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Intergenerational transmission of health inequalities: towards a life course approach to socioeconomic inequalities in health – a review

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ABSTRACT

Adult health inequalities are a persistent public health problem. Explanations are usually sought in behaviours and environments in adulthood, despite evidence on the importance of early life conditions for life course outcomes. We review evidence from a broad range of fields to unravel to what extent, and how, socioeconomic health inequalities are intergenerationally transmitted. We find that transmission of socioeconomic and associated health (dis)advantages from parents to offspring, and its underlying structural determinants, contributes substantially to socioeconomic inequalities in adult health. In the first two decades of life—from conception to early adulthood—parental socioeconomic position (SEP) and parental health strongly influence offspring adult SEP and health. Socioeconomic and health (dis)advantages are largely transmitted through the same broad mechanisms. Socioeconomic inequalities in the fetal environment contribute to inequalities in fetal development and birth outcomes, with lifelong socioeconomic and health consequences. Inequalities in the postnatal environment—especially the psychosocial and learning environment, physical exposures and socialisation—result in inequalities in child and adolescent health, development and behavioural habits, with health and socioeconomic consequences tracking into adulthood. Structural factors shape these mechanisms in a socioeconomically patterned and time-specific and place-specific way, leading to distinct birth-cohort patterns in health inequality. Adult health inequalities are for an important part intergenerationally transmitted. Effective health inequality reduction requires addressing intergenerational transmission of (dis)advantage by creating societal circumstances that allow all children to develop to their full potential.

INTRODUCTION

Socioeconomic health inequalities are a persistent public health problem. Even in welfare states, low socioeconomic position (SEP) remains associated with worse health outcomes.¹ Several factors may contribute to this.² An important factor that has received insufficient attention is intergenerational transmission.

What is the role of intergenerational transmission in explaining health inequalities? Socioeconomic health inequalities have been observed across birth cohorts for many health outcomes.¹ However, this does not necessarily imply intergenerational transmission. Adult health inequalities could possibly

WHAT IS ALREADY KNOWN ON THIS TOPIC

- ⇒ There is a long research tradition of quantifying and explaining socioeconomic inequalities in adult health. Despite growing evidence on the importance of early life factors for adult health and social outcomes, explanations for adult health inequalities are usually sought in adulthood.
- ⇒ To what extent are adult health inequalities the consequence of early life inequalities? We critically discuss the empirical evidence on early life inequalities and how they contribute to adult health inequalities.

WHAT THIS STUDY ADDS

- ⇒ Socioeconomic inequalities in adult health can for an important part be explained by inequalities in the first two decades of life.
- ⇒ Broadly the same mechanisms in early life strongly influence both adult health and adult SEP.
- ⇒ Structural factors shape inequalities in the fetal and postnatal environment and contribute to birth cohort-specific patterns in adult health inequality.

HOW THIS STUDY MIGHT AFFECT RESEARCH, PRACTICE OR POLICY

- ⇒ Socioeconomic health inequalities can be effectively reduced by providing supportive environments to (expectant) families.

also arise out of factors in adulthood that are largely independent of early life (dis)advantages. Yet, there are good reasons to believe that intergenerational transmission of health inequalities is important or, in other words, that transmission of socioeconomic and associated health (dis)advantages from parents to offspring—through the in utero environment and postnatal environment and the underlying structural drivers of socioeconomic inequalities in these environments—contributes to socioeconomic inequalities in adult health.

First, parental and offspring relative positions in the social hierarchy are closely related, as observed for educational attainment, income, wealth, and occupational class—although with time and place variations (figure 1).^{3–5} For intergenerational transmission of health inequalities to occur, it is sufficient that SEP is intergenerationally transmitted, provided that adult SEP influences adult health.



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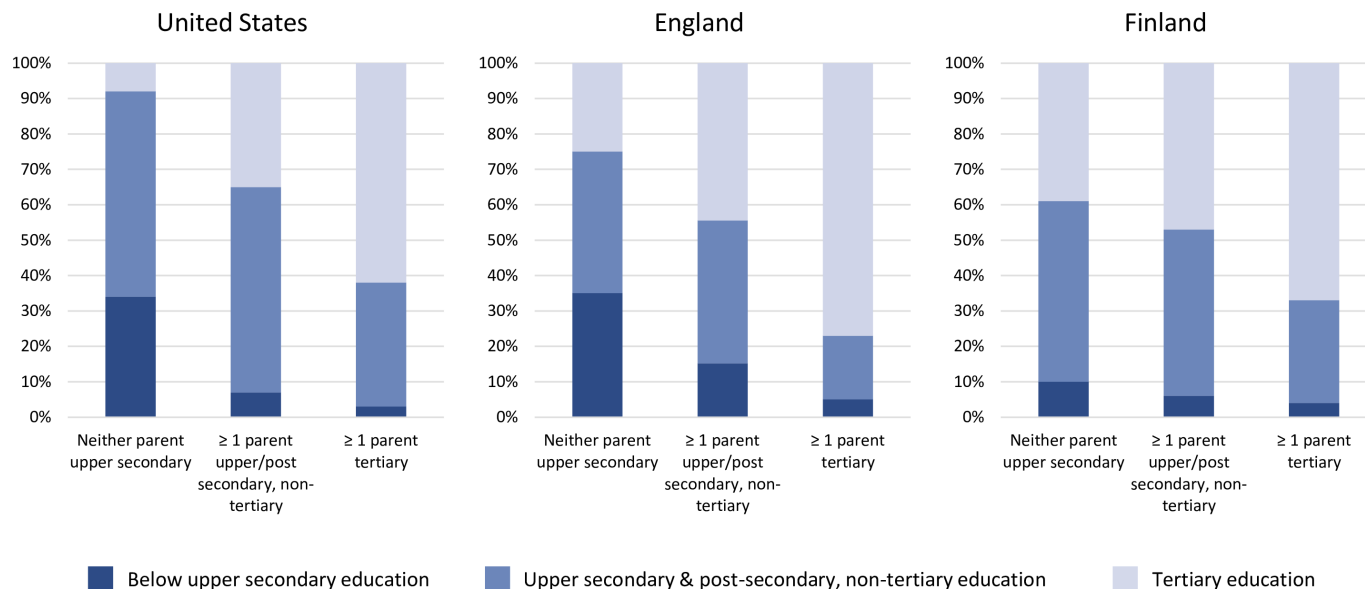


Figure 1 Educational attainment (25–44 years) by parental education, 2012. Figure based on OECD data; data relate to men and women combined.⁵

Second, many health determinants and health problems associated with SEP are intergenerationally transmitted, through latent, cumulative and pathway effects of early life (dis)advantage.⁶ Parents of low SEP, on average, live in less healthy environments, exhibit less healthy behaviours and have worse health status than better-off parents.¹ By shaping fetal, child and adolescent health and development, and childhood and adolescent socialisation, these disadvantages—and their underlying structural determinants—affect offspring health determinants and health outcomes as well as SEP.⁷

Indeed, studies have reported persistent relationships between parental SEP and offspring adult health, partly mediated by offspring SEP, with the relative contributions of parental and offspring SEP varying between outcomes.^{8–10} While these studies do not directly answer the methodologically more complex question of what proportion of adult health inequalities is explained by parental SEP and how this proportion varies across contexts, they are indicative of the importance of parental SEP for understanding adult health inequalities.

Recent advances in neuroscience, genetics and research on the fetal environment—when combined with evidence from epidemiology, sociology, economics, developmental psychology and education sciences—can further our understanding of the intergenerational transmission of health inequalities. Our paper builds on earlier work^{6 11–13} and seeks to bridge disciplinary gaps by bringing together these disparate lines in the literature to answer the question of the extent to which, and the mechanisms through which, socioeconomic health inequalities are intergenerationally transmitted. We discuss the fetal and postnatal environment as main ‘channels’ through which SEP and associated health (dis)advantages are transmitted from parents to children. We also discuss the role of genetics. We conclude with a section on the important role of structural drivers of these transmission channels and policy implications. Figure 2 illustrates our findings. In a companion paper, we propose an analytical framework and agenda for research on the intergenerational transmission of health inequalities.¹⁴

FETAL ENVIRONMENT

The fetal environment is a channel through which health inequalities are transmitted intergenerationally. Research on the developmental origins of health and disease in low-income countries¹⁵ and historical and contemporary cohorts in high-income countries shows that the fetal environment influences adult health and SEP.^{16 17} The fetal environment is influenced by circumstances before and during pregnancy. For example, there is evidence that lifetime trauma and history of maternal childhood trauma affect fetal development via dysregulation of the maternal stress response system and may render exposed infants more vulnerable to future illness.^{18 19} Studies on the Dutch World War II famine have shown that undernutrition in pregnancy can lead to an increased risk of cardiovascular disease (CVD) in offspring,²⁰ with effects possibly spanning multiple generations.^{21 22}

In contemporary high-income countries, women of low SEP are more likely to be overweight/obese and to smoke in the preconception period than women of high SEP.^{23 24} During pregnancy, they are more likely to continue smoking,²⁵ and if they smoke, they usually smoke more.²⁶ They are also more likely to be exposed to passive smoking,²⁷ suffer from gestational hypertension,²⁴ mental health problems²⁸ and unhealthy weight gain during pregnancy,²⁹ and are less likely to, in a timely manner, take folic acid supplementation (fortification of grain flour is currently uncommon in European countries).³⁰ Inequalities in such risk factors are substantial. Over 43% of lower-educated pregnant women in the generation R cohort (Netherlands) smoked during pregnancy, compared with 5% of high-educated women.²⁵ In France, only 23% of women with primary education took folic acid supplementation, compared with 53% of women with a master’s degree.³⁰

These risk factors have important effects on perinatal health, including fetal growth, brain development,^{31–35} preterm birth³¹ and low birth weight¹⁹ (due to intrauterine growth restriction and/or preterm birth). Even in the first weeks of pregnancy, embryos are smaller when mothers smoke and do not take folic acid supplementation.³⁶ Fetal growth is more heavily restricted when mothers smoke more (dose-response relationship).^{32 37}

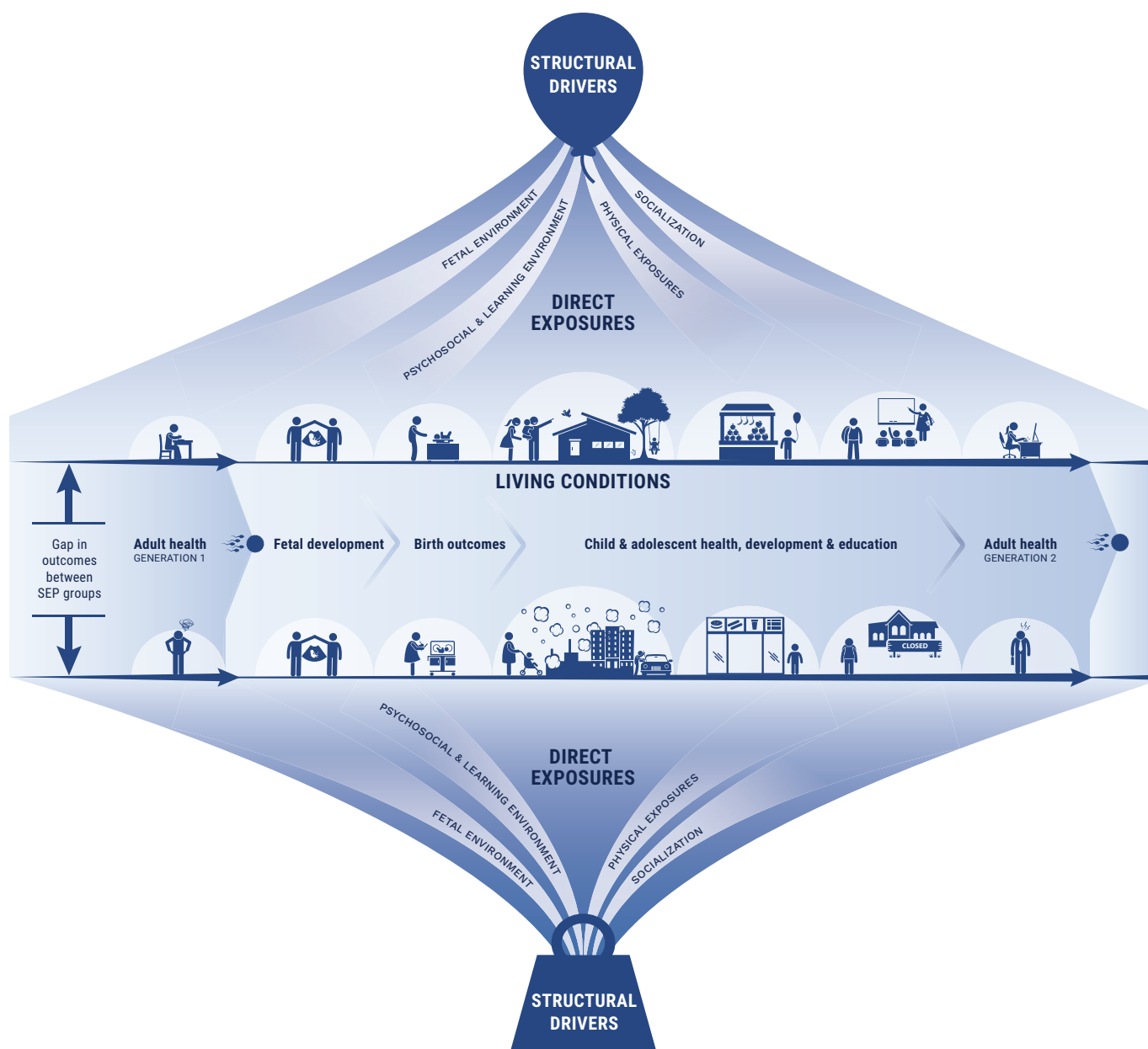


Figure 2 Intergenerational transmission of health inequalities: contributing factors. The figure describes the factors that contribute to the intergenerational transmission of socioeconomic and health (dis)advantage at the family level, leading to the persistence of health inequalities at the population level. Fewer socioeconomic resources and—on average—worse health make it more difficult for lower socioeconomic position (SEP) (lower line) parents (generation 1) to provide a nurturing environment for their children than for higher SEP parents (upper line). Inequalities in direct exposures in the fetal period (eg, maternal health and health behaviours) contribute to worse birth outcomes in children (generation 2) born to parents of low SEP, with subsequent effects for child health and development. Inequalities in the postnatal environment (especially the psychosocial and learning environment, physical exposures and socialisation) further contribute to inequalities in child and adolescent health, development, behavioural habits, educational attainment and subsequently to adult health and the ability to make healthy choices throughout the life course. The conditions in which low SEP parents live, and their structural drivers, make it more difficult to provide a nurturing environment for children. These structural drivers pull, as it were, the upper line up and the lower line down. For example, economies are structured such that incomes and job security are better for jobs requiring a higher education, leaving children of less educated parents at greater risk of growing up in poverty. On reaching adulthood, attained health, SEP and behavioural habits in generation 2, in turn, influence the odds of leading a flourishing life in generation 3. Developed by Liesbeth Smith, The Online Scientist, commissioned by and in collaboration with the authors.

Fetal growth is slower in fetuses of women with lower education.^{25 38} In some recent European cohorts, socioeconomic inequalities in fetal growth were primarily explained by smoking inequalities.^{25 26} A Dutch cohort study, for example, found that 15.5% of infants of low-educated mothers were born small for gestational age, compared with 8.5% among high-educated

mothers. Two-thirds of this difference was explained by maternal smoking.²⁶

Adverse fetal circumstances and adverse perinatal outcomes can have long-term effects on adult SEP and health. Fetal growth rate and length of gestation can have latent health effects, resulting from environmental influences experienced during

critical or sensitive periods.^{6 39} Fetal growth is associated with health in adult life and old age, and these associations are well documented for cardiovascular health.^{40–42} The risk of hypertension, for example, is higher as birth weight declines.⁴³ Birth outcomes can also become part of the life course processes of accumulation of risk and multiple pathway effects, in that they are also associated with motor development, child and adult cognitive functioning and educational attainment,^{28 44–48} with possibly stronger effects at lower ends of the (eg, birth weight) distribution.^{49 50}

Research on possible postnatal confounding of the associations between fetal circumstances and adult SEP and health remains scarce. For childhood cognitive function, there is some evidence that its association with fetal conditions is partly confounded by the family environment.⁴⁸ Nevertheless, sibling studies—in which associations between and within families are compared—suggest that fetal growth remains associated with child and adult cognitive function independently from fixed maternal and family factors.⁴⁴

The effect of the fetal environment may be modified by the postnatal environment. For cognitive development, evidence remains limited and mixed, with some studies showing stronger negative effects of preterm birth on cognitive outcomes for low SEP children and other studies showing no interaction.⁵¹ Evidence on effect modification is, however, substantial for blood pressure, CVD and diabetes: impaired fetal growth is more strongly associated with these outcomes in overweight/obese adults and/or those with an unhealthy lifestyle.^{52–54}

Research on mediation of the effect of parental SEP on offspring health and development by fetal conditions in contemporary cohorts remains scarce. One study found that birth weight only modestly explained socioeconomic inequalities in childhood blood pressure and prehypertension, but fetal growth was not examined.⁵⁵ Another study found that inequalities in all-cause mortality by parental SEP were not explained by gestational age at birth and birth weight, but no formal mediation analysis was done.⁵⁶ Limited evidence on cognitive development suggests that the mediating effect of fetal conditions is small, absent or only observed in the lowest SEP,^{57–59} although third-trimester fetal growth was arguably not adequately captured. Furthermore, the magnitude of the effect of, and part of the variance in cognitive development explained by, fetal factors appears moderate compared with that of parental SEP, suggesting that the postnatal environment may be more important for cognitive functioning than fetal growth.^{44 45 48}

Summarising, substantial inequalities in the fetal environment—and the structural drivers of these inequalities discussed further below—contribute to socioeconomic inequalities in adult health and SEP. The magnitude of this contribution remains uncertain; the limited evidence available suggests it may be moderate compared with the contribution of the postnatal environment.

THE POSTNATAL CHILD AND ADOLESCENT ENVIRONMENT

The postnatal environment strongly influences child and adolescent health and development. The physical and (psycho)social environment becomes literally embodied,⁶⁰ for example, by influencing brain development. Through socialisation, the social and physical environment become embedded in the child's and adolescents' repertoire of beliefs, values and behavioural habits, which track into adulthood. Socioeconomic inequalities in the postnatal environment are substantial, with enduring consequences for health and SEP through a combination of latent, cumulative and pathway effects.^{61 62}

Physical environment

The physical environment is strongly socioeconomically patterned, shaping inequalities in child health and development, and adult health and educational attainment. Children in poor households are, for example, more likely to be exposed to outdoor and indoor air pollution and toxins such as lead,^{63 64} impacting neurological and lung development.⁶⁵ They are exposed to poorer food environments, at home, school and in their neighbourhoods,⁶⁶ affecting health via nutrition-related outcomes.⁶⁷ Physical environments also indirectly affect health, by influencing the learning of (un)healthy eating habits.⁶⁸

Psychosocial and learning environment

In the first years of life, the brain develops most rapidly and is highly sensitive to external influences. The early childhood psychosocial environment and the quality of stimulation in particular affect socioemotional and cognitive development. Cognitive development includes, among others, language development and executive functions,⁶⁹ and the underlying brain functions of self-control, working memory and mental flexibility.^{70–72} In turn, these influence educational attainment, mental health and ability to make healthy choices throughout the life course.^{69 73} Research on high-quality early child development interventions shows that the psychosocial and learning environment can have substantial causal effects on child development and later life health and social outcomes.^{61 62}

Socioeconomic inequalities in cognitive stimulation and the psychosocial home environment have been observed in UK cohort studies,^{70 71} possibly partly explained by parental stress and mental health problems associated with low SEP.^{74–76} Experimental and non-experimental longitudinal research has shown that improvements in family income are associated with improvements in child executive functions and school achievement,^{77 78} possibly partly mediated by improved maternal mental health.⁷⁹ Adverse childhood experiences (ACEs) are more common in children growing up in poorer households.^{80 81} These experiences become embodied, through neural sculpting of the developing brain.^{82 83} ACEs, including child maltreatment, are associated with lower adulthood SEP, higher prevalence of mental and physical health problems and harmful behaviours, and greater ACE risks for the subsequent generation.^{84–86} Inequalities in the psychosocial and home learning environment are compounded by lower participation in high-quality early childhood education programmes among children of disadvantaged households.⁸⁷

Socialisation

Childhood and adolescent socialisation—the process by which patterns of beliefs, values and behaviours that are acceptable in a social context are learnt and internalised, first from direct care takers and then from peers and their wider social environment—is another mechanism through which health inequalities are reproduced across generations.⁸⁸ It leads to intergenerational similarity in health-related behaviours.⁸⁸ Adolescents are, for example, more likely to start smoking when their parents smoke.⁸⁹ Contemporary socioeconomic inequalities in childhood smoking uptake are largely explained by parental smoking.⁹⁰ And (un)healthy behaviours, such as smoking, diet and physical (in)activity track from childhood into adulthood.^{68 91}

Inequalities in the postnatal environment translate into inequalities in child and adolescent health and development. Socioeconomic inequalities in physical child health, including body mass index, dental health and asthma, are substantial.⁹² There are also socioeconomic inequalities in childhood brain development, especially

in areas linked to memory, self-regulation and emotion, although the sample size of these studies is generally small.⁸² Similarly, socioeconomic inequalities in child cognitive and social-emotional development and mental health have been observed.^{77 92–95} Child and adolescent cognitive and socioemotional development, in turn, affect educational attainment,⁷³ future labour market participation, occupational status and income.^{73 96}

Mediation analyses suggest that contemporary socioeconomic inequalities in childhood cognitive and social-emotional development can for a substantial part be explained by the psychosocial and home learning environment^{70 77 97} and only for a small part by prenatal smoking, alcohol use and low birth weight.⁹⁵ Yet, little is known about how this contribution varies across contexts. Also, little is known about the extent to which adult health inequalities are mediated by inequalities in the childhood and adolescent environment. A substantial literature links parental SEP to offspring adult health.^{8 9 98} The effect of parental SEP on stomach cancer is partly mediated by childhood infection with *Helicobacter pylori*, whereas its effect on lung cancer is partly mediated by adult SEP and associated smoking behaviours.⁹⁸ Yet, the contribution of early life inequalities to social inequalities in different specific health outcomes in adulthood is rarely quantified.

Summarising, there is substantial evidence that the postnatal environment plays an important role in the intergenerational transmission of health inequalities, through the effects of the physical, psychosocial and learning environment, and of socialisation, and their underlying structural determinants. Yet, few studies quantify their contribution to adult health inequalities, let alone the extent to which this contribution varies across contexts.

GENETIC FACTORS

Genetics may, to some extent, also contribute to the intergenerational transmission of health inequalities. Adult SEP has consistently been associated with genetic factors in large twin studies and genome-wide association studies.^{99–106} Also the degree of similarity in educational attainment between parents and offspring has been partly explained by genetics.^{107–110} While it has been hypothesised that heritability estimates for educational attainment are higher in more meritocratic societies, evidence remains mixed.^{105 111–114} In addition to transmitted alleles, also non-transmitted parental alleles are associated with offspring educational attainment, possibly by shaping the nurturing environment, so-called genetic nurture or social genetic effects.^{115–118}

Furthermore, genetic risk factors for health outcomes that affect SEP might contribute to the intergenerational transmission of health inequalities.¹¹⁹ For example, genetic factors contribute to the risk of developing schizophrenia,¹²⁰ which in turn can, for example, lead to job loss.¹²¹ Inheritance of genetic risk factors for such conditions may reproduce the negative health-SEP link in subsequent generations.

Importantly, heritability estimates are not fixed properties but are context specific, dependent on country, birth cohort, and age,¹¹⁰ and only capture variance around a context-specific mean (eg, in educational attainment)¹⁰⁹ and not the large differences in that mean across countries and time periods induced by social-structural factors. Heritability estimates are thus not a global measure of modifiability or importance of genetic versus environmental factors.^{100 110 122} Moreover, heritability estimates of complex social traits such as educational attainment can be substantially biased by population-level phenomena such as population stratification.^{123 124} Even if part of the observed correlations represent underlying causal effects, their

pathways of influence are far from clear-cut. The genetic correlates of complex traits result from the combined effect of ‘thousands (and probably millions) of genetic variants’, with each a ‘tiny effect size’.^{125 126} The pathways are complex and operate via environmental mediators and moderators.^{100 125} For example, the expression of genetic risk factors for certain mental health problems like schizophrenia and psychotic disorders is influenced by environmental conditions such as adverse life events,¹²⁷ implying that sensitivity to unfavourable circumstances is partly genetically inherited.¹²⁷ Consequently, when social environments become reproduced across generations due to structural inequalities in society, such genetic factors might reinforce the intergenerational transmission of health inequalities.

Summarising, while the intergenerational transmission of genetic factors associated with SEP, and of genetic factors associated with health outcomes that affect SEP, may play a role in highly complex interrelation with the broader social environment, genetic associations may also be confounded by structural societal inequalities. Moreover, they do not explain the large variation across countries and time periods in educational attainment, health outcomes and social mobility associated with social-structural factors, which are more relevant for policy-making.

STRUCTURAL DETERMINANTS OF THE TRANSMISSION CHANNELS

Why are unhealthy behaviours and maternal health problems in pregnancy more prevalent in lower SEP groups? And why are children of low SEP families more often exposed to unhealthy physical environments, less nurturing psychosocial and home learning environments and socialisation processes that make it more difficult to adopt health-promoting behaviours?

Poverty, low education, job insecurity, low housing quality, unsafe and unhealthy neighbourhoods and lower quality (pre)schooling make it more difficult for families to provide a nurturing fetal and postnatal environment.⁷² They make it more difficult to purchase healthy meals, books and computer and provide a quiet room to study and a safe and stimulating space for outdoor playing.^{128 129} And poverty, insecurity and debts may reduce the cognitive bandwidth needed to make healthy choices and provide a nurturing environment.¹³⁰

These ‘instantaneous’ effects of low SEP are amplified by the effects of the ‘long arm’ of adverse fetal and postnatal conditions in previous generations.^{131 132} (Expectant) parents’ ability to make healthy choices and provide a nurturing environment is influenced by their own socialisation, mental health, cognitive ability and executive functions, as shaped by their own childhood circumstances. Grandparental SEP influences their grandchildren’s cognitive ability through its influence on parental cognitive ability and education and their effects on parenting practices (eg, stimulating learning environment, lack of coercive discipline and parental aspirations).⁷⁰ Experiences of child maltreatment or other ACEs increase the risk of adulthood mental health problems and addiction⁸⁶ and of maltreatment in the next generation.¹³³ Mediation of these effects by intergenerational transmission of consequences of environmental insults through epigenetic changes, that is, changes in gene expression, has been proposed by some authors, although direct evidence from studies in humans remains scarce.^{134 135}

There are more structural factors behind these mechanisms. Social segregation is an important one. Socioeconomic inequalities between families are shaped by the similarity between partners in educational attainment, which has increased during the second half of the 20th century in Western Europe and the USA.^{136–138} Educational homogamy will arguably reinforce the intergenerational

transmission of health inequalities, largely through biological embodiment in utero and social and biological pathways in childhood. Not only partners but also friends tend to resemble each other socioeconomically, that is, there is a social network homophily.¹³⁹ And, low-income families tend to live together with similar families in the least healthy neighbourhoods—with less green space, fewer safe places to play and more fast food stores⁶⁶—and their children are more likely to attend more deprived schools.¹⁴⁰ Social segregation, through homogeneity, social network homophily and residential and school segregation, shapes the socioeconomic patterning of social learning, that is, the learning of behavioural habits and styles of thinking from the social environment, through observation, modelling and imitation.¹⁴¹ The higher prevalence of unhealthy behaviours in the social environment of low SEP pregnant families makes it more difficult for them to behave healthily.¹⁴² So the social environment, in which low socioeconomic families live, can reinforce the intergenerational transmission of disadvantage.

Furthermore, economies are currently structured such that incomes are higher for jobs requiring higher education, with greater financial returns to education in more unequal countries.¹⁴³ Rapid technological breakthroughs have benefited high-skilled workers most, at the detriment of low-skilled workers.¹⁴⁴ Furthermore, the rates of return on capital are greater than economic growth, leading to an increasing share of capital incomes in national income,⁴ with consequent rising income and wealth inequalities in high-income countries in the last decades.¹⁴⁴ The gap between top and bottom income earners has increased due to a sharp rise in top salaries, and stagnation at the middle and bottom, whereas tax systems have become less progressive.¹⁴⁴ Hence, children of less educated parents are at greater risk of growing up in poverty.¹⁴⁵ Across European countries, poverty and severe material and housing deprivation are especially high in single-parent households.¹⁴⁵ Combined with the educational gradient in single parenthood observed in Northern Europe and North America,¹⁴⁶ this leads to a triple disadvantage for children in such households: less educated parents, family disruption and poverty.

Policies and institutions can reduce, or reinforce, these inequalities. Childhood poverty, for example, can be reduced through family-friendly social protection policies and labour market conditions that allow people to make a healthy living through work while combining this with care for children.¹⁴⁷ This would arguably reduce the intergenerational transmission of health inequalities, by reducing parental stress and increasing resources available to children. Also health protection and promotion policies and legislation, including antismoking legislation and universal policies to support a healthy diet, can plausibly reduce the intergenerational transmission of health inequalities.¹⁴⁸ The sugar levy in the UK has swiftly led to strong reduction in the sugar content of soft drinks.¹⁴⁸ Similarly, mandatory folic fortification of wheat flour has potential to reduce inequalities in birth outcomes.¹⁴⁹

Finally, whereas education systems can be a 'powerful driver of social selection',⁹⁶ when set up well, they may also reduce the intergenerational transmission of disadvantage. High-quality preschool education and child care, including targeted language support, can reduce cognitive disparities between socioeconomic groups when made accessible and affordable to all¹⁵⁰ and can have long-run effects on behavioural and socioeconomic outcomes.^{87 151} Intergenerational social mobility is generally higher, that is, the link between parental and offspring SEP is weaker, in countries with lower-income inequality.¹⁴³ This is linked to the greater accessibility to high-quality education.¹⁴³

Other social forces may be more difficult to influence for governments. For instance, the earlier timing of female emancipation in

Northern compared with Southern Europe—combined with the vicious promotion of cigarettes as 'torches of freedom'¹⁵² and a lack of government regulation—was associated with an earlier start of the smoking epidemic in women in Northern countries. The female smoking epidemic started among higher SEP groups—who were also the first to give up smoking—and spread to lower SEP groups.¹⁵³ These patterns—over time, place, SEP and gender—are also reflected in variations in the intergenerational transmission of smoking.¹⁵⁴ Differential diffusion by SEP has also been observed for obesity¹⁵⁵ and formula-feeding of infants,¹⁵⁶ arguably giving rise to similarly complex intergenerational patterns over time and place as for smoking. More broadly speaking, social forces, such as female emancipation, urbanisation, changing food systems and changing global economic systems, shape the postnatal and childhood environment. Their influence is socioeconomically patterned, shaping the intergenerational transmission of health inequalities. Recognising these dynamics, which lead to time and place variations in the magnitude of health inequalities and their intergenerational transmission, is important and requires an understanding of the social and epidemiological context.

POLICY-MAKING

To what extent should policies to reduce adult health inequalities be aimed at the early years of life rather than at adults? In a way, this is a false contrast. The circumstances in which adults—often (future) parents—live, influence offspring outcomes. Yet, our analysis helps us understand why it can be so difficult to address unhealthy lifestyles in adulthood.¹⁵⁷ Early life circumstances become embodied and lay the foundation for living circumstances and socioeconomic and health trajectories in adulthood. Furthermore, the structural drivers of early life inequalities persist into adulthood.

Our analysis suggests it is important to provide supportive environments to (expectant) families. Health inequalities, and their intergenerational transmission, can be reduced through policies that soften—rather than harden—the class structure: family friendly social protection policies, education systems that allow all children to flourish, and fair employment conditions.¹⁵⁸ Importantly, health inequalities can be reduced through a legislative and policy environment that supports healthy living conditions and through programmes that support parents to enable their children to develop to their full potential.^{147 159 160}

CONCLUSION

The first two decades of life—from conception to early adulthood—play a critical role in the development of socioeconomic health inequalities and contribute to the persistence of these inequalities. It is time to give more attention to these early years in research on, and policies to tackle, health inequalities. Obviously, SEP and health are not fully predetermined in early life, and children can be resilient to adversity.^{133 161} At the same time, systemic inequalities lower the odds of children of low SEP parents to lead a long, healthy life. This is important to address, so that all children can develop to their full potential.¹⁴⁷ It is the right thing to do, and it is arguably much more effective than remedial action in adulthood.¹⁶²

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