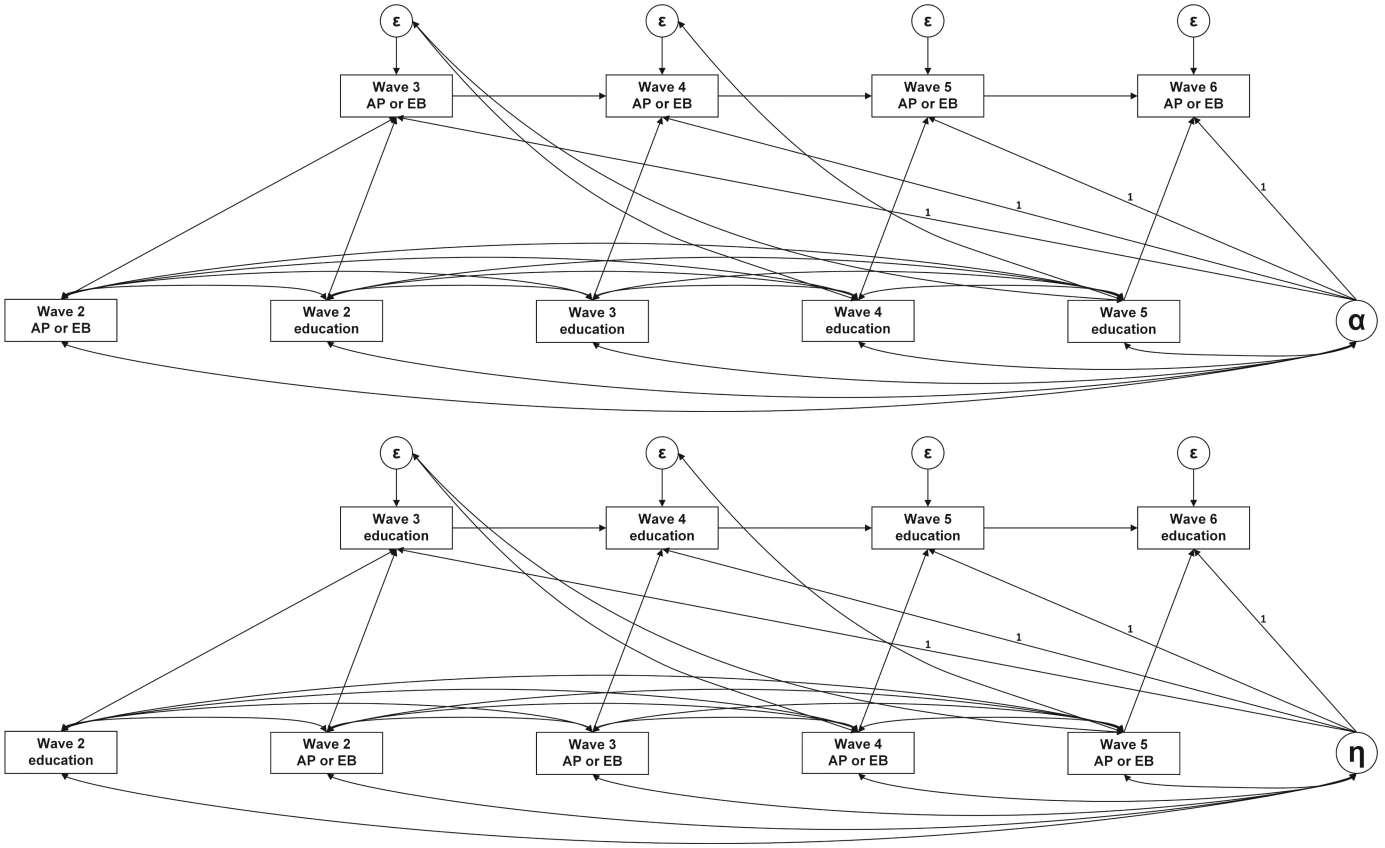
Edu = educational level; AP = attention problems; EB = externalizing behaviour.

FIGURE 3.S2 Path diagram of a cross-lagged panel model with multiple time-varying variables, adjusted for time-invariant baseline characteristics



Baseline (wave 1) covariates include age, gender, area of residence, ethnicity, parental SES, and IQ and predict all time-varying variables (i.e., education, attention problems, externalizing behaviour) at all subsequent waves. Arrows from wave 1 covariates pointing into the time-varying variables are not displayed in the figure for reasons of clarity. Edu = educational level; AP = attention problems; EB = externalizing behaviour.

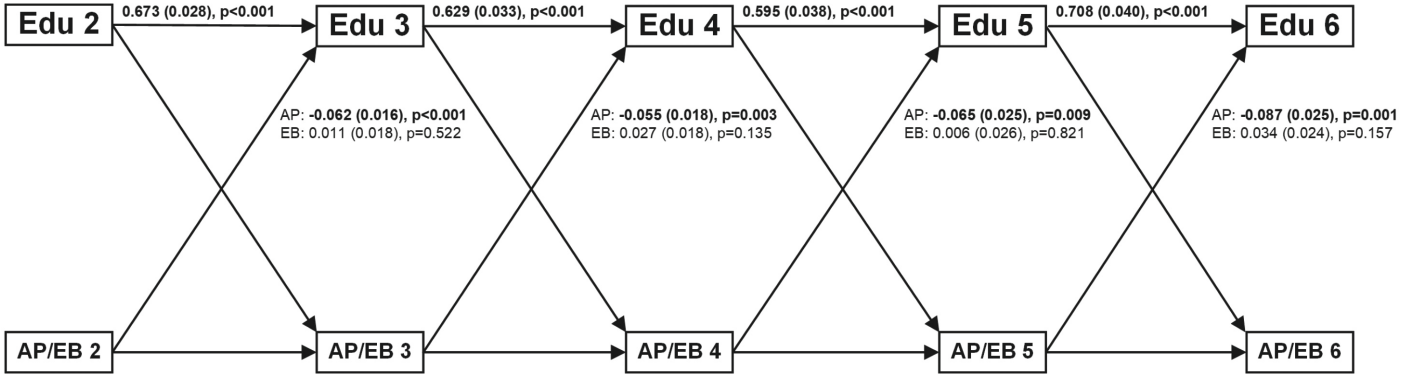
FIGURE 3.S3 Path diagrams of one-sided cross-lagged panel models with fixed effects (without covariates) according to the specification by Allison et al. (2017); separate fixed effects models were fit to assess each of the two hypothesized causal directions between educational level and attention problems (AP) and externalizing behaviour (EB)



Edu = educational level; AP = attention problems; EB = externalizing behaviour.

A

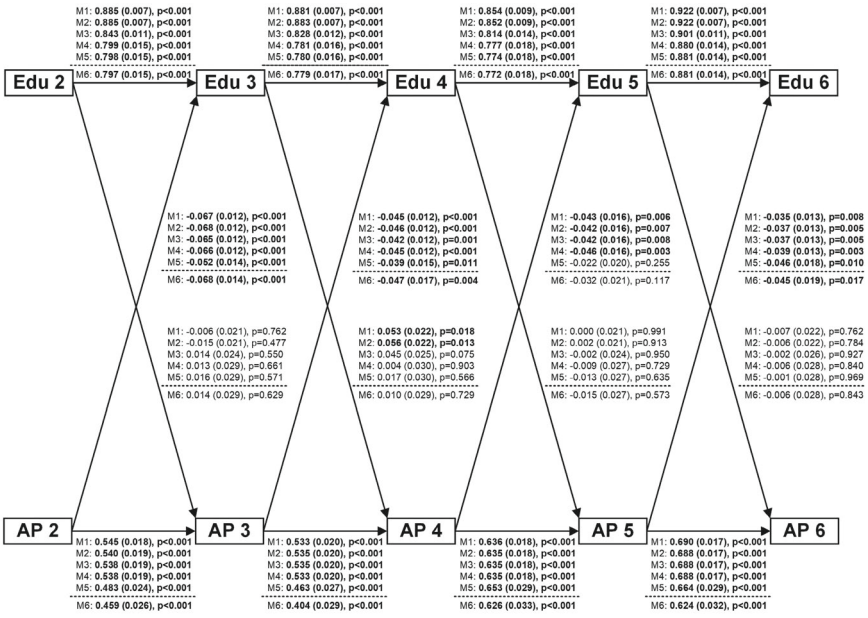
FIGURE 3.S4 Covariate-adjusted one-sided cross-lagged panel model with fixed effects for the direction from attention problems/externalizing behaviour (AP/EB) to educational level (i.e., health-related selection) in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value)



	Chi-square	df	CFI	TLI	SRMR	RMSEA
AP/*EB* → *Education*	29.183	14	0.999	0.987	0.003	0.022

Adjusted for baseline (wave 1) covariates (i.e., age, gender, area of residence, ethnicity, parental SES, and IQ). Attention problems and externalizing behaviour are mutually adjusted. Adjustment for unassessed time-invariant characteristics was performed by inclusion of a latent variable. Edu = educational level; AP = attention problems; EB = externalizing behaviour. ***Boldface*** *denotes statistical significance at p < 0.05.*

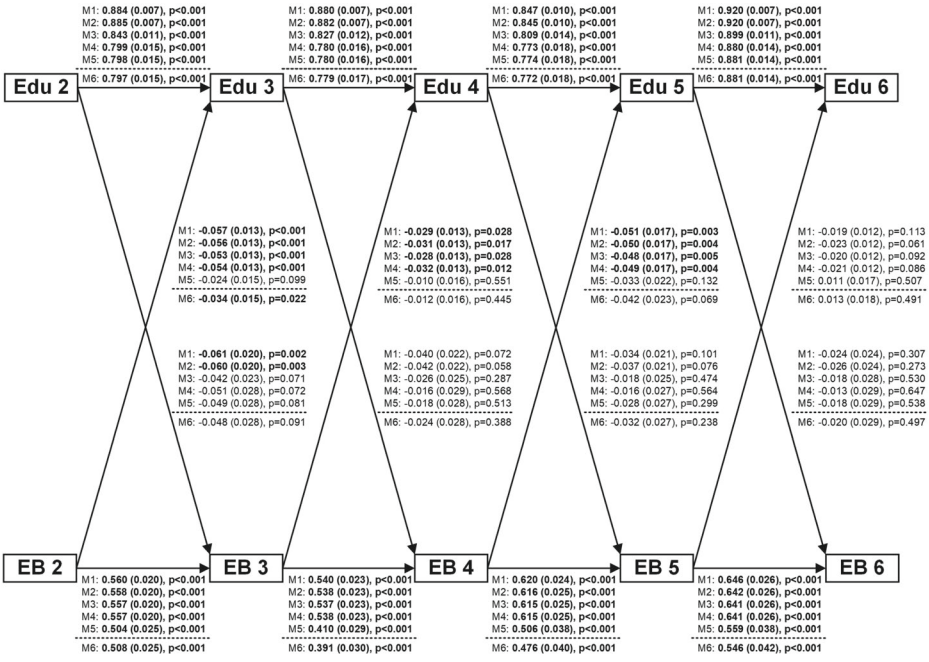
FIGURE 3.S5 Bidirectional associations between educational level and attention problems (AP) in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); sequentially adjusted linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) for different sets of covariates



	Chi-square	df	CFI	TLI	SRMR	RMSEA
Model 1	271.691	24	0.977	0.957	0.064	0.068
Model 2	272.372	24	0.980	0.901	0.042	0.068
Model 3	256.292	24	0.982	0.900	0.039	0.066
Model 4	233.468	24	0.985	0.906	0.036	0.063
Model 5	457.851	54	0.976	0.900	0.040	0.058
Model 6	632.364	96	0.977	0.915	0.046	0.050

Model 1: bivariate cross-lagged panel model. Model 2: cross-lagged panel model adjusted for demographics (age, gender, area of residence, and ethnicity) at baseline (wave 1). Model 3: cross-lagged panel model adjusted for demographics and parental SES at baseline (wave 1). Model 4: cross-lagged panel model adjusted for demographics, parental SES, and IQ at baseline (wave 1). Model 5: cross-lagged panel model adjusted for demographics, parental SES, and IQ at baseline (wave 1), and externalizing behaviour at each preceding wave. Model 6: cross-lagged panel model adjusted for demographics, parental SES, and IQ at baseline (wave 1), and externalizing behaviour and depression/anxiety at each preceding wave. Edu = educational level; AP = attention problems. ***Boldface*** *denotes statistical significance at p < 0.05.*

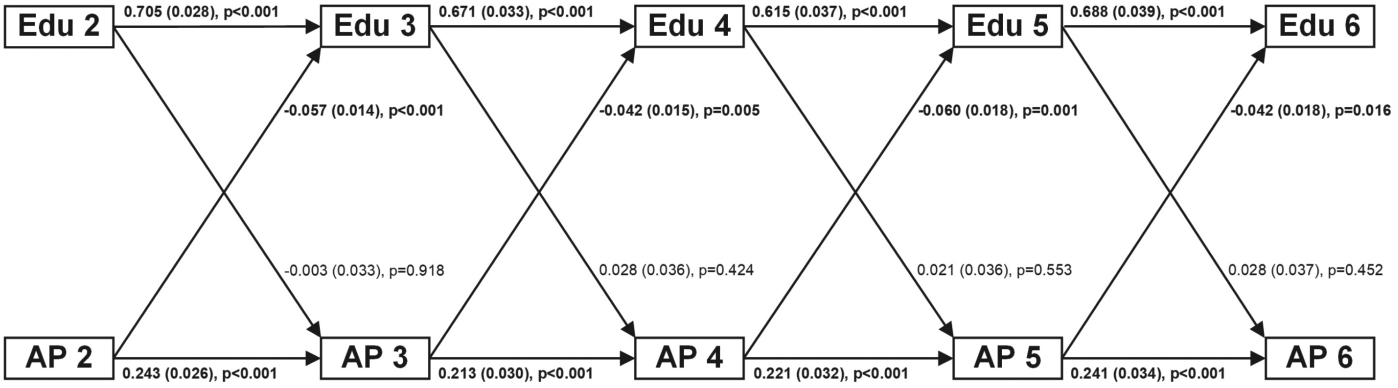
FIGURE 3.S6 Bidirectional associations between educational level and externalizing behaviour (EB) in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); sequentially adjusted linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) for different sets of covariates



	Chi-square	df	CFI	TLI	SRMR	RMSEA
Model 1	342.449	24	0.964	0.933	0.079	0.077
Model 2	337.659	24	0.973	0.866	0.050	0.077
Model 3	322.577	24	0.976	0.862	0.047	0.075
Model 4	305.299	24	0.979	0.866	0.045	0.073
Model 5	457.851	54	0.976	0.900	0.040	0.058
Model 6	632.364	96	0.977	0.915	0.046	0.050

Model 1: bivariate cross-lagged panel model. Model 2: cross-lagged panel model adjusted for demographics (age, gender, area of residence, and ethnicity) at baseline (wave 1). Model 3: cross-lagged panel model adjusted for demographics and parental SES at baseline (wave 1). Model 4: cross-lagged panel model adjusted for demographics, parental SES, and IQ at baseline (wave 1). Model 5: cross-lagged panel model adjusted for demographics, parental SES, and IQ at baseline (wave 1), and attention problems at each preceding wave. Model 6: cross-lagged panel model adjusted for demographics, parental SES, and IQ at baseline (wave 1), and attention problems and depression/anxiety at each preceding wave. Edu = educational level; EB = externalizing behaviour. ***Boldface*** *denotes statistical significance at p < 0.05.*

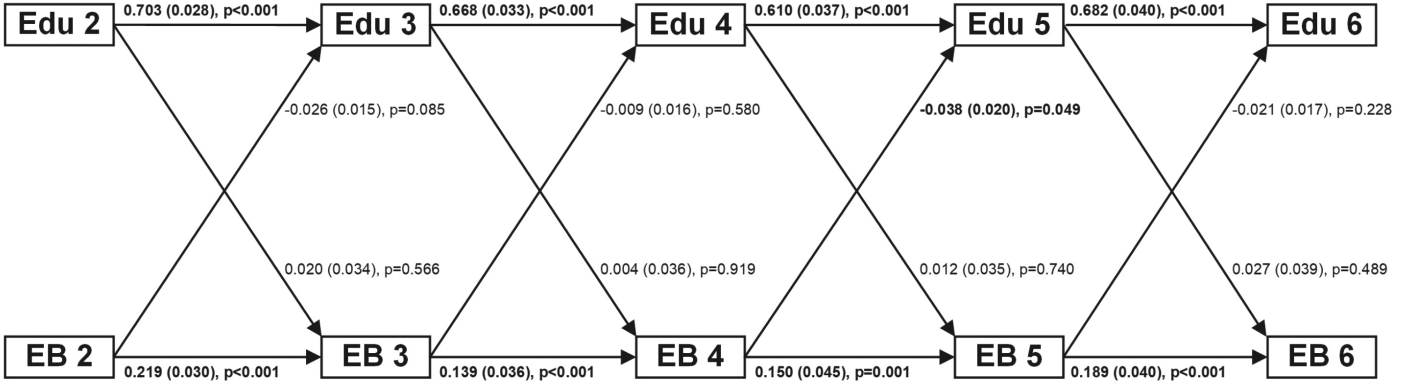
FIGURE 3.S7 Bidirectional associations between educational level and attention problems (AP), using amended scales, in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) from cross-lagged panel models with fixed effects



	Chi-square	df	CFI	TLI	SRMR	RMSEA
a) Education → AP	38.225	9	0.997	0.987	0.019	0.038
b) AP → Education	20.608	9	0.999	0.995	0.005	0.024

Adjustment for time-invariant characteristics was performed by inclusion of a latent variable. Edu = educational level; AP = attention problems. ***Boldface*** *denotes statistical significance at p < 0.05.*

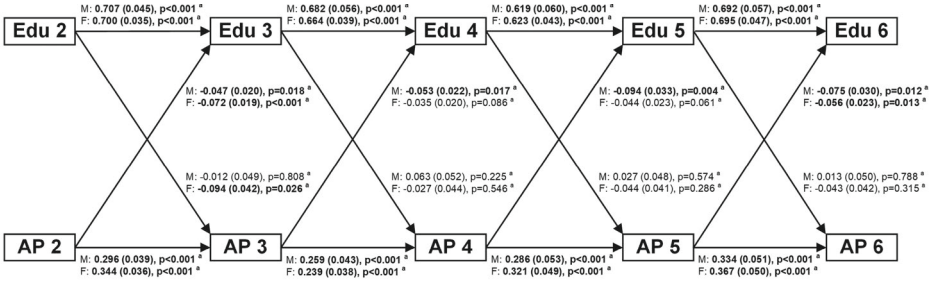
FIGURE 3.S8 Bidirectional associations between educational level and externalizing behaviour (EB), using amended scales, in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) from cross-lagged panel models with fixed effects



	Chi-square	df	CFI	TLI	SRMR	RMSEA
a) Education → EB	35.122	9	0.996	0.985	0.020	0.036
b) EB → Education	28.983	9	0.998	0.991	0.007	0.032

Adjustment for time-invariant characteristics was performed by inclusion of a latent variable. Edu = educational level; EB = externalizing behaviour. ***Boldface*** *denotes statistical significance at p < 0.05.*

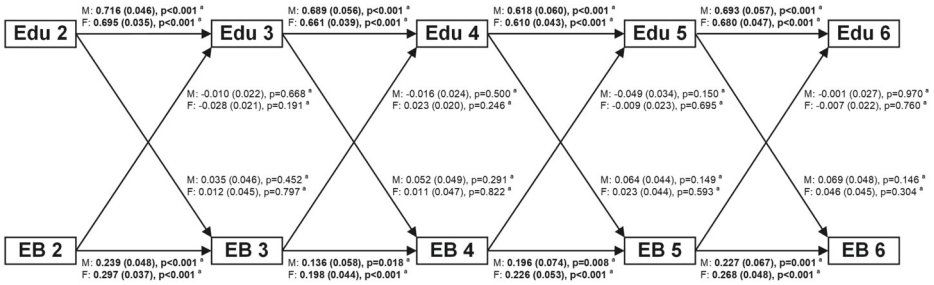
FIGURE 3.S9 Bidirectional associations between educational level and attention problems (AP) in males and females, in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) from cross-lagged panel models with fixed effects



	Chi-square	df	Scaling correction factor	CFI	TLI	SRMR	RMSEA
Unconstrained models							
a) Education → AP	64.246	21	1.0462	0.995	0.984	0.024	0.043
b) AP → Education	39.765	21	1.1312	0.998	0.994	0.015	0.028
Constrained models							
a) Education → AP	67.735	28	1.0363	0.996	0.989	0.024	0.036
b) AP → Education	49.374	28	1.1072	0.998	0.995	0.017	0.026
Model comparison	**Difference Test Scaling Correction (CD)**		**Sattora-Bentler Scaled Chi-Square Difference (TRd)**		**Difference is Degrees of Freedom (Δdf)**		**P-value for TRd, Δdf**
a) Education → AP	1.0066		2.9601		7		0.8887
b) AP → Education	1.0352		9.3554		7		0.2281

Adjustment for time-invariant characteristics was performed by inclusion of a latent variable. Edu = educational level; AP = attention problems; M = male; F = Female. ***Boldface*** *denotes statistical significance at p < 0.05. Parameters with different superscripts differ significantly from each other at p < 0.05, as determined by Wald Tests of Parameter Constraints. Potential gender differences in cross-lagged associations were investigated both by assessing gender differences in individual paths, using the 'Model Test' command in Mplus, as well as by comparing the fit of a model in which cross-lagged paths were constrained to be equal across genders to one allowing for gender differences in cross-lagged paths (van Lier et al., 2012). To test differences in fit between the constrained and unconstrained model, we applied the chi-square difference test using the Satorra-Bentler scaled chi-square (Muthén & Muthén, 2021) .*

FIGURE 3.S10 Bidirectional associations between educational level and externalizing behaviour (EB) in males and females, in the TRAILS study (the Netherlands, 2000–2017, N = 2,229); linear regression coefficients (stdyx-standardized ß-coefficient, robust standard error, p-value) from cross-lagged panel models with fixed effects



	Chi-square	df	Scaling correction factor	CFI	TLI	SRMR	RMSEA
Unconstrained models							
a) Education → EB	64.204	21	1.2193	0.994	0.980	0.027	0.043
b) EB → Education	55.662	21	1.0942	0.996	0.987	0.016	0.038
Constrained models							
a) Education → EB	69.200	28	1.1702	0.994	0.986	0.028	0.036
b) EB → Education	62.113	28	1.0883	0.996	0.991	0.018	0.033
Model comparison	**Difference Test Scaling Correction (CD)**		**Sattora-Bentler Scaled Chi-Square Difference (TRd)**		**Difference is Degrees of Freedom (Δdf)**		**P-value for TRd, Δdf**
a) Education → EB	1.0229		2.6336		7		0.9167
b) EB → Education	1.0706		6.2509		7		0.5108

Adjustment for time-invariant characteristics was performed by inclusion of a latent variable. Edu = educational level; EB = externalizing behaviour; M = male; F = Female. ***Boldface*** *denotes statistical significance at p < 0.05. Parameters with different superscripts differ significantly from each other at p < 0.05, as determined by Wald Tests of Parameter Constraints. Potential gender differences in cross-lagged associations were investigated both by assessing gender differences in individual paths, using the 'Model Test' command in Mplus, as well as by comparing the fit of a model in which cross-lagged paths were constrained to be equal across genders to one allowing for gender differences in cross-lagged paths (van Lier et al., 2012). To test differences in fit between the constrained and unconstrained model, we applied the chi-square difference test using the Satorra-Bentler scaled chi-square (Muthén & Muthén, 2021).*

TABLE 3.S1 Attrition analysis – characteristics of young adults remaining in the TRAILS study (the Netherlands, 2000–2017, N = 2,229) at wave 6, compared to participants who had dropped out of the cohort between wave 2 and wave 5

	Participants remaining in TRAILS by wave 6		**Drop-outs**		**P-value**
N participants (%)	1,616	(72.50)	613	(27.50)	
Baseline characteristics					
Male gender, N (%)	735	(45.48)	363	(59.22)	<0.001
Non-Dutch ethnicity, N (%)	155	(9.59)	146	(23.82)	<0.001
Age, mean (SD)	11.09	(0.56)	11.16	(0.54)	0.006
Parental socioeconomic status (SES), mean (SD)	0.10	(0.76)	-0.44	(0.77)	<0.001
Wechsler Intelligence Deviation Quotient (IQ), mean (SD)	99.64	(14.54)	90.67	(14.24)	<0.001
Educational level, mean (SD)					
Wave 2	2.53	(1.14)	1.72	(0.97)	<0.001
Wave 3	2.64	(1.10)	1.95	(1.06)	<0.001
Wave 4	2.73	(0.95)	2.39	(1.02)	<0.001
Wave 5	2.83	(0.90)	2.29	(1.01)	<0.001
Attention problems, mean (SD)					
Wave 2	0.56	(0.34)	0.56	(0.33)	0.864
Wave 3	0.59	(0.35)	0.62	(0.33)	0.227
Wave 4	0.45	(0.32)	0.49	(0.34)	0.088
Wave 5	0.43	(0.32)	0.45	(0.32)	0.639
Externalizing behaviour, mean (SD)					
Wave 2	0.30	(0.20)	0.32	(0.23)	0.106
Wave 3	0.31	(0.21)	0.37	(0.22)	<0.001
Wave 4	0.22	(0.21)	0.26	(0.23)	0.010
Wave 5	0.19	(0.18)	0.20	(0.17)	0.735

SD = standard deviation.
P-values were computed using chi-squared tests for categorical variables and two-sample t-tests for continuous variables.

TABLE 3.S2 Characteristics of participants with classifiable educational level compared to those with missing/unclassifiable educational level from wave 2 to 6 in the TRAILS study (the Netherlands, 2000–2017, N = 2,229)

	N participants per wave		**Male gender**		**Non-Dutch ethnicity**	
	N	(%)	N	(%)	N	(%)
Wave 2	2,148	(100)				
Education complete	1,927	(89.71)	924	(47.95)	237	(12.30)
Education unclassifiable/missing	221	(10.29)	130	(58.82)	35	(15.84)
P-value				0.002		0.134
Wave 3	1,818	(100)				
Education complete	1,529	(84.10)	704	(46.04)	175	(11.45)
Education unclassifiable/missing	289	(15.90)	163	(56.40)	36	(12.46)
P-value				0.001		0.623
Wave 4	1,880	(100)				
Education complete	1,507	(80.16)	671	(44.53)	160	(10.62)
Education unclassifiable/missing	373	(19.84)	227	(60.86)	52	(13.94)
P-value				<0.001		0.069
Wave 5	1,781	(100)				
Education complete	1,429	(80.24)	624	(43.67)	147	(10.29)
Education unclassifiable/missing	352	(19.76)	219	(62.22)	52	(14.77)
P-value				<0.001		0.017
Wave 6	1,616	(100)				
Education complete	1,192	(73.76)	474	(39.77)	106	(8.89)
Education unclassifiable/missing	424	(26.24)	261	(61.56)	49	(11.56)
P-value				<0.001		0.110

SD = standard deviation.
P-values were computed using chi-squared tests for categorical variables and 2-sample t-tests for continuous variables.

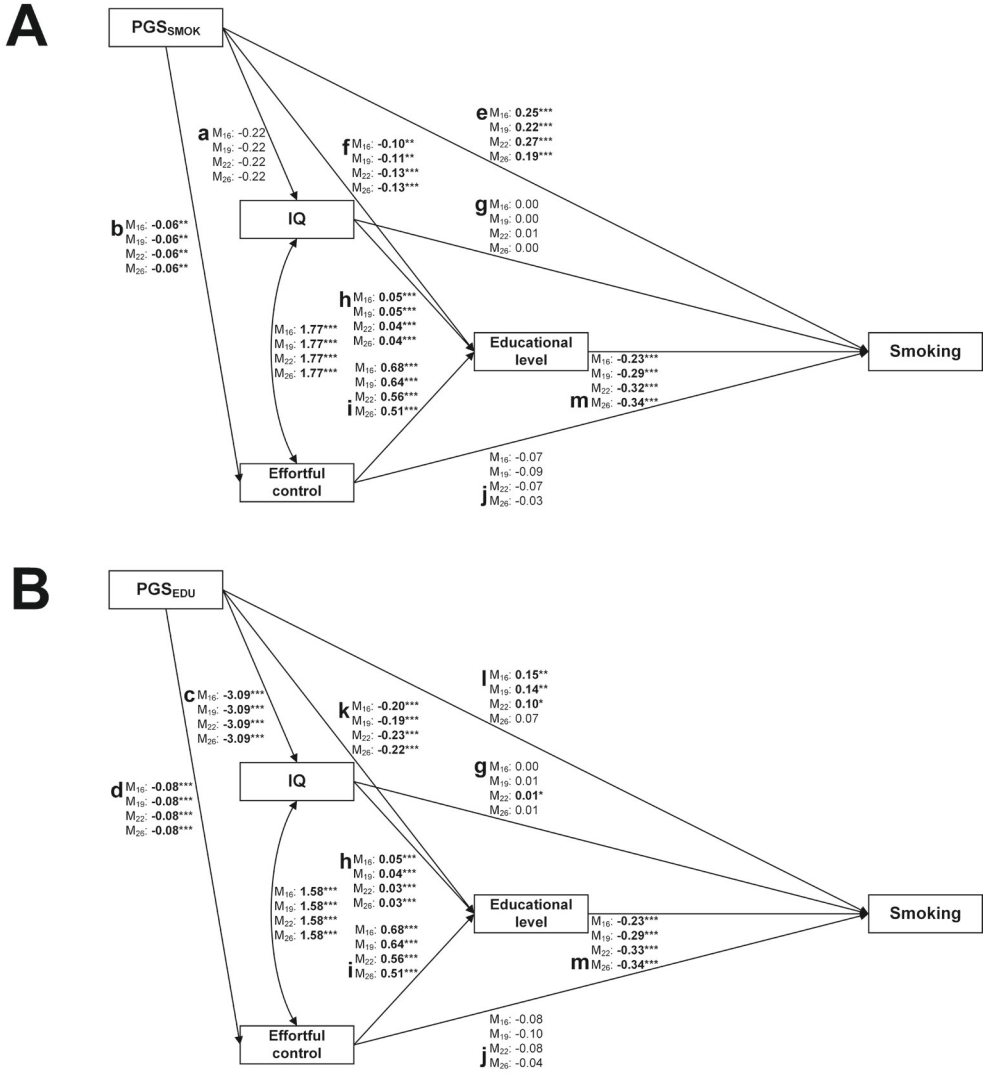
Age at baseline		Parental socioeconomic status (SES)		Wechsler intelligence Deviation Quotient (IQ)		Concurrent attention problems		Concurrent externalizing behaviour	
Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
11.11	(0.56)	-0.03	(0.80)	97.58	(15.02)	0.57	(0.34)	0.31	(0.20)
11.07	(0.53)	-0.07	(0.75)	96.01	(13.44)	0.51	(0.34)	0.30	(0.23)
	0.344		0.531		0.138		0.035		0.874
11.09	(0.56)	0.08	(0.78)	99.75	(14.85)	0.60	(0.34)	0.32	(0.21)
11.19	(0.57)	-0.32	(0.78)	91.25	(13.22)	0.57	(0.36)	0.33	(0.22)
	0.004		<0.001		<0.001		0.323		0.489
11.09	(0.57)	0.13	(0.76)	100.34	(14.52)	0.46	(0.32)	0.23	(0.21)
11.10	(0.52)	-0.33	(0.74)	91.58	(13.13)	0.41	(0.34)	0.23	(0.23)
	0.693		<0.001		<0.001		0.038		0.786
11.10	(0.56)	0.12	(0.76)	100.13	(14.68)	0.43	(0.32)	0.19	(0.18)
11.11	(0.55)	-0.23	(0.76)	93.78	(13.88)	0.46	(0.35)	0.25	(0.20)
	0.608		<0.001		<0.001		0.310		<0.001
11.09	(0.56)	0.17	(0.75)	100.82	(14.47)	0.44	(0.33)	0.19	(0.18)
11.10	(0.56)	-0.09	(0.76)	96.31	(14.22)	0.51	(0.39)	0.27	(0.24)
	0.630		<0.001		<0.001		0.010		<0.001

TABLE 3.S3 Amended scales with consistent items across YSR/ASR

YSR	ASR
Attention problems	
I fail to finish things that I start	I fail to finish things I should do
I have trouble sitting still	I have trouble sitting still
I have trouble concentrating or paying attention	I have trouble concentrating or paying attention for long
I feel confused or in a fog	I feel confused or in a fog
I daydream a lot	I daydream a lot
I act without stopping to think	I am impulsive or act without thinking
I'm too dependent on adults	I am too dependent on others
Externalizing behaviour	
I argue a lot	I argue a lot
I am mean to others	I am mean to others
I try to get a lot of attention	I try to get a lot of attention
I destroy my own things	I damage or destroy my things
I destroy things belonging to others	I damage or destroy things belonging to others
I get in many fights	I get in many fights
I physically attack people	I physically attack people
I scream a lot	I scream or yell a lot
I am stubborn	I am stubborn, sullen, or irritable
My moods or feelings change suddenly	My moods or feelings change suddenly
I tease others a lot	I tease others a lot
I have a hot temper	I have a hot temper
I threaten to hurt people	I threaten to hurt people
I am louder than other kids	I am louder than others
I don't feel guilty after doing something I shouldn't	I don't feel guilty after doing something I shouldn't
I break rules at home, school, or elsewhere	I break rules at work or elsewhere
I hang around with kids who get in trouble	I hang around people who get in trouble
I lie or cheat	I lie or cheat
I would rather be with older kids than kids my own age	I would rather be with older people than with people of my own age
Mean score of "I steal at home" and "I steal from places other than home"	I steal
I cut classes or skip school	I stay away from my job even when I'm not sick or not on vacation
I brag	I brag
I show off or clown	I show off or clown
I talk too much	I talk too much

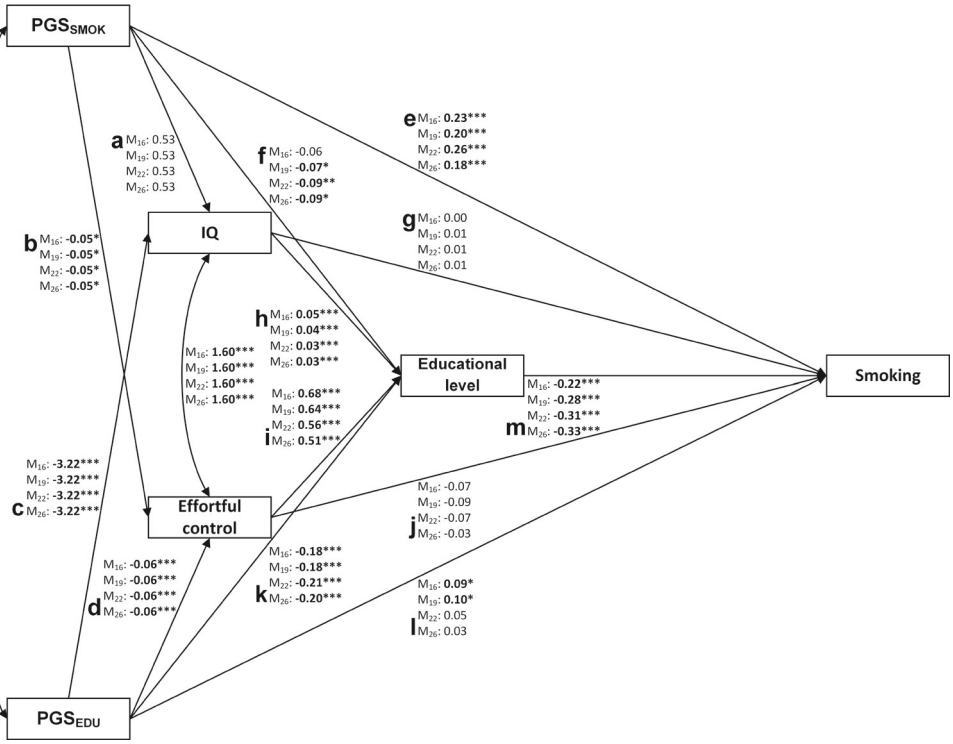
SUPPLEMENTARY MATERIALS – CHAPTER 4

FIGURE 4.S1 The associations of PGS_{SMOK} (A) and PGS_{EDU} (B) with daily smoking (yes/no) around age 16, 19, 22, and 26 in separate models; potential mediators were IQ and effortful control measured around age 11 and educational level (ordinal) measured concurrently with smoking; linear and probit regression models (WLSMV estimator; beta-coefficient)



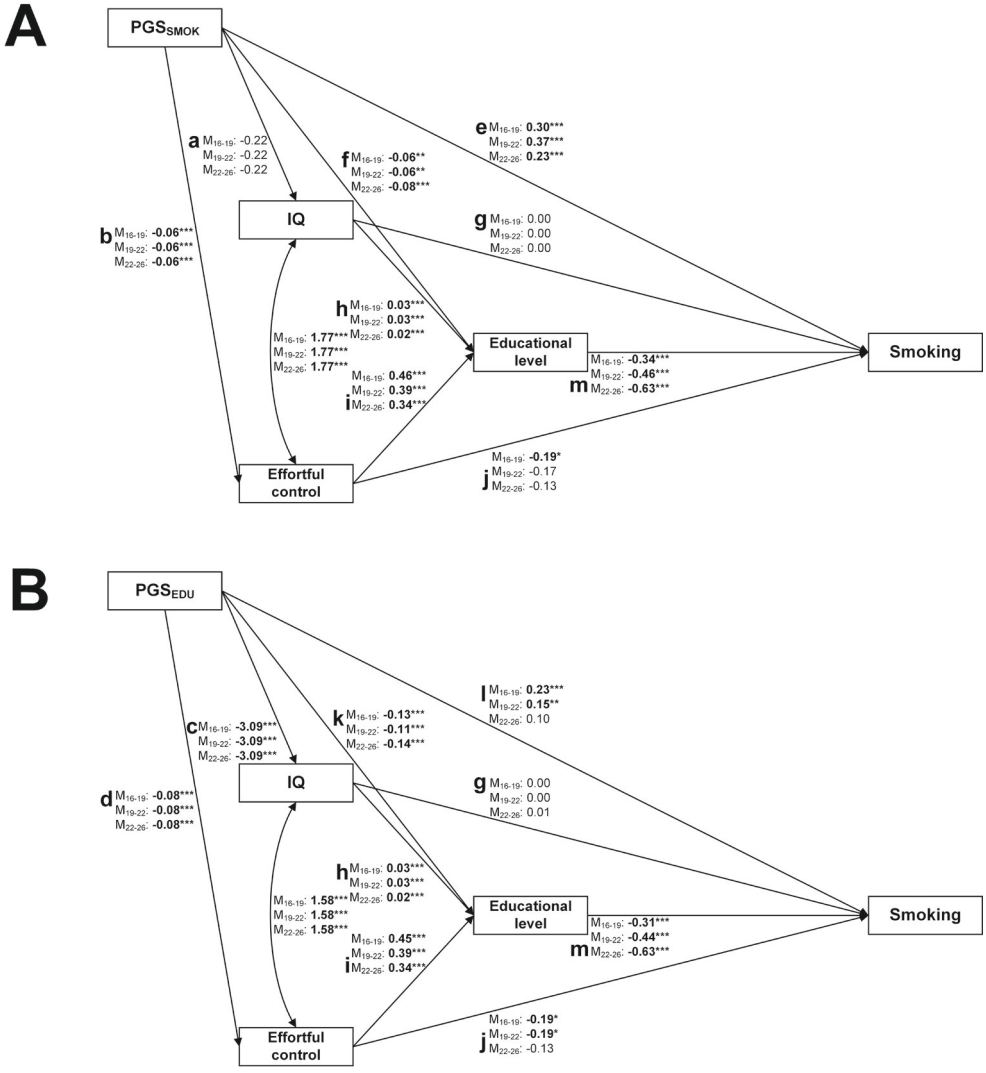
** p<0.05; **p<0.01; ***p<0.001. All regressions were adjusted for age, sex, cohort type, parental education, and parental smoking. Separate models were used to predict smoking around age 16 (M_{16}), 19 (M_{19}), 22 (M_{22}), and 26 (M_{26}). Educational level was measured concurrently with smoking.*

FIGURE 4.S2 The associations of PGS_{EDU} and PGS_{SMOK} with daily smoking (yes/no) around age 16, 19, 22, and 26 in models combining both PGSs; potential mediators were IQ and effortful control measured around age 11 and educational level (ordinal) measured concurrently with smoking; linear and probit regression models (WLSMV estimator; beta-coefficient)



** p<0.05; **p<0.01; ***p<0.001. All regressions were adjusted for age, sex, cohort type, parental education, and parental smoking. Separate models were used to predict smoking around age 16 (M_{16}), 19 (M_{19}), 22 (M_{22}), and 26 (M_{26}). Educational level was measured concurrently with smoking.*

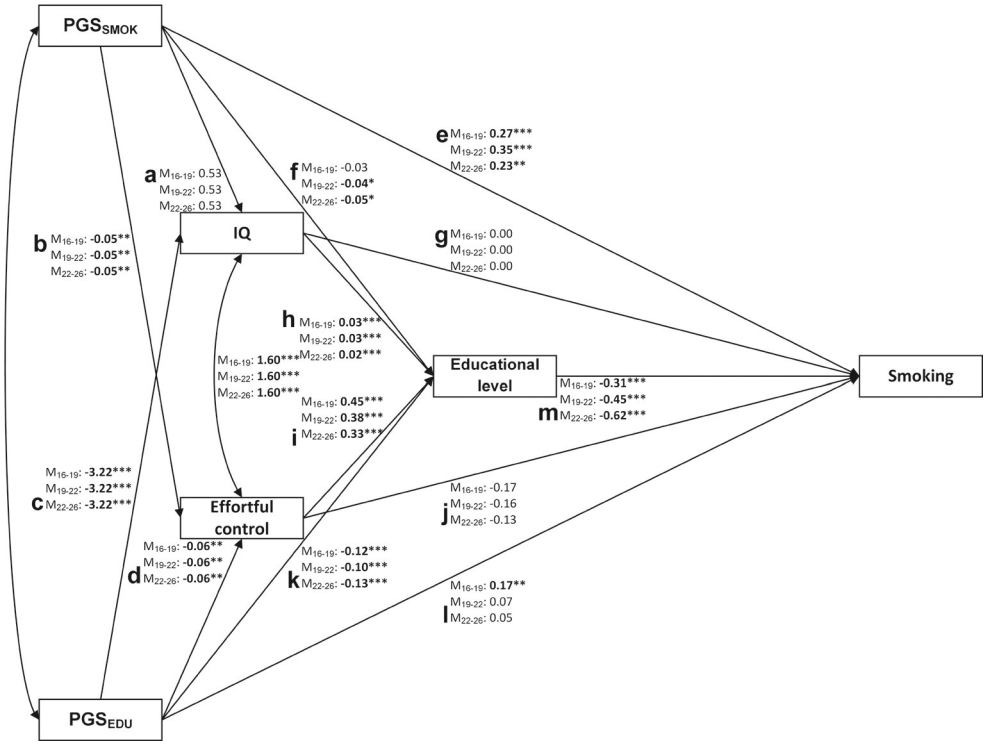
FIGURE 4.S3 The associations of PGS_{SMOK} (A) and PGS_{EDU} (B) with smoking around age 19, 22, and 26 in separate models; potential mediators were IQ and effortful control, measured around age 11, and educational level, assessed one wave before smoking, i.e., around age 16, 19, and 22; linear and negative binomial regression models (MLR estimator; beta-coefficient)



A

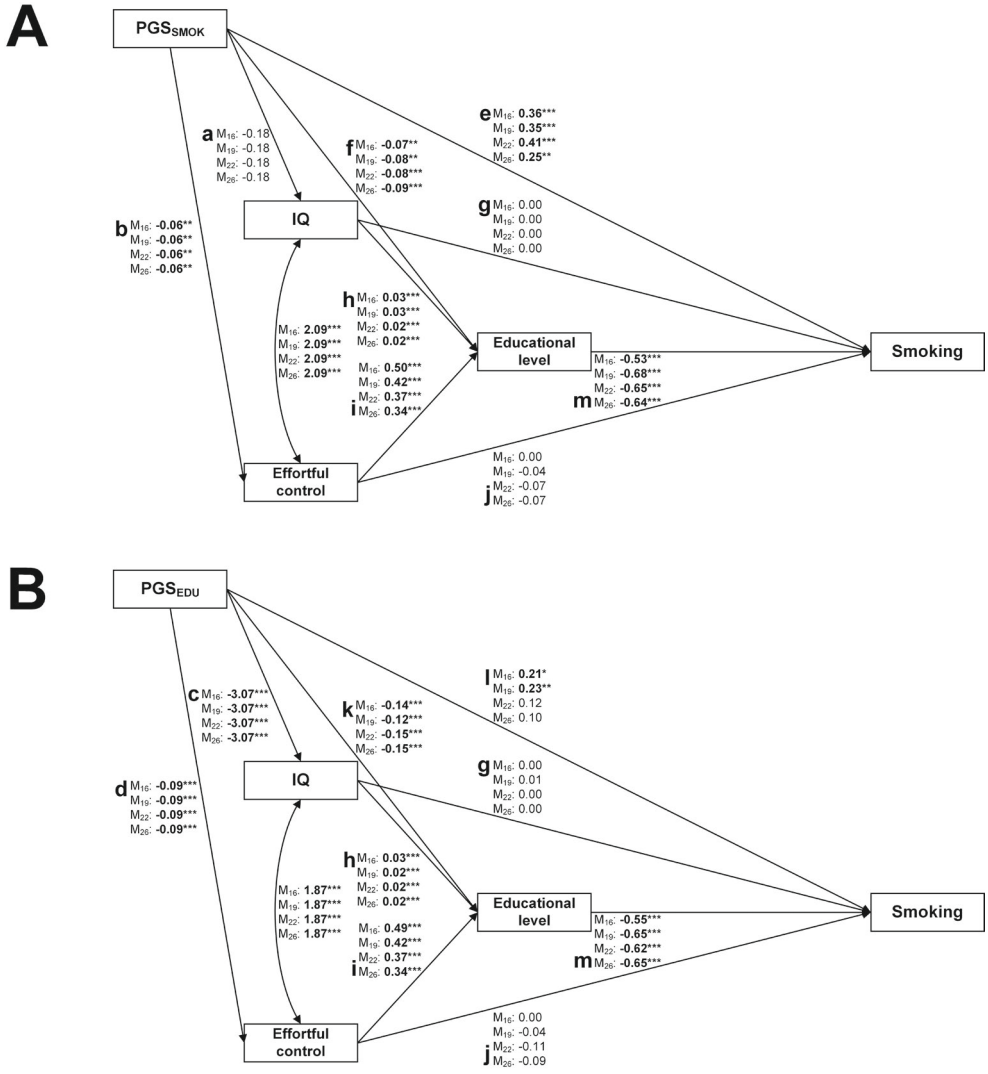
** p<0.05; **p<0.01; ***p<0.001. All regressions were adjusted for age, sex, cohort type, parental education, and parental smoking. Separate models were used to predict educational level around age 16 and smoking around age 19 ($M_{16\text{-}19}$), educational level around age 19 and smoking around age 22 ($M_{19\text{-}22}$), and, respectively, educational level around age 22 and smoking around age 26 ($M_{22\text{-}26}$).*

FIGURE 4.S4 The associations of PGS_{EDU} and PGS_{SMOK} with smoking around age 19, 22, and 26 in models combining both PGSs; potential mediators were IQ and effortful control, measured around age 11, and educational level, assessed one wave before smoking, i.e., around age 16, 19, and 22; linear and negative binomial regression models (MLR estimator; beta-coefficient)



** p<0.05; **p<0.01; ***p<0.001. All regressions were adjusted for age, sex, cohort type, parental education, and parental smoking. Separate models were used to predict educational level around age 16 and smoking around age 19 ($M_{16\text{-}19}$), educational level around age 19 and smoking around age 22 ($M_{19\text{-}22}$), and, respectively, educational level around age 22 and smoking around age 26 ($M_{22\text{-}26}$).*

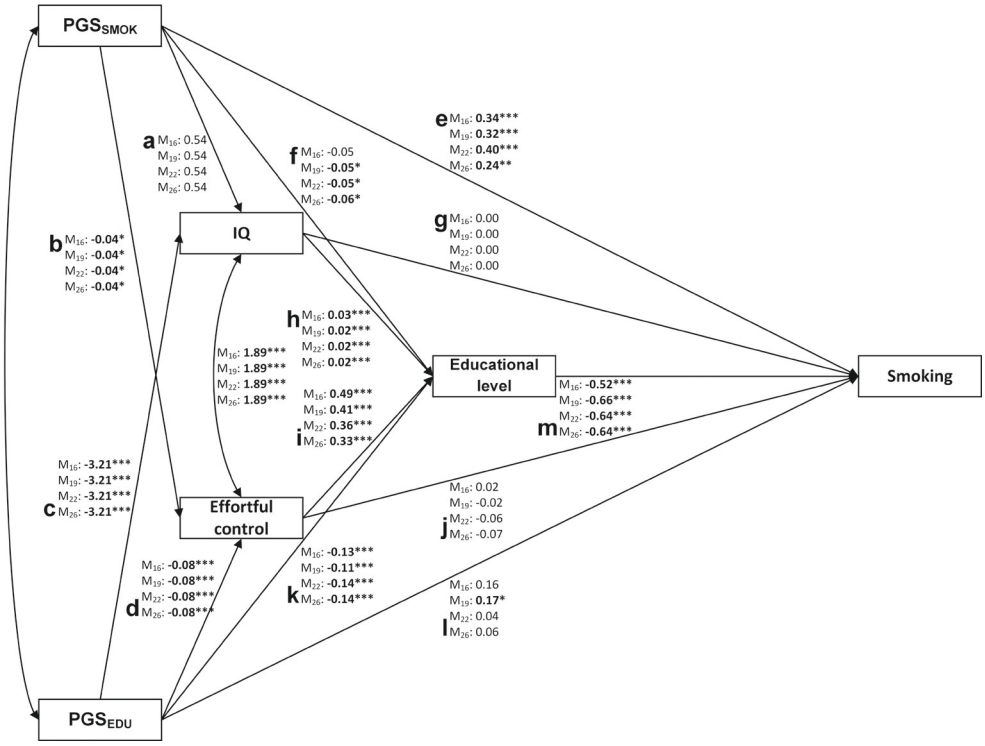
FIGURE 4.S5 The associations of PGS_{SMOK} (A) and PGS_{EDU} (B) with smoking around age 16, 19, 22, and 26 in separate models in the TRAILS general population cohort (N = 1,248); potential mediators were IQ and effortful control (around age 11) and educational level measured concurrently with smoking; linear and negative binomial regression models (MLR estimator; beta-coefficient)



A

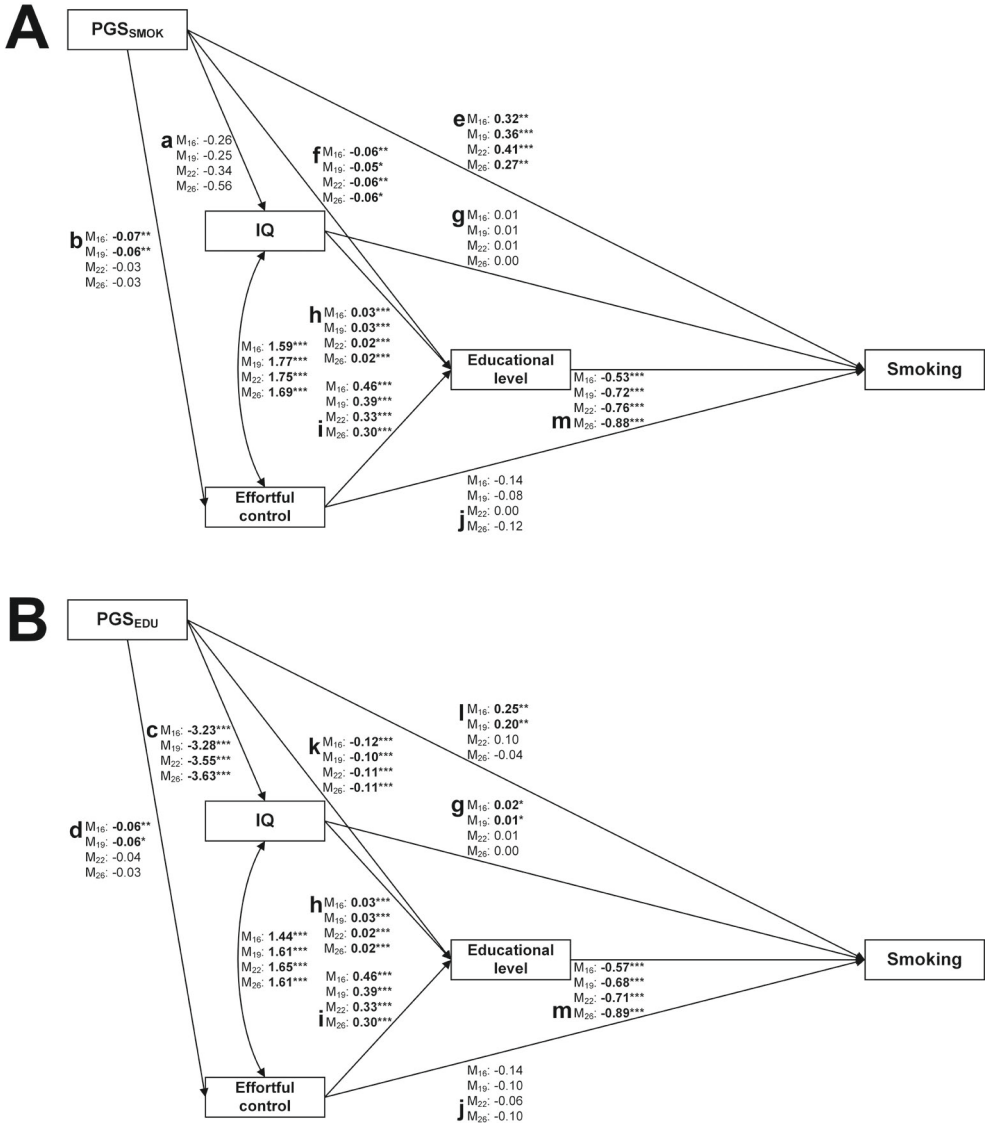
** p<0.05; **p<0.01; ***p<0.001. All regressions were adjusted for age, sex, cohort type, parental education, and parental smoking. Separate models were used to predict smoking around age 16 (M_{16}), 19 (M_{19}), 22 (M_{22}), and 26 (M_{26}). Educational level was measured concurrently with smoking.*

FIGURE 4.S6 The associations of PGS_{EDU} and PGS_{SMOK} with smoking around age 16, 19, 22, and 26 in models combining both PGSs in the TRAILS general population cohort (N = 1,248); potential mediators were IQ and effortful control (around age 11) and educational level measured concurrently with smoking; linear and negative binomial regression models (MLR estimator; beta-coefficient)



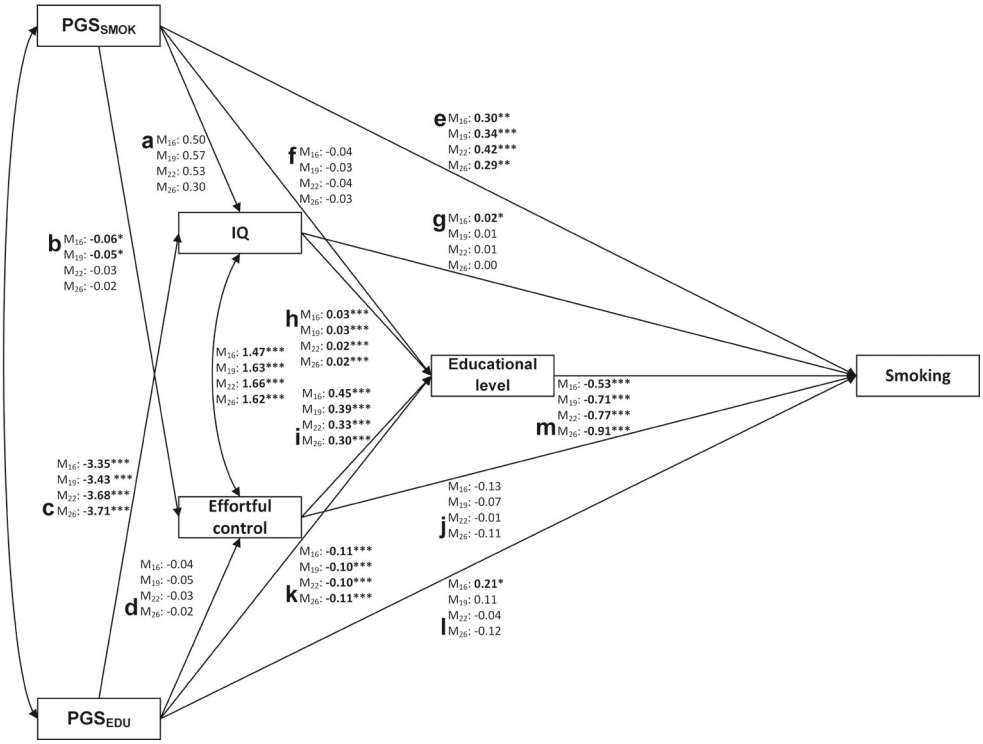
** p<0.05; **p<0.01; ***p<0.001. All regressions were adjusted for age, sex, parental education, and parental smoking. Separate models were used to predict smoking around age 16 (M_{16}), 19 (M_{19}), 22 (M_{22}), and 26 (M_{26}). Educational level was measured concurrently with smoking.*

FIGURE 4.S7 The associations of PGS_{SMOK} (A) and PGS_{EDU} (B) with smoking around age 16, 19, 22, and 26 in separate models in complete case analyses; potential mediators were IQ and effortful control (around age 11) and educational level measured concurrently with smoking; linear and negative binomial regression models (MLR estimator; beta-coefficient)



** p<0.05; **p<0.01; ***p<0.001. All regressions were adjusted for age, sex, cohort type, parental education, and parental smoking. Separate models were used to predict smoking around age 16 (M_{16}), 19 (M_{19}), 22 (M_{22}), and 26 (M_{26}). Educational level was measured concurrently with smoking. The sample sizes of these complete case analyses are as follows: for A/B-M_{16} N = 1,107, for A/B-M_{19} N = 1,032, for A/B-M_{22} N = 970, and for A/B-M_{26} N = 838.*

FIGURE 4.S8 The associations of PGS_{EDU} and PGS_{SMOK} with smoking around age 16, 19, 22, and 26 in models combining both PGSs in complete case analyses; potential mediators were IQ and effortful control (around age 11) and educational level measured concurrently with smoking; linear and negative binomial regression models (MLR estimator; beta-coefficient)



** p<0.05; **p<0.01; ***p<0.001. All regressions were adjusted for age, sex, cohort type, parental education, and parental smoking. Separate models were used to predict smoking around age 16 (M_{16}), 19 (M_{19}), 22 (M_{22}), and 26 (M_{26}). Educational level was measured concurrently with smoking. The sample sizes of these complete case analyses are as follows: for M_{16} N = 1,107, for M_{19} N = 1,032, for M_{22} N = 970, and for M_{26} N = 838.*

TABLE 4.S1 Attrition analysis – characteristics of young adults remaining in the TRAILS study at wave 6, compared to participants who had dropped out of the cohort between wave 1 and wave 6

	Participants remaining in TRAILS by wave 6		**Drop-outs**		**P-value**
N participants (%)	1,371	(86.72)	210	(13.28)	
PGS_{SMOK}, mean (SD)	-0.04	(0.99)	0.24	(1.04)	<0.001
PGS_{EDU}, mean (SD)	-0.06	(0.99)	0.38	(0.98)	<0.001
Wechsler Intelligence Deviation Quotient (IQ), mean (SD)	100.67	(14.38)	93.43	(15.09)	<0.001
Effortful control, mean (SD)	3.15	(0.72)	2.85	(0.72)	<0.001
Male sex, N (%)	690	(50.33)	131	(62.38)	0.001
Age at baseline, mean (SD)	11.08	(0.55)	11.09	(0.51)	0.854
Parental education, mean (SD)	3.21	(0.88)	2.76	(0.83)	<0.001
Parental smoking, mean (SD)	0.61	(0.76)	0.86	(0.80)	<0.001
Clinical cohort, N (%)	280	(20.42)	53	(25.24)	0.111
Educational level, mean (SD)					
Wave 3	2.56	(1.11)	1.89	(0.99)	<0.001
Wave 4	2.66	(0.98)	2.26	(0.94)	0.001
Wave 5	2.78	(0.93)	2.36	(0.92)	0.003
Smoking, mean (SD)					
Wave 3	2.11	(4.93)	3.68	(6.26)	<0.001
Wave 4	3.24	(6.37)	5.63	(8.11)	<0.001
Wave 5	3.44	(6.58)	6.73	(8.18)	<0.001

SD = standard deviation.
P-values were computed using chi-squared tests for categorical variables and two-sample t-tests for continuous variables.

TABLE 4.S2 Characteristics of participants with classifiable educational level compared to those with missing/ unclassifiable educational level from wave 2 to 4 in the TRAILS study

	N participants per wave		**PGS_{SMOK}**		**PGS_{EDU}**		**Wechsler Intelligence Deviation Quotient (IQ)**	
	N	(%)	Mean	(SD)	Mean	(SD)	Mean	(SD)
Wave 3	1,547	(100.00)						
Education complete	1,341	(86.68)	-0.03	(0.99)	-0.06	(1.00)	100.99	(14.55)
Education unclassifiable/missing	206	(13.32)	0.18	(1.01)	0.32	(0.92)	92.49	(13.13)
P-value			0.006		<0.001		<0.001	
Wave 4	1,498	(100.00)						
Education complete	1,276	(85.18)	-0.07	(0.99)	-0.10	(0.99)	101.46	(14.40)
Education unclassifiable/missing	222	(14.82)	0.30	(0.96)	0.34	(0.96)	92.53	(13.22)
P-value			<0.001		<0.001		<0.001	
Wave 5	1,455	(100.00)						
Education complete	1,215	(83.51)	-0.06	(1.00)	-0.10	(1.00)	101.51	(14.41)
Education unclassifiable/missing	240	(16.49)	0.12	(0.88)	0.28	(0.93)	94.28	(13.34)
P-value			0.008		<0.001		<0.001	
Wave 6	1,371	(100.00)						
Education complete	1,029	(75.05)	-0.10	(1.00)	-0.13	(0.99)	101.78	(14.33)
Education unclassifiable/missing	342	(24.95)	0.15	(0.95)	0.17	(0.96)	97.31	(14.02)
P-value			<0.001		<0.001		<0.001	

SD = standard deviation.
P-values were computed using chi-squared tests for categorical variables and 2-sample t-tests for continuous variables.

Effortful control		Male sex		Age at baseline		Parental education		Parental smoking		Clinical cohort		Concurrent smoking	
Mean	(SD)	N	(%)	Mean	(SD)	Mean	(SD)	Mean	(SD)	N	(%)	Mean	(SD)
3.17	(0.72)	676	(50.41)	11.08	(0.54)	3.21	(0.89)	0.61	(0.76)	235	(17.52)	2.10	(4.91)
2.76	(0.67)	122	(59.22)	11.11	(0.54)	2.76	(0.73)	0.84	(0.81)	71	(34.47)	3.69	(6.33)
<0.001		0.018		0.344		<0.001		<0.001		<0.001		<0.001	
3.19	(0.72)	638	(50.00)	11.08	(0.55)	3.25	(0.87)	0.60	(0.76)	239	(18.73)	2.96	(6.06)
2.80	(0.68)	132	(59.46)	11.06	(0.49)	2.68	(0.76)	0.85	(0.81)	64	(28.83)	6.98	(8.75)
<0.001		0.009		0.562		<0.001		<0.001		0.001		<0.001	
3.20	(0.72)	598	(49.22)	11.09	(0.55)	3.25	(0.88)	0.60	(0.76)	224	(18.44)	3.18	(6.24)
2.82	(0.67)	150	(62.50)	11.07	(0.52)	2.81	(0.81)	0.76	(0.79)	65	(27.08)	7.65	(9.23)
<0.001		<0.001		0.584		<0.001		0.009		0.002		<0.001	
3.24	(0.71)	462	(44.90)	11.09	(0.55)	3.28	(0.88)	0.57	(0.75)	181	(17.59)	2.17	(4.86)
2.88	(0.68)	228	(66.67)	11.06	(0.53)	2.98	(0.84)	0.73	(0.81)	99	(28.95)	6.29	(7.95)
<0.001		<0.001		0.393		<0.001		0.004		<0.001		<0.001	

TABLE 4.S3 Sequentially adjusted associations between educational level and concurrent smoking around age 16, 19, 22, and 26 in the TRAILS study; negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Association between education and smoking around age 16	**Association between education and smoking around age 19**	**Association between education and smoking around age 22**	**Association between education and smoking around age 26**
Model 1	**-0.596 (0.069), <0.001**	**-0.601 (0.058), <0.001**	**-0.662 (0.058), <0.001**	**-0.666 (0.066), <0.001**
Model 2	**-0.539 (0.077), <0.001**	**-0.632 (0.071), <0.001**	**-0.644 (0.068), <0.001**	**-0.631 (0.078), <0.001**
Model 3	**-0.460 (0.080), <0.001**	**-0.571 (0.075), <0.001**	**-0.610 (0.074), <0.001**	**-0.605 (0.083), <0.001**
Model 4	**-0.482 (0.097), <0.001**	**-0.585 (0.091), <0.001**	**-0.581 (0.085), <0.001**	**-0.590 (0.093), <0.001**

Boldface *denotes statistical significance at p<0.05.*
Model 1 represents bivariate associations between educational level and smoking.
Model 2 is adjusted for gender, age, cohort type, parental education, and parental smoking.
Model 3 is adjusted for gender, age, cohort type, parental education, parental smoking, PGS_{EDU}, and PGS_{SMOK}.
Model 4 is adjusted for gender, age, cohort type, parental education, parental smoking, PGS_{EDU}, PGS_{SMOK}, IQ, and effortful control.

TABLE 4.S4 Total, direct, and indirect effects of PGS_{SMOK} on daily smoking (yes/no) around age 16, 19, 22, and 26 in the TRAILS study; potential mediators were measured around age 11 (IQ and effortful control) and concurrently with smoking (educational level); linear and probit regression (WLSMV estimator; beta-coefficient, standard error, p-value)

	Smoking around age 16	**Smoking around age 19**	**Smoking around age 22**	**Smoking around age 26**
Model 1				
Total effect	**0.285 (0.044), <0.001**	**0.266 (0.041), <0.001**	**0.330 (0.044), <0.001**	**0.246 (0.044), <0.001**
Direct effect	**0.248 (0.043), <0.001**	**0.216 (0.040), <0.001**	**0.271 (0.042), <0.001**	**0.188 (0.043), <0.001**
Total indirect effect	**0.038 (0.012), 0.001**	**0.050 (0.014), <0.001**	**0.060 (0.014), <0.001**	**0.058 (0.015), <0.001**
Specific indirect effects				
PGS_{SMOK}→effortful control→smoking	0.004 (0.005), 0.364	0.005 (0.004), 0.215	0.004 (0.004), 0.345	0.002 (0.004), 0.638
PGS_{SMOK}→IQ→smoking	0.000 (0.001), 0.808	-0.001 (0.002), 0.621	-0.001 (0.002), 0.602	-0.001 (0.002), 0.630
PGS_{SMOK}→education→smoking	**0.022 (0.009), 0.019**	**0.031 (0.011), 0.005**	**0.043 (0.012), <0.001**	**0.044 (0.013), 0.001**
PGS_{SMOK}→effortful control→ education→smoking	**0.009 (0.004), 0.007**	**0.011 (0.004), 0.005**	**0.011 (0.004), 0.003**	**0.010 (0.004), 0.005**
PGS_{SMOK}→IQ→education→smoking	0.002 (0.004), 0.552	0.003 (0.005), 0.553	0.003 (0.004), 0.554	0.003 (0.004), 0.555
Model 2				
Total effect	**0.249 (0.044), <0.001**	**0.226 (0.042), <0.001**	**0.299 (0.044), <0.001**	**0.219 (0.045), <0.001**
Direct effect	**0.231 (0.044), <0.001**	**0.199 (0.041), <0.001**	**0.262 (0.043), <0.001**	**0.184 (0.044), <0.001**
Total indirect effect	0.018 **(**0.011**),** 0.091	**0.028 (0.012), 0.025**	**0.036 (0.013), 0.006**	**0.035 (0.014), 0.011**
Specific indirect effects				
PGS_{SMOK}→effortful control→smoking	0.003 (0.004), 0.395	0.004 (0.003), 0.240	0.003 (0.003), 0.363	0.001 (0.003), 0.651
PGS_{SMOK}→IQ →smoking	0.001 (0.002), 0.660	0.003 (0.003), 0.342	0.003 (0.003), 0.300	0.002 (0.003), 0.370
PGS_{SMOK}→education→smoking	0.013 (0.008), 0.129	0.019 (0.010), 0.055	**0.028 (0.011), 0.013**	**0.029 (0.012), 0.017**
PGS_{SMOK}→effortful control→ education→smoking	**0.007 (0.003), 0.031**	**0.008 (0.004), 0.027**	**0.008 (0.003), 0.022**	**0.008 (0.003), 0.026**
PGS_{SMOK}→IQ→education→smoking	-0.005 (0.004), 0.173	-0.006 (0.005), 0.164	-0.006 (0.004), 0.160	-0.006 (0.004), 0.161

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{EDU}.

TABLE 4.S5 Total, direct, and indirect effects of PGS_{EDU} on daily smoking (yes/no) around age 16, 19, 22, and 26 in the TRAILS study; potential mediators were measured around age 11 (IQ and effortful control) and concurrently with smoking (educational level); linear and probit regression (WLSMV estimator; beta-coefficient, standard error, p-value)

	Smoking around age 16	Smoking around age 19	Smoking around age 22	Smoking around age 26
Model 1				
Total effect	**0.230 (0.046), <0.001**	**0.235 (0.042), <0.001**	**0.209 (0.041), <0.001**	**0.176 (0.046), <0.001**
Direct effect	**0.145 (0.046), 0.002**	**0.140 (0.043), 0.001**	**0.102 (0.043), 0.017**	0.068 (0.047), 0.147
Total indirect effect	**0.085 (0.017), <0.001**	**0.095 (0.018), <0.001**	**0.107 (0.019), <0.001**	**0.109 (0.021), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→smoking	0.006 (0.006), 0.265	0.007 (0.005), 0.145	0.006 (0.005), 0.234	0.003 (0.005), 0.524
PGS_{EDU}→IQ→smoking	-0.010 (0.012), 0.389	-0.019 (0.011), 0.096	**-0.021 (0.011), 0.047**	-0.018 (0.012), 0.126
PGS_{EDU}→education→smoking	**0.045 (0.013), <0.001**	**0.055 (0.014), <0.001**	**0.075 (0.016), <0.001**	**0.076 (0.017), <0.001**
PGS_{EDU}→effortful control→education→smoking	**0.012 (0.004), 0.002**	**0.014 (0.004), 0.001**	**0.014 (0.004), 0.001**	**0.013 (0.004), 0.001**
PGS_{EDU}→IQ→education→smoking	**0.032 (0.008), <0.001**	**0.038 (0.008), <0.001**	**0.034 (0.007), <0.001**	**0.034 (0.007), <0.001**
Model 2				
Total effect	**0.174 (0.047), <0.001**	**0.184 (0.043), <0.001**	**0.143 (0.043), 0.001**	**0.126 (0.047), 0.007**
Direct effect	**0.094 (0.047), 0.047**	**0.095 (0.044), 0.031**	0.046 (0.044), 0.303	0.027 (0.048), 0.574
Total indirect effect	**0.080 (0.016), <0.001**	**0.089 (0.018), <0.001**	**0.097 (0.018), <0.001**	**0.099 (0.020), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→smoking	0.004 (0.005), 0.382	0.005 (0.004), 0.219	0.004 (0.004), 0.346	0.002 (0.004), 0.649
PGS_{EDU}→IQ→smoking	-0.006 (0.012), 0.635	-0.015 (0.012), 0.193	-0.017 (0.011), 0.126	-0.015 (0.012), 0.228
PGS_{EDU}→education→smoking	**0.040 (0.012), 0.001**	**0.049 (0.014), <0.001**	**0.065 (0.015), <0.001**	**0.066 (0.017), <0.001**
PGS_{EDU}→effortful control→education→smoking	**0.009 (0.003), 0.006**	**0.011 (0.004), 0.004**	**0.011 (0.004), 0.003**	**0.011 (0.004), 0.004**
PGS_{EDU}→IQ → education→smoking	**0.033 (0.008), <0.001**	**0.038 (0.009), <0.001**	**0.034 (0.007), <0.001**	**0.035 (0.007), <0.001**

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{SMOK}.

TABLE 4.S6 Total, direct, and indirect effects of PGS_{SMOK} on educational level as ordinal variable around age 16, 19, 22, and 26 in the TRAILS study; potential mediators were measured around age 11 (IQ and effortful control); linear and probit regression (WLSMV estimator; beta-coefficient, standard error, p-value)

	Educational level around age 16	**Educational level around age 19**	**Educational level around age 22**	**Educational level around age 26**
Model 1				
Total effect	**-0.147 (0.042), <0.001**	**-0.156 (0.040), <0.001**	**-0.175 (0.038), <0.001**	**-0.171 (0.038), <0.001**
Direct effect	**-0.096 (0.034), 0.005**	**-0.107 (0.034), 0.002**	**-0.133 (0.033), <0.001**	**-0.132 (0.034), <0.001**
Total indirect effect	**-0.052 (0.023), 0.025**	**-0.048 (0.022), 0.025**	**-0.042 (0.018), 0.021**	**-0.039 (0.017), 0.024**
Specific indirect effects				
PGS_{SMOK}→effortful control→educational level	**-0.041 (0.012), 0.001**	**-0.039 (0.012), 0.001**	**-0.034 (0.010), 0.001**	**-0.031 (0.010), 0.001**
PGS_{SMOK}→IQ→educational level	-0.010 (0.017), 0.550	-0.010 (0.016), 0.550	-0.008 (0.013), 0.550	-0.008 (0.013), 0.551
Model 2				
Total effect	-0.064 (0.042), 0.125	-0.077 (0.040), 0.057	**-0.098 (0.038), 0.010**	**-0.096 (0.038), 0.013**
Direct effect	-0.058 (0.035), 0.100	**-0.071 (0.035), 0.042**	**-0.090 (0.034), 0.008**	**-0.090 (0.035), 0.010**
Total indirect effect	-0.007 (0.023), 0.769	-0.006 (0.021), 0.761	-0.007 (0.017), 0.668	-0.006 (0.016), 0.722
Specific indirect effects				
PGS_{SMOK}→effortful control→educational level	**-0.031 (0.013), 0.013**	**-0.029 (0.012), 0.014**	**-0.025 (0.010), 0.014**	**-0.023 (0.010), 0.015**
PGS_{SMOK}→IQ→educational level	0.024 (0.017), 0.146	0.023 (0.016), 0.147	0.018 (0.012), 0.148	0.017 (0.012), 0.148

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{EDU}.

TABLE 4.S7 Total, direct, and indirect effects of PGS_{EDU} on educational level as ordinal variable around age 16, 19, 22, and 26 in the TRAILS study; potential mediators were measured around age 11 (IQ and effortful control); linear and probit regression (WLSMV estimator; beta-coefficient, standard error, p-value)

	Educational level around age 16	**Educational level around age 19**	**Educational level around age 22**	**Educational level around age 26**
Model 1				
Total effect	**-0.391 (0.042), <0.001**	**-0.374 (0.042), <0.001**	**-0.377 (0.038), <0.001**	**-0.363 (0.039), <0.001**
Direct effect	**-0.197 (0.035), <0.001**	**-0.194 (0.036), <0.001**	**-0.231 (0.034), <0.001**	**-0.224 (0.035), <0.001**
Total indirect effect	**-0.194 (0.024), <0.001**	**-0.181 (0.023), <0.001**	**-0.146 (0.019), <0.001**	**-0.139 (0.018), <0.001**
Specific indirect effects				
PGS_{EDU} → effortful control → educational level	**-0.051 (0.013), <0.001**	**-0.048 (0.012), <0.001**	**-0.042 (0.011), <0.001**	**-0.039 (0.010), <0.001**
PGS_{EDU} → IQ → educational level	**-0.143 (0.019), <0.001**	**-0.132 (0.018), <0.001**	**-0.104 (0.015), <0.001**	**-0.100 (0.014), <0.001**
Model 2				
Total effect	**-0.376 (0.043), <0.001**	**-0.356 (0.043), <0.001**	**-0.354 (0.039), <0.001**	**-0.341 (0.040), <0.001**
Direct effect	**-0.183 (0.036), <0.001**	**-0.176 (0.037), <0.001**	**-0.209 (0.035), <0.001**	**-0.203 (0.036), <0.001**
Total indirect effect	**-0.193 (0.025), <0.001**	**-0.179 (0.023), <0.001**	**-0.145 (0.020), <0.001**	**-0.138 (0.019), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→educational level	**-0.043 (0.013), 0.001**	**-0.041 (0.012), 0.001**	**-0.035 (0.011), 0.001**	**-0.032 (0.010), 0.001**
PGS_{EDU}→IQ→educational level	**-0.150 (0.019), <0.001**	**-0.139 (0.018), <0.001**	**-0.110 (0.015), <0.001**	**-0.106 (0.015), <0.001**

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{SMOK}.

TABLE 4.S8 Total, direct, and indirect effects of PGS_{SMOK} on smoking around age 19, 22, and 26 in the TRAILS study; potential mediators were IQ and effortful control, measured around age 11, and educational level, assessed one wave before smoking, i.e., around age 16, 19, and 22; linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Smoking around age 19	**Smoking around age 22**	**Smoking around age 26**
Model 1			
Total effect	**0.347 (0.062), <0.001**	**0.418 (0.063), <0.001**	**0.308 (0.069), <0.001**
Direct effect	**0.304 (0.061), <0.001**	**0.365 (0.061), <0.001**	**0.234 (0.067), <0.001**
Total indirect effect	**0.043 (0.013), 0.001**	**0.053 (0.014), <0.001**	**0.074 (0.017), <0.001**
Specific indirect effects			
PGS_{SMOK}→effortful control→smoking	0.011 (0.007), 0.082	0.010 (0.006), 0.103	0.008 (0.007), 0.231
PGS_{SMOK}→IQ→smoking	0.000 (0.001), 0.762	0.000 (0.001), 0.916	-0.001 (0.002), 0.738
PGS_{SMOK}→education→smoking	**0.020 (0.009), 0.019**	**0.029 (0.010), 0.006**	**0.050 (0.015), 0.001**
PGS_{SMOK}→effortful control→education→smoking	**0.009 (0.003), 0.007**	**0.011 (0.004), 0.004**	**0.013 (0.004), 0.003**
PGS_{SMOK}→IQ→education→smoking	0.002 (0.004), 0.545	0.003 (0.004), 0.544	0.003 (0.005), 0.543
Model 2			
Total effect	**0.289 (0.063), <0.001**	**0.378 (0.064), <0.001**	**0.268 (0.069), <0.001**
Direct effect	**0.270 (0.063), <0.001**	**0.351 (0.062), <0.001**	**0.226 (0.067), 0.001**
Total indirect effect	0.020 (0.011), 0.084	**0.027 (0.013), 0.041**	**0.042 (0.016), 0.010**
Specific indirect effects			
PGS_{SMOK}→effortful control→smoking	0.008 (0.005), 0.138	0.007 (0.005), 0.153	0.006 (0.005), 0.267
PGS_{SMOK}→IQ →smoking	0.000 (0.003), 0.997	0.000 (0.003), 0.951	0.002 (0.003), 0.560
PGS_{SMOK}→education→smoking	0.010 (0.007), 0.162	0.018 (0.010), 0.065	**0.032 (0.014), 0.021**
PGS_{SMOK}→effortful control→education→smoking	**0.006 (0.003), 0.031**	**0.008 (0.003), 0.022**	**0.009 (0.004), 0.018**
PGS_{SMOK}→IQ→education→smoking	-0.005 (0.004), 0.166	-0.006 (0.004), 0.155	-0.007 (0.005), 0.151

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{EDU}.

TABLE 4.S9 Total, direct, and indirect effects of PGS_{EDU} on smoking around age 19, 22, and 26 in the TRAILS study; potential mediators were IQ and effortful control, measured around age 11, and educational level, assessed one wave before smoking, i.e., around age 16, 19, and 22; linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Smoking around age 19	Smoking around age 22	Smoking around age 26
Model 1			
Total effect	**0.322 (0.056), <0.001**	**0.258 (0.055), <0.001**	**0.233 (0.065), <0.001**
Direct effect	**0.232 (0.057), <0.001**	**0.149 (0.057), 0.009**	0.095 (0.069), 0.170
Total indirect effect	**0.090 (0.020), <0.001**	**0.109 (0.021), <0.001**	**0.139 (0.026), <0.001**
Specific indirect effects			
PGS_{EDU}→effortful control→smoking	0.014 (0.008), 0.060	**0.014 (0.007), 0.049**	0.010 (0.008), 0.218
PGS_{EDU}→IQ→smoking	-0.005 (0.015), 0.752	-0.003 (0.014), 0.857	-0.017 (0.018), 0.330
PGS_{EDU}→education→smoking	**0.041 (0.012), 0.001**	**0.050 (0.013), <0.001**	**0.090 (0.020), <0.001**
PGS_{EDU}→effortful control→education→smoking	**0.011 (0.004), 0.005**	**0.013 (0.004), 0.002**	**0.016 (0.005), 0.001**
PGS_{EDU}→IQ→education→smoking	**0.029 (0.008), <0.001**	**0.034 (0.008), <0.001**	**0.040 (0.008), <0.001**
Model 2			
Total effect	**0.256 (0.059), <0.001**	**0.176 (0.057), 0.002**	**0.178 (0.067), 0.007**
Direct effect	**0.168 (0.060), 0.005**	0.072 (0.059), 0.224	0.047 (0.070), 0.503
Total indirect effect	**0.088 (0.020), <0.001**	**0.104 (0.021), <0.001**	**0.131 (0.026), <0.001**
Specific indirect effects			
PGS_{EDU}→effortful control→smoking	0.011 (0.007), 0.097	0.010 (0.007), 0.120	0.008 (0.007), 0.233
PGS_{EDU}→IQ →smoking	0.000 (0.015), 0.999	-0.001 (0.015), 0.952	-0.012 (0.018), 0.503
PGS_{EDU}→education→smoking	**0.038 (0.012), 0.002**	**0.047 (0.013), <0.001**	**0.080 (0.019), <0.001**
PGS_{EDU}→effortful control→education→smoking	**0.009 (0.004), 0.012**	**0.011 (0.004), 0.008**	**0.013 (0.005), 0.005**
PGS_{EDU}→IQ→education→smoking	**0.030 (0.008), <0.001**	**0.037 (0.009), <0.001**	**0.042 (0.009), <0.001**

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{SMOK}.

TABLE 4.S10 Total, direct, and indirect effects of PGS_{SMOK} on smoking around age 16, 19, 22, and 26 in the TRAILS general population cohort (N = 1,248); potential mediators were measured around age 11 (IQ and effortful control) and concurrently with smoking (educational level); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Smoking around age 16	Smoking around age 19	Smoking around age 22	Smoking around age 26
Model 1				
Total effect	**0.413 (0.085), <0.001**	**0.420 (0.076), <0.001**	**0.481 (0.077), <0.001**	**0.322 (0.078), <0.001**
Direct effect	**0.355 (0.084), <0.001**	**0.348 (0.074), <0.001**	**0.409 (0.075), <0.001**	**0.247 (0.075), 0.001**
Total indirect effect	**0.058 (0.019), 0.003**	**0.073 (0.020), <0.001**	**0.072 (0.019), <0.001**	**0.075 (0.021), <0.001**
Specific indirect effects				
PGS_{SMOK}→effortful control→smoking	0.000 (0.009), 0.977	0.002 (0.006), 0.707	0.004 (0.006), 0.543	0.004 (0.007), 0.531
PGS_{SMOK}→IQ→smoking	0.001 (0.002), 0.774	0.000 (0.001), 0.883	0.000 (0.001), 0.945	0.000 (0.002), 0.851
PGS_{SMOK}→education→smoking	**0.039 (0.015), 0.009**	**0.051 (0.017), 0.002**	**0.052 (0.016), 0.001**	**0.055 (0.018), 0.002**
PGS_{SMOK}→effortful control→education→smoking	**0.015 (0.006), 0.012**	**0.016 (0.006), 0.009**	**0.014 (0.005), 0.008**	**0.013 (0.005), 0.011**
PGS_{SMOK}→IQ→education→smoking	0.003 (0.006), 0.640	0.003 (0.007), 0.640	0.002 (0.005), 0.641	0.002 (0.005), 0.640
Model 2				
Total effect	**0.360 (0.085), <0.001**	**0.357 (0.076), <0.001**	**0.440 (0.077), <0.001**	**0.278 (0.078), <0.001**
Direct effect	**0.336 (0.084), <0.001**	**0.318 (0.074), <0.001**	**0.403 (0.076), <0.001**	**0.239 (0.076), 0.002**
Total indirect effect	0.025 (0.018), 0.167	**0.038 (0.018), 0.037**	**0.037 (0.018), 0.038**	**0.039 (0.019), 0.044**
Specific indirect effects				
PGS_{SMOK}→effortful control→smoking	-0.001 (0.006), 0.900	0.001 (0.004), 0.857	0.003 (0.005), 0.584	0.003 (0.005), 0.576
PGS_{SMOK}→IQ →smoking	-0.001 (0.004), 0.858	0.002 (0.003), 0.597	0.000 (0.003), 0.971	0.000 (0.004), 0.900
PGS_{SMOK}→education→smoking	0.024 (0.014), 0.080	**0.033 (0.015), 0.030**	**0.032 (0.015), 0.033**	**0.035 (0.016), 0.031**
PGS_{SMOK}→effortful control→education→smoking	0.010 (0.005), 0.065	0.011 (0.006), 0.060	0.009 (0.005), 0.057	0.008 (0.005), 0.062
PGS_{SMOK}→IQ→education→smoking	-0.008 (0.006), 0.181	-0.009 (0.006), 0.176	-0.007 (0.005), 0.176	-0.007 (0.005), 0.178

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{EDU}.

TABLE 4.S11 Total, direct, and indirect effects of PGS_{EDU} on smoking around age 16, 19, 22, and 26 in the TRAILS general population cohort (N = 1,248); potential mediators were measured around age 11 (IQ and effortful control) and concurrently with smoking (educational level); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Smoking around age 16	Smoking around age 19	Smoking around age 22	Smoking around age 26
Model 1				
Total effect	**0.356 (0.086), <0.001**	**0.373 (0.068), <0.001**	**0.271 (0.066), <0.001**	**0.257 (0.074), 0.001**
Direct effect	**0.214 (0.086), 0.013**	**0.234 (0.069), 0.001**	0.117 (0.068), 0.085	0.097 (0.079), 0.218
Total indirect effect	**0.142 (0.030), <0.001**	**0.138 (0.026), <0.001**	**0.154 (0.027), <0.001**	**0.160 (0.031), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→smoking	0.000 (0.013), 0.982	0.004 (0.009), 0.680	0.009 (0.009), 0.303	0.007 (0.010), 0.439
PGS_{EDU}→IQ→smoking	-0.005 (0.022), 0.808	-0.017 (0.017), 0.327	-0.004 (0.016), 0.802	-0.003 (0.019), 0.888
PGS_{EDU}→education→smoking	**0.076 (0.020), <0.001**	**0.080 (0.020), <0.001**	**0.093 (0.021), <0.001**	**0.098 (0.024), <0.001**
PGS_{EDU}→effortful control→education→smoking	**0.023 (0.007), 0.002**	**0.024 (0.007), 0.001**	**0.020 (0.006), 0.001**	**0.019 (0.006), 0.001**
PGS_{EDU}→IQ→education→smoking	**0.048 (0.012), <0.001**	**0.048 (0.010), <0.001**	**0.036 (0.008), <0.001**	**0.038 (0.009), <0.001**
Model 2				
Total effect	**0.293 (0.086), 0.001**	**0.308 (0.072), <0.001**	**0.186 (0.068), 0.007**	**0.207 (0.075), 0.006**
Direct effect	0.158 (0.086), 0.068	**0.173 (0.073), 0.017**	0.036 (0.071), 0.609	0.055 (0.080), 0.491
Total indirect effect	**0.135 (0.029), <0.001**	**0.135 (0.027), <0.001**	**0.150 (0.027), <0.001**	**0.152 (0.031), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→smoking	-0.002 (0.012), 0.896	0.002 (0.008), 0.851	0.005 (0.008), 0.563	0.005 (0.009), 0.555
PGS_{EDU}→IQ→smoking	0.005 (0.023), 0.842	-0.011 (0.018), 0.555	0.000 (0.017), 0.978	0.003 (0.020), 0.885
PGS_{EDU}→education→smoking	**0.065 (0.019), 0.001**	**0.072 (0.019), <0.001**	**0.088 (0.021), <0.001**	**0.088 (0.023), <0.001**
PGS_{EDU}→effortful control→education→smoking	**0.019 (0.007), 0.004**	**0.021 (0.007), 0.002**	**0.018 (0.006), 0.003**	**0.016 (0.006), 0.003**
PGS_{EDU}→IQ → education→smoking	**0.047 (0.012), <0.001**	**0.051 (0.011), <0.001**	**0.040 (0.009), <0.001**	**0.040 (0.009), <0.001**

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{SMOK}.

TABLE 4.S12 Total, direct, and indirect effects of PGS_{SMOK} on educational level around age 16, 19, 22, and 26 in the TRAILS general population cohort (N = 1,248); potential mediators were measured around age 11 (IQ and effortful control); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Educational level around age 16	**Educational level around age 19**	**Educational level around age 22**	**Educational level around age 26**
Model 1				
Total effect	**-0.107 (0.029), <0.001**	**-0.104 (0.026), <0.001**	**-0.105 (0.025), <0.001**	**-0.109 (0.026), <0.001**
Direct effect	**-0.073 (0.024), 0.002**	**-0.075 (0.021), 0.001**	**-0.080 (0.022), <0.001**	**-0.085 (0.024), <0.001**
Total indirect effect	**-0.034 (0.016), 0.039**	**-0.029 (0.014), 0.040**	**-0.025 (0.012), 0.035**	**-0.024 (0.012), 0.043**
Specific indirect effects				
PGS_{SMOK}→effortful control→educational level	**-0.028 (0.010), 0.004**	**-0.024 (0.008), 0.004**	**-0.021 (0.007), 0.004**	**-0.020 (0.007), 0.005**
PGS_{SMOK}→IQ→educational level	-0.005 (0.011), 0.637	-0.005 (0.010), 0.637	-0.004 (0.008), 0.637	-0.004 (0.008), 0.638
Model 2				
Total effect	-0.050 (0.029), 0.085	**-0.055 (0.026), 0.034**	**-0.054 (0.025), 0.031**	**-0.058 (0.026), 0.028**
Direct effect	-0.046 (0.024), 0.056	**-0.051 (0.022), 0.020**	**-0.050 (0.022), 0.024**	**-0.055 (0.024), 0.021**
Total indirect effect	-0.004 (0.016), 0.791	-0.004 (0.014), 0.785	-0.004 (0.011), 0.716	-0.003 (0.011), 0.801
Specific indirect effects				
PGS_{SMOK}→effortful control→educational level	**-0.020 (0.010), 0.046**	**-0.017 (0.008), 0.047**	**-0.015 (0.007), 0.047**	**-0.013 (0.007), 0.049**
PGS_{SMOK}→IQ→educational level	0.015 (0.011), 0.162	0.013 (0.009), 0.163	0.010 (0.007), 0.164	0.011 (0.008), 0.165

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{EDU}.

TABLE 4.S13 Total, direct, and indirect effects of PGS_{EDU} on educational level around age 16, 19, 22, and 26 in the TRAILS general population cohort (N = 1,248); potential mediators were measured around age 11 (IQ and effortful control); and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Educational level around age 16	Educational level around age 19	Educational level around age 22	Educational level around age 26
Model 1				
Total effect	**-0.266 (0.029), <0.001**	**-0.232 (0.026), <0.001**	**-0.241 (0.025), <0.001**	**-0.240 (0.027), <0.001**
Direct effect	**-0.137 (0.025), <0.001**	**-0.122 (0.022), <0.001**	**-0.150 (0.023), <0.001**	**-0.152 (0.025), <0.001**
Total indirect effect	**-0.129 (0.017), <0.001**	**-0.109 (0.015), <0.001**	**-0.091 (0.013), <0.001**	**-0.089 (0.013), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→educational level	**-0.042 (0.011), <0.001**	**-0.036 (0.009), <0.001**	**-0.032 (0.008), <0.001**	**-0.029 (0.008), <0.001**
PGS_{EDU}→IQ→educational level	**-0.087 (0.012), <0.001**	**-0.073 (0.011), <0.001**	**-0.059 (0.009), <0.001**	**-0.060 (0.010), <0.001**
Model 2				
Total effect	**-0.254 (0.030), <0.001**	**-0.219 (0.026), <0.001**	**-0.227 (0.027), <0.001**	**-0.226 (0.028), <0.001**
Direct effect	**-0.125 (0.026), <0.001**	**-0.110 (0.023), <0.001**	**-0.138 (0.024), <0.001**	**-0.138 (0.025), <0.001**
Total indirect effect	**-0.128 (0.018), <0.001**	**-0.109 (0.015), <0.001**	**-0.090 (0.013), <0.001**	**-0.088 (0.013), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→educational level	**-0.037 (0.011), <0.001**	**-0.032 (0.009), 0.001**	**-0.028 (0.008), 0.001**	**-0.025 (0.007), 0.001**
PGS_{EDU}→IQ→educational level	**-0.091 (0.013), <0.001**	**-0.077 (0.011), <0.001**	**-0.062 (0.010), <0.001**	**-0.063 (0.010), <0.001**

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{SMOK}.

TABLE 4.S14 Total, direct, and indirect effects of PGS_{SMOK} on smoking around age 16, 19, 22, and 26 in the TRAILS study in complete case analyses; potential mediators were measured around age 11 (IQ and effortful control) and concurrently with smoking (educational level); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Smoking around age 16	**Smoking around age 19**	**Smoking around age 22**	**Smoking around age 26**
Model 1	N = 1,107	N = 1,032	N = 970	N = 838
Total effect	**0.384 (0.095), <0.001**	**0.418 (0.083), <0.001**	**0.468 (0.086), <0.001**	**0.337 (0.087), <0.001**
Direct effect	**0.324 (0.094), 0.001**	**0.361 (0.081), <0.001**	**0.411 (0.084), <0.001**	**0.268 (0.082), 0.001**
Total indirect effect	**0.059 (0.018), 0.001**	**0.057 (0.019), 0.003**	**0.057 (0.019), 0.003**	**0.069 (0.025), 0.005**
Specific indirect effects				
PGS_{SMOK}→effortful control→smoking	0.009 (0.011), 0.382	0.004 (0.006), 0.510	0.000 (0.004), 0.984	0.003 (0.004), 0.482
PGS_{SMOK}→IQ→smoking	-0.003 (0.005), 0.548	-0.002 (0.004), 0.599	-0.003 (0.004), 0.500	-0.001 (0.004), 0.736
PGS_{SMOK}→education→smoking	**0.032 (0.014), 0.023**	**0.034 (0.016), 0.031**	**0.045 (0.017), 0.009**	**0.050 (0.021), 0.019**
PGS_{SMOK}→effortful control→education→smoking	**0.017 (0.006), 0.006**	**0.016 (0.007), 0.017**	0.008 (0.005), 0.144	0.007 (0.006), 0.293
PGS_{SMOK}→IQ→education→smoking	0.004 (0.007), 0.512	0.005 (0.008), 0.555	0.006 (0.007), 0.427	0.011 (0.009), 0.222
Model 2	N = 1,107	N = 1,032	N = 970	N = 838
Total effect	**0.341 (0.094), <0.001**	**0.364 (0.085), <0.001**	**0.453 (0.085), <0.001**	**0.326 (0.090), <0.001**
Direct effect	**0.301 (0.095), 0.001**	**0.336 (0.083), <0.001**	**0.424 (0.082), <0.001**	**0.293 (0.084), 0.001**
Total indirect effect	**0.041 (0.018), 0.022**	0.029 (0.019), 0.135	0.029 (0.019), 0.131	0.032 (0.026), 0.216
Specific indirect effects				
PGS_{SMOK}→effortful control→smoking	0.008 (0.009), 0.412	0.003 (0.005), 0.563	0.000 (0.003), 0.954	0.002 (0.004), 0.563
PGS_{SMOK}→IQ →smoking	0.008 (0.007), 0.284	0.006 (0.006), 0.307	0.004 (0.004), 0.416	0.000 (0.002), 0.955
PGS_{SMOK}→education→smoking	0.019 (0.013), 0.148	0.018 (0.015), 0.254	0.028 (0.017), 0.104	0.030 (0.022), 0.171
PGS_{SMOK}→effortful control→education→smoking	**0.014 (0.006), 0.015**	0.012 (0.006), 0.054	0.006 (0.006), 0.272	0.005 (0.007), 0.424
PGS_{SMOK}→IQ→education→smoking	-0.008 (0.006), 0.222	-0.010 (0.008), 0.182	-0.009 (0.007), 0.221	-0.006 (0.009), 0.509

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{EDU}.

TABLE 4.S15 Total, direct, and indirect effects of PGS_{EDU} on smoking around age 16, 19, 22, and 26 in the TRAILS study in complete case analyses; potential mediators were measured around age 11 (IQ and effortful control) and concurrently with smoking (educational level); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Smoking around age 16	**Smoking around age 19**	**Smoking around age 22**	**Smoking around age 26**
Model 1	N = 1,107	N = 1,032	N = 970	N = 838
Total effect	**0.339 (0.090), <0.001**	**0.302 (0.072), <0.001**	**0.210 (0.074), 0.004**	0.134 (0.083), 0.105
Direct effect	**0.254 (0.093), 0.006**	**0.197 (0.074), 0.008**	0.096 (0.076), 0.208	-0.043 (0.086), 0.620
Total indirect effect	**0.085 (0.032), 0.007**	**0.104 (0.027), <0.001**	**0.114 (0.029), <0.001**	**0.177 (0.038), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→smoking	0.008 (0.009), 0.390	0.006 (0.007), 0.394	0.002 (0.004), 0.587	0.003 (0.004), 0.509
PGS_{EDU}→IQ→smoking	**-0.059 (0.026), 0.026**	-0.042 (0.022), 0.056	-0.028 (0.022), 0.200	-0.004 (0.026), 0.880
PGS_{EDU}→education→smoking	**0.067 (0.019), 0.001**	**0.070 (0.018), <0.001**	**0.078 (0.021), <0.001**	**0.102 (0.027), <0.001**
PGS_{EDU}→effortful control→education→smoking	**0.015 (0.007), 0.021**	**0.015 (0.007), 0.022**	0.009 (0.006), 0.129	0.008 (0.007), 0.277
PGS_{EDU}→IQ→education→smoking	**0.055 (0.013), <0.001**	**0.056 (0.011), <0.001**	**0.053 (0.011), <0.001**	**0.069 (0.014), <0.001**
Model 2	N = 1,107	N = 1,032	N = 970	N = 838
Total effect	**0.278 (0.092), 0.002**	**0.217 (0.076), 0.005**	0.078 (0.073), 0.283	0.058 (0.088), 0.509
Direct effect	**0.206 (0.094), 0.028**	0.105 (0.079), 0.182	-0.042 (0.077), 0.581	-0.118 (0.092), 0.200
Total indirect effect	**0.073 (0.032), 0.024**	**0.111 (0.029), <0.001**	**0.121 (0.030), <0.001**	**0.175 (0.041), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→smoking	0.006 (0.007), 0.430	0.003 (0.005), 0.548	0.000 (0.003), 0.954	0.003 (0.004), 0.526
PGS_{EDU}→IQ→smoking	-0.054 (0.028), 0.052	-0.034 (0.023), 0.139	-0.024 (0.023), 0.280	-0.002 (0.028), 0.955
PGS_{EDU}→education→smoking	**0.057 (0.018), 0.002**	**0.068 (0.019), <0.001**	**0.077 (0.022), 0.001**	**0.096 (0.028), 0.001**
PGS_{EDU}→effortful control→education→smoking	0.010 (0.006), 0.075	0.013 (0.007), 0.064	0.008 (0.006), 0.216	0.006 (0.007), 0.384
PGS_{EDU}→IQ → education→smoking	**0.053 (0.013), <0.001**	**0.061 (0.012), <0.001**	**0.060 (0.012), <0.001**	**0.072 (0.015), <0.001**

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{SMOK}.

TABLE 4.S16 Total, direct, and indirect effects of PGS_{SMOK} on educational level around age 16, 19, 22, and 26 in the TRAILS study in complete case analyses; potential mediators were measured around age 11 (IQ and effortful control); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Educational level around age 16	**Educational level around age 19**	**Educational level around age 22**	**Educational level around age 26**
Model 1	N = 1,107	N = 1,032	N = 970	N = 838
Total effect	**-0.099 (0.028), <0.001**	**-0.076 (0.025), 0.003**	**-0.078 (0.024), 0.001**	**-0.077 (0.027), 0.004**
Direct effect	**-0.059 (0.023), 0.009**	**-0.047 (0.021), 0.023**	**-0.059 (0.021), 0.005**	**-0.057 (0.023), 0.015**
Total indirect effect	**-0.039 (0.016), 0.017**	-0.029 (0.015), 0.056	-0.018 (0.013), 0.159	-0.020 (0.014), 0.133
Specific indirect effects				
PGS_{SMOK}→effortful control→educational level	**-0.031 (0.009), 0.001**	**-0.022 (0.009), 0.010**	-0.011 (0.007), 0.138	-0.007 (0.007), 0.289
PGS_{SMOK}→IQ→educational level	-0.008 (0.012), 0.510	-0.007 (0.011), 0.554	-0.008 (0.010), 0.426	-0.013 (0.010), 0.218
Model 2	N = 1,107	N = 1,032	N = 970	N = 838
Total effect	-0.047 (0.028), 0.092	-0.028 (0.026), 0.273	-0.033 (0.025), 0.182	-0.033 (0.027), 0.223
Direct effect	-0.036 (0.023), 0.126	-0.025 (0.021), 0.247	-0.036 (0.022), 0.095	-0.033 (0.024), 0.168
Total indirect effect	-0.012 (0.016), 0.465	-0.003 (0.015), 0.820	0.003 (0.013), 0.809	0.000 (0.013), 0.970
Specific indirect effects				
PGS_{SMOK}→effortful control→educational level	**-0.027 (0.009), 0.005**	**-0.018 (0.009), 0.042**	-0.008 (0.007), 0.268	-0.006 (0.007), 0.422
PGS_{SMOK}→IQ→educational level	0.015 (0.012), 0.204	0.014 (0.010), 0.173	0.011 (0.009), 0.214	0.006 (0.010), 0.507

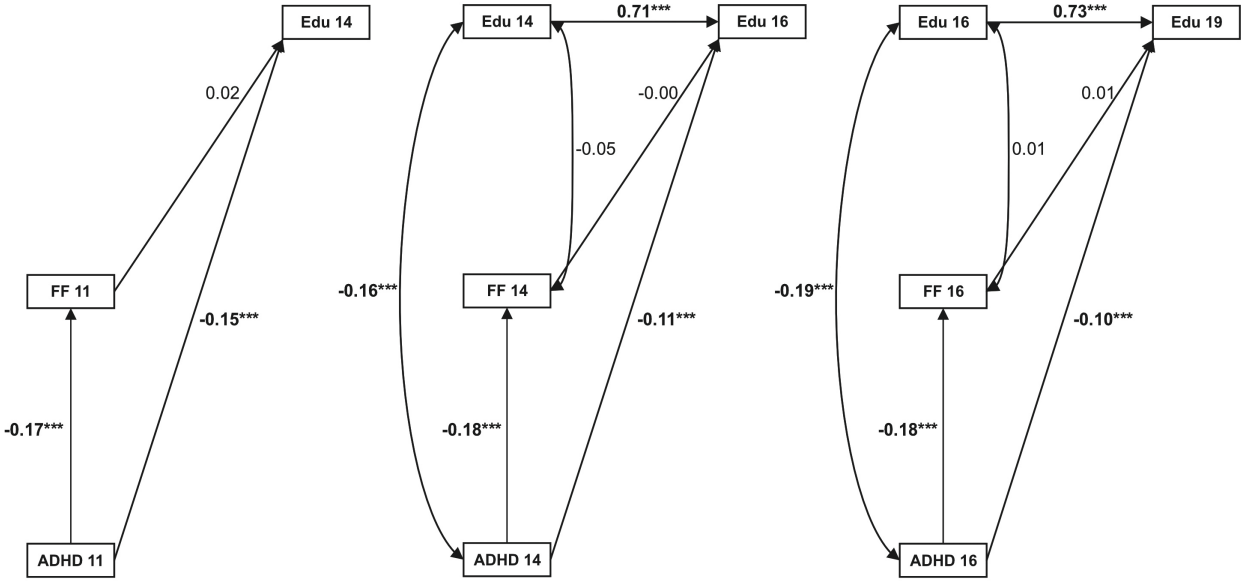
Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{EDU}.

TABLE 4.S17 Total, direct, and indirect effects of PGS_{EDU} on educational level around age 16, 19, 22, and 26 in the TRAILS study in complete case analyses; potential mediators were measured around age 11 (IQ and effortful control); linear and negative binomial regression models (MLR estimator; beta-coefficient, standard error, p-value)

	Educational level around age 16	**Educational level around age 19**	**Educational level around age 22**	**Educational level around age 26**
Model 1	N = 1,107	N = 1,032	N = 970	N = 838
Total effect	**-0.240 (0.029), <0.001**	**-0.208 (0.026), <0.001**	**-0.197 (0.027), <0.001**	**-0.200 (0.030), <0.001**
Direct effect	**-0.118 (0.025), <0.001**	**-0.103 (0.023), <0.001**	**-0.110 (0.024), <0.001**	**-0.114 (0.027), <0.001**
Total indirect effect	**-0.122 (0.017), <0.001**	**-0.105 (0.015), <0.001**	**-0.087 (0.014), <0.001**	**-0.086 (0.015), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→educational level	**-0.026 (0.010), 0.010**	**-0.023 (0.009), 0.014**	-0.012 (0.008), 0.119	-0.009 (0.008), 0.275
PGS_{EDU}→IQ→educational level	**-0.096 (0.013), <0.001**	**-0.082 (0.011), <0.001**	**-0.075 (0.011), <0.001**	**-0.077 (0.012), <0.001**
Model 2	N = 1,107	N = 1,032	N = 970	N = 838
Total effect	**-0.228 (0.030), <0.001**	**-0.201 (0.027), <0.001**	**-0.189 (0.029), <0.001**	**-0.192 (0.031), <0.001**
Direct effect	**-0.109 (0.026), <0.001**	**-0.096 (0.023), <0.001**	**-0.101 (0.025), <0.001**	**-0.106 (0.028), <0.001**
Total indirect effect	**-0.120 (0.018), <0.001**	**-0.104 (0.016), <0.001**	**-0.088 (0.015), <0.001**	**-0.086 (0.016), <0.001**
Specific indirect effects				
PGS_{EDU}→effortful control→educational level	-0.020 (0.010), 0.056	-0.018 (0.009), 0.054	-0.010 (0.008), 0.210	-0.007 (0.008), 0.382
PGS_{EDU}→IQ→educational level	**-0.100 (0.014), <0.001**	**-0.086 (0.012), <0.001**	**-0.078 (0.011), <0.001**	**-0.079 (0.013), <0.001**

Boldface *denotes statistical significance at p<0.05. Models 1 are adjusted for sex, age, cohort type, parental education, and parental smoking; Models 2 are additionally adjusted for* PGS_{SMOK}.

FIGURE 5.S1 Direct and indirect effects of ADHD symptoms on (changes in) educational level in each subsequent wave in the TRAILS study (wave 1 – 4, the Netherlands, 2000–2010, N = 2,229); family functioning (FF; potential mediator) was measured concurrently with ADHD symptoms; linear and probit regression (WLSMV estimator; standardized beta-coefficient, 95% Confidence Interval, p-value)



	IE	DE	PM	Mean fit statistics					
				$\chi 2$ test	df	CFI	TLI	SRMR	RMSEA
Wave 1 - wave 2	0.00 (-0.01 – 0.00), 0.232	**-0.15 (-0.19 – -0.12), <0.001**	0.03	0.000	0	1.000	1.000	0.000	0.000
Wave 2 - wave 3	0.00 (0.00 – 0.01), 0.880	**-0.11 (-0.14 – -0.08), <0.001**	0.00	0.000	0	1.000	1.000	0.000	0.000
Wave 3 - wave 4	0.00 (-0.01 – 0.01), 0.689	**-0.10 (-0.13 – -0.07), <0.001**	0.01	0.000	0	1.000	1.000	0.000	0.000

*IE = indirect effect; DE = direct effect; PM = proportion mediated; Edu = educational level; FF = family functioning; * p<0.05; ** p<0.01; *** p<0.001.* ***Boldface*** *denotes statistical significance at p<0.05. All regressions were adjusted for time-stable covariates measured at baseline (i.e., gender, ethnicity, IQ, parental SES) and age assessed in the same wave ADHD symptoms and potential mediators were measured.*

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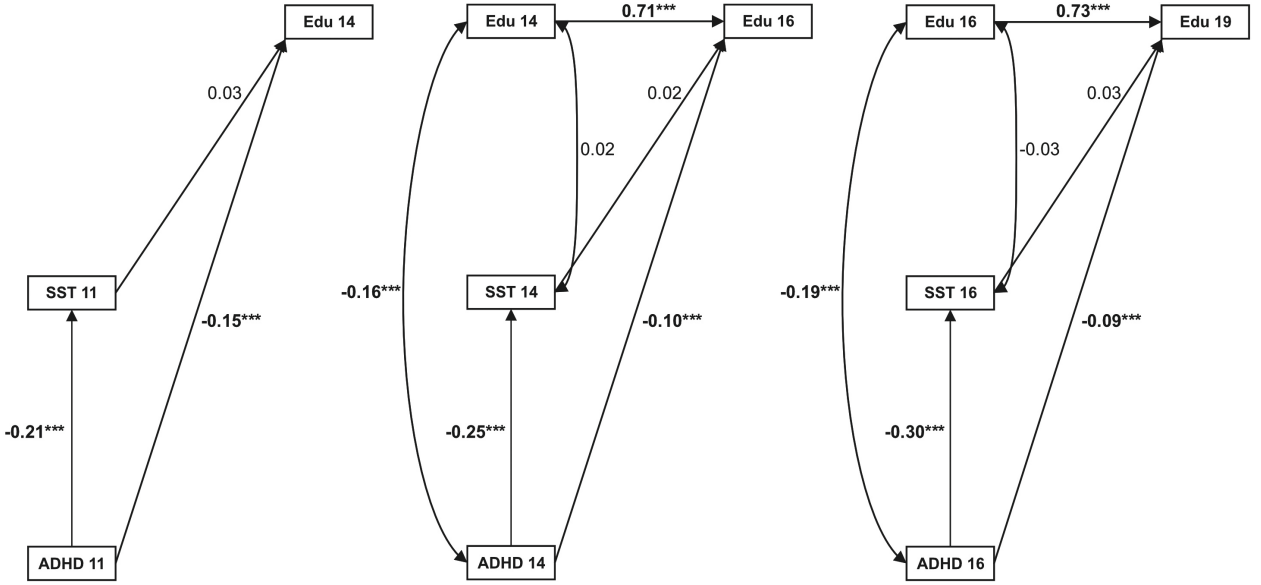
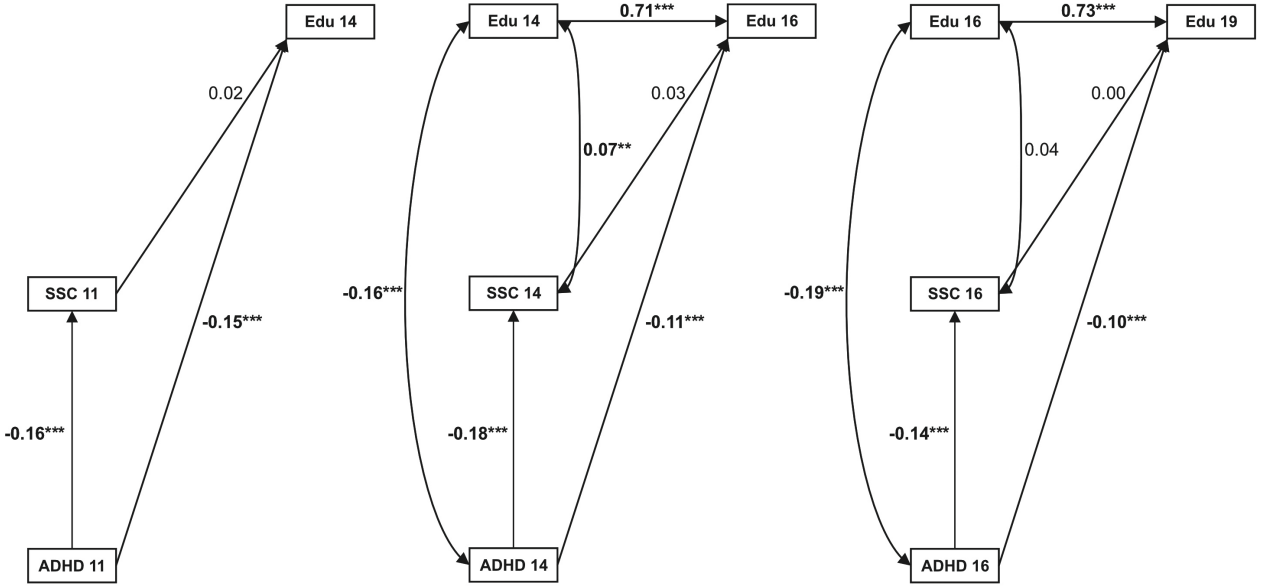
FIGURE 5.S2 Direct and indirect effects of ADHD symptoms on (changes in) educational level in each subsequent wave in the TRAILS study (wave 1 – 4, the Netherlands, 2000–2010, N = 2,229); social support by teachers (SST; potential mediator) was measured concurrently with ADHD symptoms; linear and probit regression (WLSMV estimator; standardized beta-coefficient, 95% Confidence Interval, p-value)



	IE	DE	PM	Mean fit statistics					
				χ2 test	df	CFI	TLI	SRMR	RMSEA
Wave 1 - wave 2	-0.01 (-0.01 – 0.00), 0.137	**-0.15 (-0.19 – -0.12), <0.001**	0.04	0.000	0	1.000	1.000	0.000	0.000
Wave 2 - wave 3	-0.01 (-0.01 – 0.00), 0.143	**-0.10 (-0.13 – -0.07), <0.001**	0.05	0.000	0	1.000	1.000	0.000	0.000
Wave 3 - wave 4	-0.01 (-0.02 – 0.00), 0.104	**-0.09 (-0.12 – -0.06), <0.001**	0.08	0.000	0	1.000	1.000	0.000	0.000

*IE = indirect effect; DE = direct effect; PM = proportion mediated; Edu = educational level; SST = social support by teachers; * p<0.5; ** p<0.01; *** p<0.001.* ***Boldface*** *denotes statistical significance at p<0.05. All regressions were adjusted for time-stable covariates measured at baseline (i.e., gender, ethnicity, IQ, parental SES) and age assessed in the same wave ADHD symptoms and potential mediators were measured.*

FIGURE 5.S3 Direct and indirect effects of ADHD symptoms on (changes in) educational level in each subsequent wave in the TRAILS study (wave 1 – 4, the Netherlands, 2000–2010, N = 2,229); social support by classmates (SSC; mediator) was measured concurrently with ADHD symptoms; linear and probit regression (WLSMV estimator; standardized beta-coefficient, 95% Confidence Interval, p-value)



	IE	DE	PM	Mean fit statistics					
				χ2 test	df	CFI	TLI	SRMR	RMSEA
Wave 1 - wave 2	0.00 (-0.01 – 0.00), 0.198	**-0.15 (-0.19 – -0.12), <0.001**	0.03	0.000	0	1.000	1.000	0.000	0.000
Wave 2 - wave 3	-0.01 (-0.01 – 0.00), 0.101	**-0.11 (-0.14 – -0.08), <0.001**	0.05	0.000	0	1.000	1.000	0.000	0.000
Wave 3 - wave 4	0.00 (-0.01 – 0.00), 0.868	**-0.10 (-0.13 – -0.07), <0.001**	0.00	0.000	0	1.000	1.000	0.000	0.000

*IE = indirect effect; DE = direct effect; PM = proportion mediated; Edu = educational level; SSC = social support by classmates; * p<0.5; ** p<0.01; *** p<0.001.* ***Boldface*** *denotes statistical significance at p<0.05. All regressions were adjusted for time-stable covariates measured at baseline (i.e., gender, ethnicity, IQ, parental SES) and age assessed in the same wave ADHD symptoms and potential mediators were measured.*

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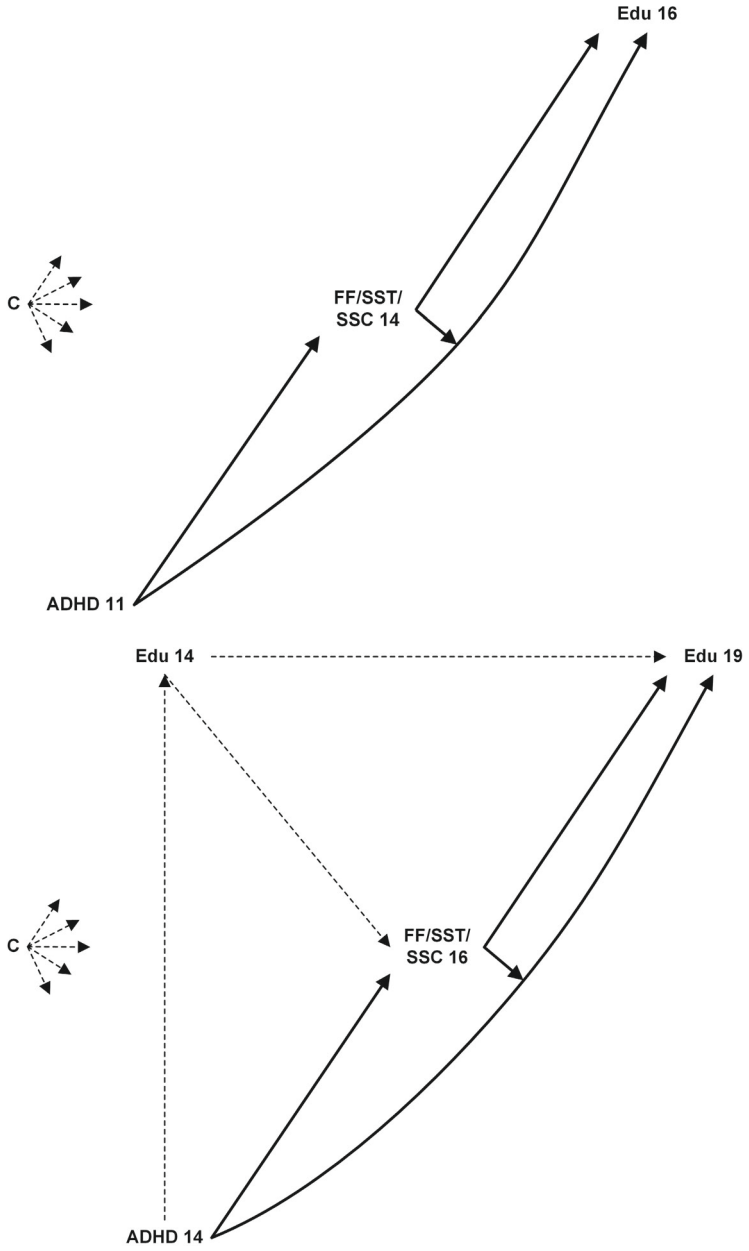
FIGURE 5.S4 Illustrations of the hypothesized relationships between ADHD symptoms, family and school factors, and educational level across adolescence, allowing each one wave time lag between the measurements of exposure, mediators, and outcomes



Edu = education; FF = family functioning; SST = social support by teachers; SSC = social support by classmates; C = covariates, which were included in all regression equations (i.e., gender, ethnicity, IQ, and parental SES measured at baseline, and age assessed in the same wave ADHD symptoms and potential mediators were measured).

TABLE 5.S1 Attrition analysis – characteristics of young adults remaining in the TRAILS study (wave 1 – 4, Netherlands, 2000–2010, N = 2,229) at wave 4, compared to participants who had dropped out of the cohort between wave 1 and wave 4

	Participants remaining in TRAILS by wave 4		**Drop-outs**		**P-value**
N participants (%)	1,880	(84.34)	349	(15.66)	
Baseline characteristics					
Male gender, N (%)	898	(47.77)	200	(57.31)	0.001
Non-Dutch ethnicity, N (%)	212	(11.28)	89	(25.50)	<0.001
Parental socioeconomic status (SES), mean (SD)	0.04	(0.78)	-0.53	(0.77)	<0.001
Wechsler Intelligence Deviation Quotient (IQ), mean (SD)	98.61	(14.67)	89.45	(14.40)	<0.001
Educational level, mean (SD)					
Wave 2	2.45	(1.15)	1.57	(0.84)	<0.001
Wave 3	2.60	(1.10)	1.56	(0.85)	<0.001
ADHD symptoms, mean (SD)					
Wave 1	0.57	(0.33)	0.61	(0.36)	0.035
Wave 2	0.54	(0.32)	0.54	(0.34)	0.970
Wave 3	0.52	(0.31)	0.55	(0.37)	0.469
Family functioning, mean (SD)					
Wave 1	3.23	(0.36)	3.20	(0.37)	0.147
Wave 2	3.38	(0.39)	3.27	(0.42)	0.001
Wave 3	3.35	(0.40)	3.39	(0.44)	0.362
Social support by teachers, mean (SD)					
Wave 1	3.82	(0.68)	3.79	(0.76)	0.480
Wave 2	3.49	(0.65)	3.44	(0.68)	0.322
Wave 3	3.43	(0.60)	3.48	(0.66)	0.418
Social support by classmates, mean (SD)					
Wave 1	3.58	(0.72)	3.61	(0.77)	0.502
Wave 2	3.60	(0.63)	3.52	(0.78)	0.079
Wave 3	3.58	(0.55)	3.51	(0.63)	0.219
Age, mean (SD)					
Wave 1	11.09	(0.56)	11.21	(0.54)	<0.001
Wave 2	13.55	(0.53)	13.69	(0.52)	<0.001
Wave 3	16.27	(0.70)	16.42	(0.82)	0.015

SD = standard deviation. P-values were computed using chi-squared tests for categorical variables and two-sample t-tests for continuous variables.

TABLE 5.S2 Characteristics of participants with classifiable educational level compared to those with missing/ unclassifiable edwucational level from wave 2 to 4 in the TRAILS study (wave 1 – 4, the Netherlands, 2000–2010, N = 2,229)

	N participants per wave		**Male gender**		**Non-Dutch ethnicity**		**Parental socio-economic status (SES)**	
	N	(%)	N	(%)	N	(SD)	Mean	(SD)
Wave 2	2,148	(100)						
Education complete	1,927	(89.71)	924	(47.95)	237	(0.56)	-0.03	(0.80)
Education unclassifiable/missing	221	(10.29)	130	(58.82)	35	(0.53)	-0.07	(0.75)
P-value				0.002		0.344		0.531
Wave 3	1,818	(100)						
Education complete	1,529	(84.10)	704	(46.04)	175	(0.56)	0.08	(0.78)
Education unclassifiable/missing	289	(15.90)	163	(56.40)	36	(0.57)	-0.32	(0.78)
P-value				0.001		0.004		<0.001
Wave 4	1,880	(100)						
Education complete	1,507	(80.16)	671	(44.53)	160	(0.57)	0.13	(0.76)
Education unclassifiable/missing	373	(19.84)	227	(60.86)	52	(0.52)	-0.33	(0.74)
P-value				<0.001		0.693		<0.001

*SD = standard deviation. P-values were computed using chi-squared tests for categorical variables and 2-sample t-tests for continuous variables. * ADHD symptoms at wave 4 were assessed by self-report only.*

	WechslerIntelli-gence Deviation Quotient (IQ)		**Concurrent ADHD symptoms***		**Concurrent family functioning**		**Concurrent aocial support by teachers**		**Concurrent social support by classmates**		**Concurrent age**	
	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)	Mean	(SD)
	97.58	(15.02)	0.55	(0.32)	3.36	(0.40)	349	(0.65)	3.59	(0.66)	13.57	(0.53)
	96.01	(13.44)	0.50	(0.33)	3.37	(0.39)	3.43	(0.69)	3.59	(0.63)	13.58	(0.53)
		0.138	0.067		0.802		0.216		0.882		0.608	
	99.75	(14.85)	0.51	(0.31)	3.35	(0.40)	3.43	(0.60)	3.57	(0.55)	16.20	(0.64)
	91.25	(13.22)	0.55	(0.33)	3.32	(0.40)	3.44	(0.65)	3.60	(0.62)	16.70	(0.90)
		<0.001	0.221		0.303		0.832		0.448		<0.001	
	100.34	(14.52)	0.45	(0.33)	3.33	(0.39)	-	-	-	-	19.03	(0.58)
	91.58	(13.13)	0.44	(0.34)	3.27	(0.41)	-	-	-	-	19.28	(0.64)
		<0.001	0.608		0.035		-	-	-	-	<0.001	

TABLE 5.S3 Direct and indirect effects of ADHD symptoms on (changes in) educational level two waves later, as well as selected estimates from mediator and outcome models in the TRAILS study (wave 1 – 4, the Netherlands, 2000–2010, N = 2,229); potential mediators were evaluated in separate models and measured one wave after ADHD symptoms; linear regression (standardized beta-coefficient, 95% Confidence Interval, p-value)

	Family functioning		
	IE	DE	PM
Direct and indirect effects			
Educational level around age 16	0.00 (-0.01 – 0.01), 0.480	**-0.18 (-0.21 – -0.14), <0.001**	0.01
Changes in educational level between around age 14 and 19	0.00 (-0.01 – 0.00), 0.259	**-0.11 (-0.14, -0.07), <0.001**	0.02
	Family functioning		
Mediator model estimates *(hypothesized mediators were measured one wave after ADHD symptoms)*			
ADHD symptoms around age 11	**-0.15 (-0.20 – -0.11), <0.001**		
ADHD symptoms around age 14	**-0.13 (-0.18 – -0.08), <0.001**		
	Educational level around age 16		
Outcome model estimates *(outcomes were measured two waves after ADHD symptoms and one wave after the hypothesized mediators)*			
Family functioning	0.02 (-0.02 – 0.05), 0.353		
Social support by teachers	**0.04 (0.01 – 0.07), 0.025**		
Social support by classmates	**0.05 (0.02 – 0.08), 0.005**		
ADHD × family functioning	0.00 (-0.03 – 0.03), 0.971		
ADHD × social support by teachers	0.00 (-0.03 – 0.03), 0.886		
ADHD × social support by classmates	0.00 (-0.03 – 0.04), 0.813		

IE = indirect effect; DE = direct effect; PM = proportion mediated. ***Boldface*** *denotes statistical significance at p<0.05. All models are adjusted for time-stable covariates measured at baseline (i.e., gender, ethnicity, parental SES) and age assessed in the same wave ADHD symptoms and potential mediators were measured. Past education, which is a potential exposure-induced mediator-outcome confounder (Chan & Leung, 2022; VanderWeele et al., 2014; Vansteelandt & Daniel, 2017), was treated as additional mediator.*

Social support by teachers			**Social support by classmates**		
IE	DE	PM	IE	DE	PM
-0.01 (-0.02 – 0.00), 0.078	**-0.17 (-0.21 – -0.14), <0.001**	0.04	**-0.01 (-0.02 – 0.00), 0.023**	**-0.17 (-0.20 – -0.13), <0.001**	0.05
-0.01 (-0.02 – 0.00), 0.055	**-0.10 (-0.13 -0.07), <0.001**	0.04	0.00 (-0.01 – 0.01), 0.809	**-0.11 (-0.14 – -0.08), <0.001**	0.01

Social support by teachers	Social support by classmates
-0.17 (-0.22 – -0.13), <0.001	**-0.18 (-0.23 – -0.14), <0.001**
-0.18 (-0.23 – -0.13), <0.001	**-0.17 (-0.22 – -0.12), <0.001**

Changes in educational level between around age 14 and 19
0.01 (-0.02 – 0.04), 0.562
0.04 (0.01 – 0.08), 0.006
0.01 (-0.02 – 0.04), 0.536
0.02 (-0.01 – 0.05), 0.310
0.00 (-0.03 – 0.02), 0.768
-0.02 (-0.04 – 0.01), 0.297

TABLE 5.S4 Associations of family functioning and social support by teachers and classmates with (changes in) education in each subsequent wave in the TRAILS study (wave 1 – 4, the Netherlands, 2000–2010, N = 2,229), adjusted for different sets of covariates; linear regression (standardized beta-coefficient, 95% Confidence Interval, p-value)

	Educational level age 14	**Changes in educational level between around age 14 and 16**	**Changes in educational level between around age 16 and 19**
Models for family functioning			
Model 1			
Family functioning	**0.16 (0.12 – 0.20), <0.001**	0.00 (-0.02 – 0.03), 0.749	0.03 (-0.00 – 0.06), 0.063
Past education	-	**0.85 (0.83 – 0.87), <0.001**	**0.87 (0.84 – 0.89), <0.001**
Model 2			
Family functioning	**0.04 (0.01 – 0.07), 0.014**	0.00 (-0.02 – 0.03), 0.793	0.02 (-0.01 – 0.05), 0.142
Past education	-	**0.78 (0.75 – 0.81), <0.001**	**0.77 (0.74 – 0.81), <0.001**
Model 3			
Family functioning	0.02 (-0.01 – 0.05), 0.269	-0.01 (-0.04 – 0.01), 0.286	0.01 (-0.02 – 0.04), 0.620
Past education	-	**0.76 (0.73 – 0.79), <0.001**	**0.75 (0.72 – 0.79), <0.001**
ADHD symptoms	**-0.14 (-0.17 – -0.11), <0.001**	**-0.09 (-0.11 – -0.06), <0.001**	**-0.08 (-0.11 – -0.05), <0.001**
ADHD × family functioning	-0.02 (-0.05 – 0.01), 0.123	0.01 (-0.01 – 0.03), 0.458	0.01 (-0.02 – 0.03), 0.688
Models for social support by teachers			
Model 1			
Social support by teachers	0.03 (-0.02 – 0.07), 0.215	**0.03 (0.01 – 0.05), 0.018**	**0.04 (0.01 – 0.06), 0.011**
Past education	-	**0.85 (0.83 – 0.87), <0.001**	**0.87 (0.84 – 0.89), <0.001**
Model 2			
Social support by teachers	**0.05 (0.02 – 0.08), 0.002**	**0.03 (0.01 – 0.06), 0.004**	**0.04 (0.02 – 0.07), 0.002**
Past education	-	**0.78 (0.75 – 0.81), <0.001**	**0.77 (0.74 – 0.81), <0.001**
Model 3			
Social support by teachers	0.02 (-0.01 – 0.05), 0.185	0.01 (-0.01 – 0.04), 0.244	0.02 (-0.01 – 0.05), 0.136
Past education	-	**0.76 (0.73 – 0.79), <0.001**	**0.75 (0.72 – 0.79), <0.001**
ADHD symptoms	**-0.14 (-0.17 – -0.10), <0.001**	**-0.08 (-0.11 – -0.06), <0.001**	**-0.08 (-0.11 – -0.04), <0.001**
ADHD × social support by teachers	0.01 (-0.02 – 0.04), 0.360	0.00 (-0.02 – 0.03), 0.703	-0.00 (-0.02 – 0.02), 0.973

TABLE 5.S4 Continued

	Educational level age 14	Changes in educational level between around age 14 and 16	Changes in educational level between around age 16 and 19
Models for social support by classmates			
Model 1			
Social support by classmates	**-0.05 (-0.10 -0.01), 0.017**	0.02 (-0.00 – 0.05), 0.051	0.00 (-0.03 – 0.03), 0.796
Past education	-	**0.85 (0.83 – 0.87), <0.001**	**0.87 (0.84 – 0.89), <0.001**
Model 2			
Social support by classmates	**0.04 (0.00 – 0.07), 0.028**	**0.02 (0.00 – 0.05), 0.038**	0.01 (-0.02 – 0.04), 0.587
Past education	-	**0.78 (0.75 – 0.81), <0.001**	**0.77 (0.74 – 0.81), <0.001**
Model 3			
Social support by classmates	0.01 (-0.02 – 0.05), 0.405	0.01 (-0.01 – 0.03), 0.424	-0.00 (-0.03 – 0.03), 0.867
Past education	-	**0.76 (0.73 – 0.79), <0.001**	**0.75 (0.72 – 0.79), <0.001**
ADHD symptoms	**-0.14 (-0.17 – -0.10), <0.001**	**-0.09 (-0.11 – -0.06), <0.001**	**-0.08 (-0.11 – -0.05), <0.001**
ADHD × social support by classmates	0.02 (-0.01 – 0.06), 0.108	0.00 (-0.02 – 0.02), 0.841	-0.00 (-0.03 – 0.02), 0.793

Boldface *denotes statistical significance at p<0.05. Coefficients in Models 2 and 3 are additionally adjusted for time-stable covariates measured at baseline (i.e., gender, ethnicity, IQ, parental SES) and age assessed in the same wave ADHD symptoms and potential mediators were measured.*

SUPPLEMENTARY MATERIALS – CHAPTER 6

TABLE 6.S1 Description of the European Social Survey-International Standard Classification of Education (ES-ISCED) (Schneider, 2020)

7-category ES-ISCED scale			Recoded 5-category version	
1	ES-ISCED I	Less than lower secondary education	1	Up to lower secondary education
2	ES-ISCED II	Lower secondary education		
3	ES-ISCED IIIb	Lower tier upper secondary education	2	Upper secondary education
4	ES-ISCED IIIa	Upper tier upper secondary education		
5	ES-ISCED IV	Advanced vocational education, sub-degree	3	Advanced vocational education, sub-degree
6	ES-ISCED V1	Lower tertiary education, Bachelor's level	4	Lower tertiary education, Bachelor's level
7	ES-ISCED V2	Higher tertiary education, Master's level or higher	5	Higher tertiary education, Master's level or higher

A

TABLE 6.S2: Stepwise multilevel linear regression models for adolescents' moderate-to-vigorous physical activity (MVPA) with country-level predictors and cross-level interaction terms between country-level predictors and individual-level family affluence in the HBSC study (2017/2018, N participants = 185,086, N countries = 32)

	Model 0			**Model 1**		
	N = 173,580			**N = 173,580**		
	B	(SE)	P-value	B	(SE)	P-value
Individual-level						
Male gender	-	-	-	**0.50**	**(0.03)**	**<0.001**
Age	-	-	-	**-0.17**	**(0.02)**	**<0.001**
Family affluence	-	-	-	**0.98**	**(0.04)**	**<0.001**
(Residual) variance in MVPA at the individual level	**4.05**	**(0.07)**	**<0.001**	**3.84**	**(0.07)**	**<0.001**
Country-level						
Social mobility	-	-	-	-	-	-
GINI	-	-	-	-	-	-
GNI	-	-	-	-	-	-
Intercept	**4.10**	**(0.07)**	**<0.001**	**4.10**	**(0.07)**	**<0.001**
(Residual) variance in MVPA at the country level	**0.17**	**(0.05)**	**<0.001**	**0.17**	**(0.05)**	**<0.001**
(Residual) variance in the slope of family affluence	-	-	-	-	-	-
Cross-level interactions						
Family affluence × social mobility	-	-	-	-	-	-
Family affluence × GINI	-	-	-	-	-	-
Family affluence × GNI	-	-	-	-	-	-
Intraclass correlation coefficient	3.9%			3.9%		
Model fit information						
Free parameter	3			6		
AIC	735742.108			726179.784		
BIC	735772.301			726240.171		

Model 0: intercept-only model, Model 1: multilevel model with individual-level predictors, Model 2: multilevel model with individual-level and country-level predictors, Model 3: multilevel model with individual-level and country-level predictors and random slope for family affluence, Model 4: multilevel model with individual-level and country-level predictors, random slope for family affluence, and cross-level interactions.
B = unstandardized linear regression coefficient; SD = standard deviation; AIC = Akaike information criterion; BIC = Bayesian information criterion; MVPA = moderate-to-vigorous physical activity.

	Model 2			**Model 3**			**Model 4**		
	N = 173,580			**N = 173,580**			**N = 173,580**		
	B	(SE)	P-value	B	(SE)	P-value	B	(SE)	P-value
	0.50	**(0.03)**	**<0.001**	**0.50**	**(0.03)**	**<0.001**	**0.50**	**(0.03)**	**<0.001**
	-0.17	**(0.02)**	**<0.001**	**-0.17**	**(0.02)**	**<0.001**	**-0.17**	**(0.02)**	**<0.001**
	0.98	**(0.04)**	**<0.001**	**0.93**	**(0.04)**	**<0.001**	**0.93**	**(0.04)**	**<0.001**
	3.84	**(0.07)**	**<0.001**	**3.83**	**(0.07)**	**<0.001**	**3.83**	**(0.07)**	**<0.001**
	0.10	(0.23)	0.671	0.45	(0.24)	0.062	0.10	(0.23)	0.670
	-3.86	**(1.75)**	**0.027**	**-3.73**	**(1.30)**	**0.004**	**-3.87**	**(1.75)**	**0.027**
	-0.08	(0.07)	0.297	-0.09	(0.06)	0.168	-0.08	(0.07)	0.298
	4.10	**(0.07)**	**<0.001**	**4.01**	**(0.07)**	**<0.001**	**4.10**	**(0.07)**	**<0.001**
	0.15	**(0.03)**	**<0.001**	**0.15**	**(0.04)**	**<0.001**	**0.15**	**(0.03)**	**<0.001**
	-	-	-	**0.04**	**(0.01)**	**<0.001**	**0.04**	**(0.01)**	**0.002**
	-	-	-	-	-	-	**0.34**	**(0.13)**	**0.009**
	-	-	-	-	-	-	0.12	(1.22)	0.921
	-	-	-	-	-	-	-0.01	(0.05)	0.892
	3.9%			3.9%			3.9%		
	9			11			14		
	726181.916			726073.331			726074.155		
	726272.496			726184.039			726215.056		

TABLE 6.S3 Stepwise multilevel linear regression models for adolescents' vigorous physical activity (VPA) with country-level predictors and cross-level interaction terms between country-level predictors and individual-level family affluence in the HBSC study (2017/2018, N participants = 185,086, N countries = 32)

	Model 0			**Model 1**		
	N = 174,119			**N = 174,119**		
	B	(SE)	P-value	B	(SE)	P-value
Individual-level						
Gender	-	-	-	**0.44**	**(0.04)**	**<0.001**
Age	-	-	-	**-0.12**	**(0.01)**	**<0.001**
Family affluence	-	-	-	**0.73**	**(0.04)**	**<0.001**
(Residual) variance in VPA at the individual level	**2.46**	**(0.08)**	**<0.001**	**2.33**	**(0.08)**	**<0.001**
Country-level						
Social mobility	-	-	-	-	-	-
GINI	-	-	-	-	-	-
GNI	-	-	-	-	-	-
Intercept	**4.03**	**(0.04)**	**<0.001**	**4.03**	**(0.04)**	**<0.001**
(Residual) variance in VPA at the country level	**0.06**	**(0.01)**	**<0.001**	**0.06**	**(0.01)**	**<0.001**
(Residual) variance in the slope of family affluence	-	-	-	-	-	-
Cross-level interactions						
Family affluence × social mobility	-	-	-	-	-	-
Family affluence × GINI	-	-	-	-	-	-
Family affluence × GNI	-	-	-	-	-	-
Intraclass correlation coefficient	2.4%			2.4%		
Model fit information						
Free parameter	3			6		
AIC	651038.901			641506.281		
BIC	651069.103			641566.686		

Model 0: intercept-only model, Model 1: multilevel model with individual-level predictors, Model 2: multilevel model with individual-level and country-level predictors, Model 3: multilevel model with individual-level and country-level predictors and random slope for family affluence, Model 4: multilevel model with individual-level and country-level predictors, random slope for family affluence, and cross-level interactions.
B = unstandardized linear regression coefficient; SD = standard deviation; AIC = Akaike information criterion; BIC = Bayesian information criterion; VPA = vigorous physical activity.

	Model 2			Model 3			Model 4		
	N = 174,119			N = 174,119			N = 174,119		
	B	(SE)	P-value	B	(SE)	P-value	B	(SE)	P-value
	0.44	**(0.04)**	**<0.001**	**0.44**	**(0.04)**	**<0.001**	**0.44**	**(0.04)**	**<0.001**
	-0.12	**(0.01)**	**<0.001**	**-0.12**	**(0.01)**	**<0.001**	**-0.12**	**(0.01)**	**<0.001**
	0.73	**(0.04)**	**<0.001**	**0.68**	**(0.04)**	**<0.001**	**0.68**	**(0.03)**	**<0.001**
	2.33	**(0.08)**	**<0.001**	**2.33**	**(0.08)**	**<0.001**	**2.33**	**(0.08)**	**<0.001**
	-0.06	(0.12)	0.623	0.17	(0.11)	0.107	-0.06	(0.12)	0.628
	-2.07	(1.07)	0.053	**-2.39**	**(0.84)**	**0.004**	-2.08	(1.07)	0.053
	0.01	(0.04)	0.829	0.02	(0.04)	0.549	0.01	(0.04)	0.829
	4.03	**(0.04)**	**<0.001**	**4.03**	**(0.04)**	**<0.001**	**4.03**	**(0.04)**	**<0.001**
	0.05	**(0.01)**	**<0.001**	**0.06**	**(0.01)**	**<0.001**	**0.05**	**(0.01)**	**<0.001**
	-	-	-	**0.04**	**(0.01)**	**<0.001**	**0.03**	**(0.01)**	**0.004**
	-	-	-	-	-	-	**0.31**	**(0.10)**	**0.002**
	-	-	-	-	-	-	-0.44	(0.89)	0.621
	-	-	-	-	-	-	0.02	(0.04)	0.586
	2.4%			2.4%			2.4%		
	9			11			14		
	641508.169			641338.786			641336.731		
	641598.776			641449.528			641477.676		

TABLE 6.S4: Stepwise multilevel linear regression models for adolescents' consumption of healthy foods with country-level predictors and cross-level interaction terms between country-level predictors and individual-level family affluence in the HBSC study (2017/2018, N participants = 185,086, N countries = 32)

	Model 0			**Model 1**		
	N = 172,240			**N = 172,240**		
	B	(SE)	P-value	B	(SE)	P-value
Individual-level						
Male gender	-	-	-	**-0.54**	**(0.03)**	**<0.001**
Age	-	-	-	**-0.16**	**(0.01)**	**<0.001**
Family affluence	-	-	-	**1.34**	**(0.11)**	**<0.001**
(Residual) variance in fruit/vegetable consumption at the individual level	**8.05**	**(0.23)**	**<0.001**	**7.76**	**(0.21)**	**<0.001**
Country-level						
Social mobility	-	-	-	-	-	-
GINI	-	-	-	-	-	-
GNI	-	-	-	-	-	-
Intercept	**7.64**	**(0.08)**	**<0.001**	**7.64**	**(0.08)**	**<0.001**
(Residual) variance in fruit/vegetable consumption at the country level	**0.18**	**(0.05)**	**<0.001**	**0.18**	**(0.05)**	**<0.001**
(Residual) variance in the slope of family affluence	-	-	-	-	-	-
Cross-level interactions						
Family affluence × social mobility	-	-	-	-	-	-
Family affluence × GINI	-	-	-	-	-	-
Family affluence × GNI	-	-	-	-	-	-
Intraclass correlation coefficient	2.2%			2.2%		
Model fit information						
Free parameter	3			6		
AIC	848111.451			841902.232		
BIC	848141.621			841962.572		

Model 0: intercept-only model, Model 1: multilevel model with individual-level predictors, Model 2: multilevel model with individual-level and country-level predictors, Model 3: multilevel model with individual-level and country-level predictors and random slope for family affluence, Model 4: multilevel model with individual-level and country-level predictors, random slope for family affluence, and cross-level interactions.
B = unstandardized linear regression coefficient; SD = standard deviation; AIC = Akaike information criterion; BIC = Bayesian information criterion.

	Model 2			Model 3			Model 4		
	N = 172,240			N = 172,240			N = 172,240		
	B	(SE)	P-value	B	(SE)	P-value	B	(SE)	P-value
	-0.54	**(0.03)**	**<0.001**	**-0.54**	**(0.03)**	**<0.001**	**-0.54**	**(0.03)**	**<0.001**
	-0.16	**(0.01)**	**<0.001**	**-0.16**	**(0.01)**	**<0.001**	**-0.16**	**(0.01)**	**<0.001**
	1.34	**(0.11)**	**<0.001**	**1.25**	**(0.06)**	**<0.001**	**1.25**	**(0.06)**	**<0.001**
	7.76	**(0.21)**	**<0.001**	**7.75**	**(0.21)**	**<0.001**	**7.75**	**(0.21)**	**<0.001**
	-0.01	(0.26)	0.959	0.02	(0.26)	0.929	-0.01	(0.26)	0.957
	-0.08	(2.57)	0.974	0.10	(2.57)	0.971	-0.12	(2.57)	0.964
	0.00	(0.10)	0.999	-0.01	(0.10)	0.946	0.00	(0.09)	0.999
	7.64	**(0.08)**	**<0.001**	**7.64**	**(0.08)**	**<0.001**	**7.64**	**(0.08)**	**<0.001**
	0.18	**(0.05)**	**<0.001**	**0.18**	**(0.05)**	**<0.001**	**0.18**	**(0.05)**	**<0.001**
	-	-	-	**0.09**	**(0.03)**	**0.004**	**0.08**	**(0.03)**	**0.005**
	-	-	-	-	-	-	0.17	(0.25)	0.481
	-	-	-	-	-	-	1.04	(1.37)	0.446
	-	-	-	-	-	-	-0.03	(0.05)	0.549
	2.2%			2.2%			2.2%		
	9			11			14		
	841908.227			841737.316			841741.993		
	841998.736			841847.939			841882.786		

TABLE 6.S5 Stepwise multilevel linear regression models for adolescents' consumption of unhealthy foods with country-level predictors and cross-level interaction terms between country-level predictors and individual-level family affluence in the HBSC study (2017/2018, N participants = 185,086, N countries = 32)

	Model 0			**Model 1**		
	N = 172,224			**N = 172,224**		
	B	(SE)	P-value	B	(SE)	P-value
Individual-level						
Male gender	-	-	-	**0.30**	**(0.03)**	**<0.001**
Age	-	-	-	**0.12**	**(0.01)**	**<0.001**
Family affluence	-	-	-	**-0.23**	**(0.08)**	**0.004**
(Residual) variance in soft drinks/sweets consumption at the individual level	**7.32**	**(0.28)**	**<0.001**	**7.26**	**(0.28)**	**<0.001**
Country-level						
Social mobility	-	-	-	-	-	-
GINI	-	-	-	-	-	-
GNI	-	-	-	-	-	-
Intercept	**5.49**	**(0.12)**	**<0.001**	**5.49**	**(0.12)**	**<0.001**
(Residual) variance in soft drinks/sweets consumption at the country level	**0.49**	**(0.11)**	**<0.001**	**0.49**	**(0.11)**	**<0.001**
(Residual) variance in the slope of family affluence	-	-	-	-	-	-
Cross-level interactions						
Family affluence × social mobility	-	-	-	-	-	-
Family affluence × GINI	-	-	-	-	-	-
Family affluence × GNI	-	-	-	-	-	-
Intraclass correlation coefficient	6.3%			6.3%		
Model fit information						
Free parameter	3			6		
AIC	831813.427			830275.027		
BIC	831843.596			830335.366		

Model 0: intercept-only model, Model 1: multilevel model with individual-level predictors, Model 2: multilevel model with individual-level and country-level predictors, Model 3: multilevel model with individual-level and country-level predictors and random slope for family affluence, Model 4: multilevel model with individual-level and country-level predictors, random slope for family affluence, and cross-level interactions. B = unstandardized linear regression coefficient; SD = standard deviation; AIC = Akaike information criterion; BIC = Bayesian information criterion.

	Model 2			**Model 3**			**Model 4**		
	N = 172,224			**N = 172,224**			**N = 172,224**		
	B	(SE)	P-value	B	(SE)	P-value	B	(SE)	P-value
	0.30	**(0.03)**	**<0.001**	**0.30**	**(0.03)**	**<0.001**	**0.30**	**(0.03)**	**<0.001**
	0.12	**(0.01)**	**<0.001**	**0.12**	**(0.01)**	**<0.001**	**0.12**	**(0.01)**	**<0.001**
	-0.23	**(0.08)**	**0.004**	**-0.19**	**(0.08)**	**0.013**	**-0.19**	**(0.07)**	**0.006**
	7.26	**(0.28)**	**<0.001**	**7.24**	**(0.28)**	**<0.001**	**7.24**	**(0.28)**	**<0.001**
	-1.16	**(0.37)**	**0.002**	**-1.05**	**(0.45)**	**0.020**	**-1.16**	**(0.37)**	**0.002**
	5.47	(2.86)	0.056	5.04	(3.00)	0.093	5.47	(2.86)	0.056
	-0.06	(0.12)	0.626	-0.10	(0.14)	0.469	-0.06	(0.12)	0.626
	5.49	**(0.01)**	**<0.001**	**5.49**	**(0.10)**	**<0.001**	**5.49**	**(0.10)**	**<0.001**
	0.31	**(0.07)**	**<0.001**	**0.31**	**(0.07)**	**<0.001**	**0.31**	**(0.07)**	**<0.001**
	-	-	-	**0.16**	**(0.05)**	**<0.001**	**0.13**	**(0.03)**	**<0.001**
	-	-	-	-	-	-	0.47	(0.31)	0.134
	-	-	-	-	-	-	-1.87	(1.94)	0.335
	-	-	-	-	-	-	-0.22	(0.12)	0.061
	6.3%			6.3%			6.3%		
	9			11			14		
	830266.587			830038.657			830038.178		
	830357.096			830149.279			830178.970		

TABLE 6.S6 Stepwise multilevel linear regression models for adolescents' breakfast consumption[a] with country-level predictors and cross-level interaction terms between country-level predictors and individual-level family affluence in the HBSC study (2017/2018, N participants = 185,086, N countries = 32)

	Model 0			**Model 1**		
	N = 163,658			**N = 163,658**		
	B	(SE)	P-value	B	(SE)	P-value
Individual-level						
Gender	-	-	-	**0.31**	**(0.05)**	**<0.001**
Age	-	-	-	**-0.21**	**(0.02)**	**<0.001**
Family-affluence	-	-	-	**0.73**	**(0.09)**	**<0.001**
(Residual) variance in breakfast days at the individual level	**4.96**	**(0.19)**	**<0.001**	**4.78**	**(0.16)**	**<0.001**
Country-level						
Social mobility	-	-	-	-	-	-
GINI	-	-	-	-	-	-
GNI	-	-	-	-	-	-
Intercept	**5.28**	**(0.08)**	**<0.001**	**5.28**	**(0.08)**	**<0.001**
(Residual) variance in breakfast days at the country level	**0.21**	**(0.04)**	**<0.001**	**0.21**	**(0.04)**	**<0.001**
(Residual) variance in the slope of family affluence	-	-	-	-	-	-
Cross-level interactions						
Family affluence × social mobility	-	-	-	-	-	-
Family affluence × GINI	-	-	-	-	-	-
Family affluence × GNI	-	-	-	-	-	-
Intraclass correlation coefficient	4.0%			4.0%		
Model fit information						
Free parameter	3			6		
AIC	726720.500			720704.106		
BIC	726750.517			720764.139		

Model 0: intercept-only model, Model 1: multilevel model with individual-level predictors, Model 2: multilevel model with individual-level and country-level predictors, Model 3: multilevel model with individual-level and country-level predictors and random slope for family affluence, Model 4: multilevel model with individual-level and country-level predictors, random slope for family affluence, and cross-level interactions.
B = unstandardized linear regression coefficient; SD = standard deviation; AIC = Akaike information criterion; BIC = Bayesian information criterion. [a] Data on breakfast consumption is missing for Slovakia. Therefore, this analysis is based on 31 countries.

	Model 2			Model 3			Model 4		
	N = 163,658			N = 163,658			N = 163,658		
	B	(SE)	P-value	B	(SE)	P-value	B	(SE)	P-value
	0.31	**(0.05)**	**<0.001**	**0.31**	**(0.05)**	**<0.001**	**0.31**	**(0.05)**	**<0.001**
	-0.21	**(0.02)**	**<0.001**	**-0.21**	**(0.02)**	**<0.001**	**-0.21**	**(0.02)**	**<0.001**
	0.73	**(0.09)**	**<0.001**	**0.64**	**(0.06)**	**<0.001**	**0.64**	**(0.06)**	**<0.001**
	4.78	**(0.16)**	**<0.001**	**4.77**	**(0.16)**	**<0.001**	**4.77**	**(0.16)**	**<0.001**
	0.85	**(0.32)**	**0.008**	**0.87**	**(0.33)**	**0.008**	**0.85**	**(0.32)**	**0.008**
	-3.00	(1.70)	0.076	**-3.16**	**(1.61)**	**0.049**	-3.00	(1.70)	0.076
	-0.01	(0.07)	0.892	0.00	(0.07)	0.971	-0.01	(0.07)	0.892
	5.28	**(0.07)**	**<0.001**	**5.28**	**(0.07)**	**<0.001**	**5.28**	**(0.07)**	**<0.001**
	0.14	**(0.03)**	**<0.001**	**0.14**	**(0.03)**	**<0.001**	**0.14**	**(0.03)**	**<0.001**
	-	-	-	**0.10**	**(0.03)**	**<0.001**	**0.09**	**(0.02)**	**<0.001**
	-	-	-	-	-	-	0.09	(0.18)	0.619
	-	-	-	-	-	-	-0.75	(1.79)	0.673
	-	-	-	-	-	-	0.06	(0.06)	0.322
	4.0%			4.0%			4.0%		
	9			11			14		
	720697.973			720410.319			720414.265		
	720788.023			720520.380			720554.343		

TABLE 6.S7 Stepwise multilevel logistic regression models for adolescents' odds of weekly smoking with country-level predictors and cross-level interaction terms between country-level predictors and individual-level family affluence in the HBSC study (2017/2018, N participants = 185,086, N countries = 32)

	Model 1				**Model 2**			
	N = 168,271				**N = 168,271**			
	B	(SE)	OR	P-value	B	(SE)	OR	P-value
Individual-level								
Male gender	0.04	(0.07)	1.04	0.528	0.05	(0.07)	1.05	0.525
Age	**0.70**	**(0.03)**	**2.01**	**<0.001**	**0.70**	**(0.03)**	2.01	**<0.001**
Family affluence	**-0.29**	**(0.08)**	**0.75**	**<0.001**	**-0.29**	**(0.08)**	0.75	**<0.001**
Country-level								
Social mobility	-	-	-	-	**-0.58**	**(0.14)**	**0.56**	**<0.001**
GINI	-	-	-	-	2.99	(1.74)	19.93	0.085
GNI	-	-	-	-	**-0.16**	**(0.05)**	0.85	**0.001**
Threshold	**3.53**	**(0.09)**	**34.12**	**<0.001**	**3.53**	**(0.07)**	**34.06**	**<0.001**
(Residual) variance in smoking at the country level	**0.20**	**(0.05)**	**1.22**	**<0.001**	**0.09**	**(0.03)**	**1.10**	**0.001**
(Residual) variance in the slope of family affluence	-	-	-	-	-	-	-	-
Cross-level interactions								
Family affluence × social mobility	-	-	-	-	-	-	-	-
Family affluence × GINI	-	-	-	-	-	-	-	-
Family affluence × GNI	-	-	-	-	-	-	-	-
Model fit information								
Free parameter	5				8			
AIC	56075.348				56059.302			
BIC	56125.515				56139.568			

Model 0: intercept-only model, Model 1: multilevel model with individual-level predictors, Model 2: multilevel model with individual-level and country-level predictors, Model 3: multilevel model with individual-level and country-level predictors and random slope for family affluence, Model 4: multilevel model with individual-level and country-level predictors, random slope for family affluence, and cross-level interactions. B = unstandardized logistic regression coefficient; SD = standard deviation; OR = odds ratio; AIC = Akaike information criterion; BIC = Bayesian information criterion.

	Model 3				**Model 4**			
	N = 168,271				**N = 168,271**			
	B	(SE)	OR	P-value	B	(SE)	OR	P-value
	0.05	(0.07)	1.05	0.525	0.05	(0.07)	1.05	0.525
	0.70	**(0.03)**	**2.01**	**<0.001**	**0.70**	**(0.03)**	**2.01**	**<0.001**
	-0.28	**(0.06)**	**0.76**	**<0.001**	**-0.29**	**(0.06)**	**0.75**	**<0.001**
	-0.52	**(0.14)**	**0.60**	**<0.001**	**-0.57**	**(0.15)**	**0.56**	**<0.001**
	2.55	(1.66)	12.81	0.125	2.87	(1.74)	17.67	0.100
	-0.17	**(0.05)**	**0.85**	**<0.001**	**-0.17**	**(0.05)**	**0.84**	**0.001**
	3.53	**(0.07)**	**34.19**	**<0.001**	**3.53**	**(0.07)**	**34.02**	**<0.001**
	0.09	**(0.03)**	**1.10**	**0.001**	**0.09**	**(0.03)**	**1.10**	**0.001**
	0.06	**(0.02)**	**1.06**	**0.004**	**0.04**	**(0.02)**	**1.04**	**0.023**
	-	-	-	-	-0.31	(0.25)	0.74	0.219
	-	-	-	-	1.89	(1.50)	6.64	0.208
	-	-	-	-	-0.03	(0.08)	0.97	0.714
	10				13			
	56044.288				56046.076			
	56144.621				56176.509			

A

TABLE S8 Missing information on individual level variables in the HBSC study (2017/2018, N participants = 185,086, N countries = 32)

	N participants with complete information	N participants with missing information	% missing
Age	184,037	1,049	0.57
Gender	185,086	0	0.00
Family affluence	177,551	7,535	4.07
MVPA	181,247	3,839	2.07
VPA	181,297	3,789	2.05
Healthy foods consumption	180,168	4,918	2.66
Unhealthy foods consumption	180,144	4,942	2.67
Days per week with breakfast[a]	170,043	15,043	8.13
Days per week with breakfast (without counting Slovaks as missing)	170,043	10,258	5.69
Weekly smoking	175,809	9,277	5.01

MVPA = moderate-to-vigorous physical activity; VPA = vigorous physical activity.
[a] *data on breakfast consumption is missing for Slovakia.*

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BIOGRAPHY

Heiko Schmengler was born in Linsengericht, Germany, on 7th May 1992. After finishing his secondary education at Max-von-Laue-Gymnasium in Koblenz (Germany) in 2012, he followed an interdisciplinary bachelor's programme focussing on biomedical and health sciences (BSc in Liberal Arts and Sciences) at Amsterdam University College, the joint honours college of the University of Amsterdam and VU University Amsterdam (the Netherlands). In 2014, he went on a student exchange to the University of Melbourne (Australia), where he discovered his passion for public health and the study of health inequalities. To further his interests, he engaged in a short internship in the field of sexual minority health at the Melbourne School of Population and Global Health, followed by dissertation research on ethnic inequalities in obesity based at the Department of Public Health at the Academic Medical Center in Amsterdam. After completing his BSc *cum laude* in 2015, Heiko followed a double master's degree in public health (Master of Public Health) at the University of Sheffield (UK) and EHESP French School of Public Health in Paris (France) as part of the Europubhealth+ programme, an Erasmus Mundus Joint Master's programme of the European Commission. He completed his master's degree in 2018 *with Distinction (mention très bien)*. His dissertation research focussed on the neurodevelopmental health of young children of immigrant mothers in France and was based at the Institut Pierre Louis d'Epidémiologie et de Santé Publique (Inserm UMR_S 1136) in Paris. From 2018 until 2023, Heiko was PhD Candidate at the Department of Interdisciplinary Social Science at Utrecht University (the Netherlands), where he investigated the mechanisms underlying the development of socioeconomic health inequalities in adolescence and young adulthood. Heiko now works as a postdoctoral researcher at the same department to study the role of stress physiological mechanisms in associations between peer stressors and adolescent substance use.

Heiko has published his research in high-impact journals covering a diverse range of disciplines, including public health, neurodevelopment, and child psychiatry. During his PhD, he also presented at many international conferences, both in-person and online, including the European Public Health Conference (2019, 2021, 2022), the World Congress on Public Health (2020, 2023), the World Congress on Adolescent Health (2021), the World Congress of Epidemiology (2021), and the International Convention of Psychological Science (2023). In addition, he was seminar tutor for two different modules of the Bachelors' programme in Interdisciplinary Social Science at Utrecht University

SCIENTIFIC PUBLICATIONS

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