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Issue: *The Biology of Disadvantage***Childhood socioeconomic status and adult health**Sheldon Cohen,¹ Denise Janicki-Deverts,¹ Edith Chen,² and Karen A. Matthews³¹Carnegie Mellon University, Pittsburgh, USA. ²University of British Columbia, Canada. ³University of Pittsburgh, USA

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Socioeconomic status (SES) exposures during childhood are powerful predictors of adult cardiovascular morbidity, cardiovascular mortality, all-cause mortality, and mortality due to a range of specific causes. However, we still know little about when childhood SES exposures matter most, how long they need to last, what behavioral, psychological, or physiological pathways link the childhood SES experience to adult health, and which specific adult health outcomes are vulnerable to childhood SES exposures. Here, we discuss the evidence supporting the link between childhood and adolescent SES and adult health, and explore different environmental, behavioral, and physiological pathways that might explain how early SES would influence adult health. We also address the ages when SES exposures matter most for setting adult health trajectories as well as the role of exposure duration in SES influences on later health. While early childhood exposures seem to be potent predictors of a range of health outcomes, we emphasize that later childhood and adolescent exposures are risks for other health outcomes.

Keywords: socioeconomic status; health; childhood

Introduction

Socioeconomic status (SES) during adulthood is associated with a wide range of adult health outcomes with each increase on the SES hierarchy associated with an increase in health benefits.^{1–3} Recent research suggests that SES exposures during childhood are also powerful predictors of adult physical health (e.g., reviews by^{4–6}). While these data are provocative, we still know little about when childhood SES exposures matter most, how long they need to last, what behavioral, psychological, or physiological pathways link the childhood SES experience to adult health, and which specific adult health outcomes are vulnerable to childhood SES exposures.

In this paper, we discuss the evidence supporting the link between childhood and adolescent SES and adult health, and explore different environmental, behavioral, and physiological pathways that might explain how early SES would influence adult health. We then review three types of models that hypothesize the ages when SES exposures matter most for setting adult health trajectories as well as the role of duration of SES exposure in influencing later health.

Evidence linking childhood socioeconomic status and adult health

Adult and childhood SES are correlated. For example, those with college educated and relatively wealthy parents are more likely to have access to educational opportunities and to higher status, well-paying careers. Consequently, it is possible that childhood SES does not itself play a role in adult health, but merely represents a marker of future adult SES, which in turn influences adult health. Because we are interested in the hypothesis that SES in childhood and adolescence influences adult health through psychosocial, behavioral, and physiological processes important for health later in life, we limit our discussion to research that evaluates the role of childhood SES independent of (*after controlling for*) adult SES.

In two publications, Galobardes *et al.*^{4,5} reviewed 40 studies examining the link between childhood SES and adult mortality. The authors concluded that individuals with lower SES during childhood were at elevated risk of premature mortality, regardless of their socioeconomic circumstances during adulthood. The increased risk associated with childhood socioeconomic disadvantage applied both to

overall mortality as well as to mortality from specific causes. For example, among men and women, lower childhood SES was associated with increased risk of mortality from cardiovascular disease (e.g., coronary heart disease, stroke); respiratory disease; diabetes; cancers of the lung, liver, and stomach; and diseases of the digestive system. Among men, lower childhood SES was also associated with a greater risk for death due to alcoholic cirrhosis, unintentional injuries and homicides, and nervous system conditions. Interestingly, poorer childhood conditions were not associated with mortality attributable to cancers unrelated to smoking or to prostate cancer.

In a review of 49 studies, Pollitt *et al.*⁷ examined the evidence supporting a link between SES throughout the life course and adult cardiovascular outcomes. They concluded that poorer socioeconomic conditions during childhood place individuals at *modest* risk for adult cardiovascular disease and cardiovascular-related mortality, independent of adult SES. More convincing was the evidence that adult cardiovascular disease risk increases as the number of years throughout the life course (childhood included) spent in a low SES environment increases.

These reviews provide provocative evidence for the hypothesis that childhood SES contributes to adult health, independent of adult socioeconomic conditions. However, there are caveats. First, the literature on childhood SES and adult health addresses only a limited range of health outcomes with most studies focusing on mortality or risk for cardiovascular disease. Second, this work is primarily based on analyses of data collected for other purposes. Consequently, study designs often fail to address some of the complexities of the childhood SES hypothesis. For example, many of these studies ask adults to recall their SES during childhood or adolescence. These recollections may be biased and the sources of the bias (e.g., personality characteristics) might also influence current (adult) health. Longitudinal studies eliminate the problem of recall bias. However, most of the longitudinal studies in this literature lack the strength of true prospective studies in that few controlled for childhood health at the point of exposure. Third, most studies include assessments of only one SES marker, usually father's occupation or education. Consequently, it is not possible to compare individual components of childhood SES to assess unique effects on adult

health. Finally, many of the studies use crude measures of SES, or reduce their SES data into broad categories (e.g., manual vs. nonmanual social class), limiting the ability to detect a gradient of worsening adult health with decreasing childhood SES.

To provide a context for understanding the challenges and complexities of studying the question of how childhood SES might influence adult health, we present four illustrative studies that employ different designs and methodologies, focus on a range of adult health outcomes, and demonstrate some of the methodological issues important to resolve in future research. We limit our discussion to the evidence linking early SES to physical morbidity and mortality during adulthood independent of adult SES.

We begin with a study presenting provocative evidence of an association between low SES during childhood and increased all-cause and cause-specific mortality during adulthood.⁸ However, the study also illustrates some of the common limitations of the mortality studies. The sample consisted of 1,824,064 male and female Swedes born between 1944 and 1960. Childhood SES data (occupational class of household head) were obtained from the 1960 Population and Housing Census, when participants ranged in age from newborn to 16 years. Mortality data were collected from 1970 to 2001. Results indicated that individuals from manual childhood social classes were more likely to die from all causes than were individuals from nonmanual social classes, as well as from the specific causes of smoking-related cancers, stomach cancer, respiratory disease, cardiovascular disease, and diabetes. All associations were independent of adult occupational class and educational attainment. A major strength of this study is a large birth cohort sample of both men and women followed from between 10 and 31 years. However, like almost all of the prospective studies in this literature, there were no assessments for health during childhood and adolescence. In consequence, these results may be attributable to sick children becoming sick adults in contrast to childhood exposures influencing adult mortality through influences on physiological, psychological, and behavioral developmental trajectories. Moreover, the dichotomous measure of childhood social class does not allow a test of whether the associations of SES with mortality were graded with either SES exposure at baseline or with length of exposure.

The second example was selected because it examined the role of childhood SES in adult risk for a different health outcome, incidence of coronary heart disease (CHD), and used a novel design to control for adult SES. The sample included 1131 male medical students who graduated from Johns Hopkins University between 1948 and 1964.⁹ Because all of the men in this sample had achieved high socioeconomic status as adults, associations of childhood SES with health outcomes are not likely attributable to SES differences in adulthood. In addition, the men comprising this sample not only would have had access to medical care as adults, but presumably to good quality care. Childhood SES was defined as father's occupational status with low SES including farmers, machine operators, or craftsmen; service workers and laborers; and high SES including professionals, physicians, and managers. Low childhood SES conferred a 2.4-fold increased risk of developing CHD on or before age 50 but not at older ages. This association was not reduced by controls for CHD risk factors such as parental CHD history, body mass index, cholesterol level, amount of exercise, depression, smoking, hypertension, and diabetes mellitus. Limitations of this study include a dichotomous and retrospective measure of childhood SES and the lack of a health measure during childhood.

The third example examined the graded association of childhood SES measured prospectively in relation to a different health outcome, dental health in young adulthood.¹⁰ In a sample of 980 individuals born between April 1972 and March 1973 in Dunedin, New Zealand, participants were followed-up at ages 5, 7, 9, 11, 13, 15, 18, 21, and 26 years. Childhood SES was based on parental occupational status (6 = unskilled laborer, 1 = professional) and averaged across the first 15 years of participants' lives. Adult SES was based on the participant's occupational status at age 26. One unique methodological feature of this study is that it includes a measure of infant perinatal health based on maternal risk and birth complications. Analyses of dental health at age 26, including controls for perinatal health and adult SES, showed lower childhood SES was associated with poorer dental health including more plaque and gingival bleeding, and a greater proportion of individuals with periodontal disease and decayed dental surfaces. Overall the design of this study has some major strengths. How-

ever, there are limitations as well. Given that dental disease is progressive, an analysis of how soon in childhood parental SES leads to more rapid deterioration of dental health would have been helpful. Also, knowing about access to and use of dental care during childhood, which should be correlated with family SES, would have informed an understanding of potential mechanisms. Finally, more could be learned about the importance of timing of SES exposure from these data by conducting analyses comparing SES markers at different years as predictors of adult health (see section on models in this paper).

Our final example¹¹ examined the association of early life SES with adult host resistance to upper respiratory infection in a sample of 334 healthy adult volunteers. This study assessed the number of years of early exposure to a low SES (as described later in section on models) and tested when in early life the maximal association between SES and host resistance was apparent. Childhood SES was operationalized in two ways: participants' self-reports of their parents' highest level of education; and participants' self-reports of the ages during their childhood and adolescence (separate measures for each of the years from 1 to 18) when their parents owned their homes. Adult SES was operationalized similarly, with participants' highest level of education being measured by self-report and home ownership at the time of the study being obtained from real estate records. Measurement of host resistance to infection was accomplished using a novel procedure wherein participants were administered nasal drops containing one of two viruses that cause the common cold and were then monitored in quarantine for the development of clinical illness (infection together with signs and symptoms of illness). Overall 23% of the participants developed a cold. For both viruses, susceptibility to colds decreased in a graded fashion with the number of childhood years during which participants' parents owned their homes. This decreased risk was attributable to both lower risk of infection and lower risk of illness in infected subjects. These associations were independent of baseline immunity to the virus (prechallenge virus specific antibody), body mass index, parents' education, participants' education, and home ownership. Although this study suffers from the weaknesses of retrospective measurement of home ownership, controlling for personality characteristics known to

influence retrospective reporting did not alter the findings. It is unclear why they failed to find an association of parental education and disease susceptibility since parental education has been predictive in many other studies.

How does childhood SES influence adult health?

Up until this point, we have argued that one's position in the SES hierarchy during childhood and adolescence has important implications for adult health. One explanation for this association involves access to and affordability of adequate health care. In many countries, children of low SES parents are less likely than those of more affluent parents to receive necessary and preventive medical care due to their parents' inability to pay for these services. Insufficient care during childhood and adolescence could place individuals at greater risk for poor health throughout the lifecourse. Despite the plausibility of this explanation, much of the evidence for an inverse association between childhood socioeconomic conditions and adult morbidity and mortality risk has been derived from research conducted in countries that have adopted systems of nationalized health care such as Sweden,^{8,12} Norway,^{13,14} Finland,¹⁵ Great Britain,^{16,17} Scotland,¹⁸ New Zealand,¹⁰ and South Korea.¹⁹ That childhood SES should continue to predict adult health outcomes in countries where all children—irrespective of their socioeconomic circumstances—presumably receive comparable access to medical care substantially decreases the likelihood that inequitable distribution of health care across socioeconomic strata plays a central role in explaining the link between childhood socioeconomic disadvantage and adult health risk.

Outside of differences in health care, lower childhood SES is thought to be detrimental to adult health because of decreases in favorable and increases in unfavorable environmental exposures associated with declining SES. In the present context, the term *environment* refers both to physical and psychosocial exposures associated with children's homes, schools, and neighborhoods. In turn, these environments are hypothesized to influence health through their effects on developmental trajectories involved in the maturation of relevant psychological and physiological processes and in the acquisition of

health-related behaviors. The courses of these trajectories proceed through adulthood and affect the more proximal causes of disease and dysfunction. In the following sections, we outline some of the physical and psychosocial exposures that are linked to SES, and the ways in which they may in turn impact health.

Physical exposures

The top half of Figure 1 lists several examples of physical environmental exposures that differ in quality and quantity depending on level of SES. These physical exposures include factors present within the home, neighborhood, and school environments. We address many of the physical exposures thought to increase with decreasing SES, and to result in adult disease risk. However, we are not exhaustive (see reviews by^{20,21}).

Within the home environment, children from higher SES families benefit from better quality residential space that is lacking in potentially harmful exposures such as lead-based paint, carbon monoxide, or general poor maintenance.²¹ Household radon levels, for example, which are determined largely by structural deficiencies such as foundation cracks and unimproved basement floors, correlate negatively with family income.²² Moreover, higher SES families are more likely to be able to afford homes with sufficient space to accommodate their needs, thus reducing the likelihood of residential crowding.²³ Given the inverse association between socioeconomic advantage and tobacco smoking among adults,²⁴ the likelihood of children being exposed to environmental tobacco smoke within their homes increases with decreasing socioeconomic advantage as well.²⁵

Schools serving children from higher income families similarly have better physical facilities. They have fewer temporary buildings and more adequate physical plants based on roof, framing, floor, foundation, plumbing, ventilation, A/C, life safety features, and so on.²⁶ At the neighborhood level, areas with a higher relative to lower average SES are less likely to be located near sources of hazardous wastes and more likely to be characterized by clean air and water.²¹ Higher SES neighborhoods also are more likely to be insulated against excessive noise,²⁷ and have adequately maintained streets, sidewalks, and public spaces.²⁸

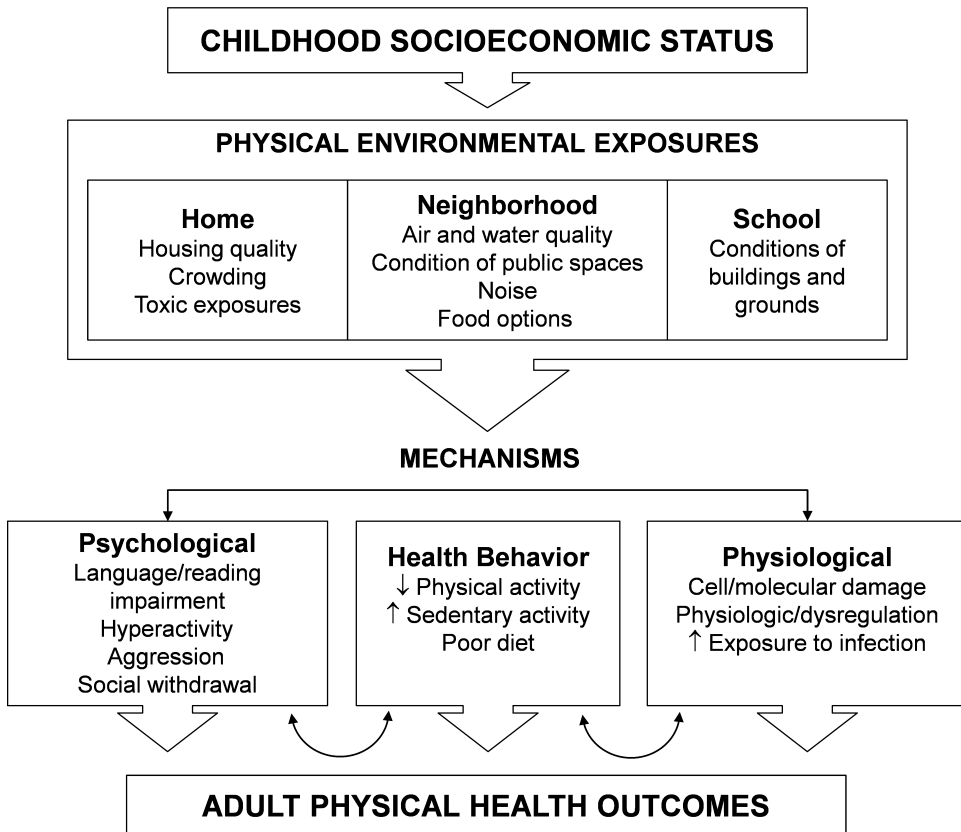


Figure 1. Examples of pathways that may link physical exposures associated with childhood and adolescent SES to adult health.

The bottom half of Figure 1 illustrates the mechanisms through which SES-related physical exposures might be translated into adult health outcomes. These mechanisms include factors influencing psychological maturation, acquisition of health behaviors, and physiological growth and development.

Physical exposures and psychological development

Growing up in a higher SES environment may benefit children's long-term health by decreasing the likelihood that they encounter adverse physical exposures associated with impaired cognitive, social, and emotional development (see reviews by Evans²⁰ and Leventhal *et al.*²⁹). Noise pollution, for example, can impede children's acquisition of language and reading skills.^{30,31} Likewise, increasing exposure to environmental tobacco smoke, as determined by children's and adolescents' serum cotinine lev-

els, has been associated with poorer performance on tests of reading, math, and visual construction abilities.³² In addition to the obvious long-term effects, such cognitive deficits might have on children's future educational and career opportunities, these deficits also carry the potential to exert more immediate effects on children's psychological development, such as increased stress and depression, school-related anxiety, inaccurate or biased expectations about their abilities, a diminished sense of mastery, and lower self-esteem. These psychological costs are in turn potential triggers of stress-related biological responses and of health-damaging behaviors that increase risk for disease over the life course.

Other toxic environmental exposures associated with lower SES have also been found to have detrimental psychological effects that are potentially harmful to adult health. Survivors of childhood lead poisoning manifest increased hyperactivity,

impulsivity, and aggression following their recoveries, with these effects often persisting throughout adulthood.^{33–35} Residential crowding during childhood is also associated with increased aggression, as well as with social withdrawal, psychological distress, and poor school adjustment (see²⁰), all potential precursors of adult disease (e.g., Refs. 36–38).

Physical exposures and health behavior development

In regard to the direct influence of physical exposures on health behaviors, availability of safe outdoor environments and recreational facilities can impact a child or adolescent's level of physical activity. With increasing SES, families are more likely to live in homes with yard and/or garden space and neighborhoods with public parks and recreational facilities.³⁹ As SES increases, so too do parents' abilities to provide their children with opportunities to participate in organized sports or other athletic or recreational activities.^{40,41} Restrictions on physical activity, the lack of parent role modeling of regular exercise, and subsequent preferences for sedentary activities place children at increased risk for obesity,^{42,43} poor bone health,⁴⁴ and decreased overall levels of fitness.^{45,46} All of these conditions may set health-damaging trajectories that continue through adulthood. For example, findings from a 36-year cohort study of children born during a specific week in 1946 and 1958 showed that lower SES during childhood was associated with lower levels of physical activity both during childhood and adulthood⁴⁷ and with adult obesity.⁴⁸

Children's dietary habits also can be influenced by their surrounding environments. Full-service supermarkets with large selections of healthy foods (e.g., fresh produce; low-fat and low-sodium options) are more prevalent in higher relative to lower SES neighborhoods,⁴⁹ whereas fast food restaurants are more prevalent in more disadvantaged areas.⁵⁰ Accordingly, lower SES children and adolescents will likely have greater exposure to unhealthy foods and less exposure to nutritious foods relative to their more affluent counterparts. These early dietary exposures can have long-term effects on adult health by initiating poor dietary habits that persist throughout adulthood, as well as by contributing to poor childhood health (obesity, high cholesterol,

poor immunity) which ultimately can result in elevated disease risk later in life.

Physical exposures and physiological development

Socioeconomic advantage may also benefit children's long-term health by protecting them from physical environmental exposures associated with impaired physiological development. As already discussed, childhood exposure to pollution increases with decreasing neighborhood SES. Irritants and toxicants present in air and water can result in cellular and molecular level damage that, when initiated during childhood or adolescence, could create vulnerabilities to future disease. For example, people who are exposed to air pollution and environmental tobacco smoke during childhood are at greater risk for developing chronic respiratory disease.^{51,52} Children chronically exposed to noise pollution exhibit elevated neuroendocrine stress hormones and blood pressure levels (see^{20,53,54}), changes that may persist and interfere with the normal development of endocrine, cardiovascular, and immune systems.

Lower SES is also associated with an increased likelihood that children are exposed to toxins within the home. Children who live in homes containing lead-based paint, for example, may develop lead toxicity, which can disrupt development of neural networks and compromise the blood-brain barrier.^{55,56} Lead may also have direct effects on the functioning of physiological systems. For example, lead measured in children's blood at the age of two is a mediator of the association between low family SES and children's vascular resistance and cortisol responses to acute stressors measured at age nine.^{57,58}

In addition, the smaller occupant-to-room ratio characteristic of higher SES families' homes may protect children from the increased risk of exposure to infectious agents associated with residential crowding.⁵⁹ Although the hygiene hypothesis suggests that early exposures to infectious agents may protect children from developing certain atopic diseases,⁶⁰ reductions in contagious exposures may also have long-term benefits for children's health, as some early infections and related inflammation can increase children's later susceptibility to more serious disease.⁶¹ In fact, lower family income and lower parental education have been associated with a higher burden of chronic infections in children.⁶² Moreover, common infections such as *Helicobacter*

pylori (*H. pylori*), cytomegalovirus (CMV), herpes simplex virus-1 (HSV-1), hepatitis A and hepatitis B have been associated with lower height-for-age as well as an increased likelihood of asthma markers, both markers of potential long-term health problems.⁶² Infection with the Hepatitis B virus during the first 5 years of life has also been found to increase lifetime risk of developing cirrhosis of the liver, liver failure, and hepatocellular cancer.⁶³ Furthermore, evidence from comparative animal research has indicated that early life exposure to endotoxin alters hypothalamic-pituitary-adrenal axis (HPA) function in such a way as to impact later stress responsivity⁶⁴ and vulnerability to inflammation.⁶⁵

Psychosocial exposures

The top half of Figure 2 provides examples of psychosocial exposures that differ according to where a child’s family is positioned on the socioeconomic hierarchy. As with physical exposures, homes, neighborhoods, and schools are all sources of psychoso-

cial exposures that act as upstream influences on more proximal determinants of adult health and disease. Although the psychosocial exposures listed in the figure are not exhaustive, they represent the major types of psychosocial experiences that take place during childhood and adolescence, vary according to SES, and have long-term effects on adult health.

Within the home, a family’s SES can substantially influence how well that family functions. As SES increases, so too does the likelihood that families are characterized by low levels of conflict, warm and attentive family relationships, and consistent parenting practices.^{66,67} High levels of conflict, cold and neglectful relationships, and harsh and inconsistent parenting are characteristic of “risky families,” which are labeled as such because of the potential increased risk of psychological and social dysfunction characteristic of children who come from them.^{68,69} Behaviors and affective states associated with risky families, in addition to being associated with low SES, may also promote further socioeconomic

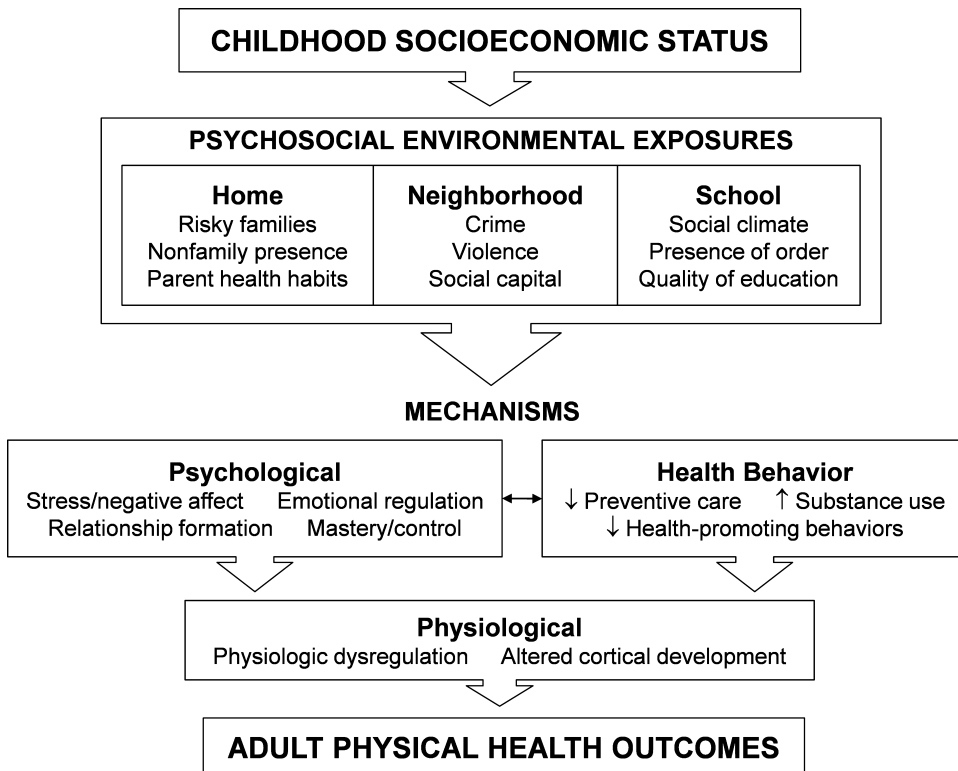


Figure 2. Examples of pathways that may link psychosocial exposures associated with childhood and adolescent SES to adult health.

disadvantage. For example, if parental conflict escalates into marital dissolution, the custodial parent (usually the mother) will likely experience a relative drop in SES, thus further perpetuating the potentially adverse psychosocial exposures associated with decreasing status.⁷⁰ Environmental chaos—minimal family structure and routinization combined with high levels of background stimulation also increases with decreasing household status.⁷¹

The social environment of the surrounding neighborhood also varies by socioeconomic factors. For example, as the average SES of a neighborhood increases, levels of social capital—community characteristics such as trust in others and norms of reciprocity—increase, and threats of crime and violence decrease.⁷² Similarly, children attending schools located in more affluent neighborhoods are less likely than children attending lower SES schools to observe or be victimized by enacted or threatened physical violence.⁷³ Moreover, as neighborhood SES increases so does the regular attendance and consistent enrollment of students, which contributes an overall order and cohesiveness of the educational environment.⁷⁴

As indicated in the bottom half of Figure 2, psychosocial characteristics associated with lower childhood SES can have long-term effects on physical health through affective pathways, through impacts on the adoption of salubrious or damaging health-related behaviors, and in turn through the potential dysregulation of physiological systems. Like physical exposures, adverse psychosocial exposures that increase in prevalence with decreasing SES have been associated with increased risk of adult physical disease.^{75,76} Unlike physical exposures, however, psychosocial exposures do not directly influence physiological mechanisms. Rather, effects are all indirect via psychological factors (e.g., psychological stress, negative affect) and health-related behaviors.

Psychosocial exposures and psychological development

Psychosocial exposures that occur within the home are particularly important with regard to influencing children's psychological well-being. For example, children from risky families, who are more likely to be of lower SES, have greater difficulties with emotion regulation.^{68,70} Poor emotion regula-

tion during childhood may predispose individuals to higher anxiety, depression, and hostility in adulthood.⁶⁸ Indeed, anger, hostility, and depression have all been associated with increased risk for cardiovascular disease morbidity and mortality (e.g., Refs. 37 and 38).

Children from risky families, particularly families where there is marital conflict, also have greater difficulties with social competence, resulting in behavioral problems and difficulties forming and maintaining close social relationships.^{69,70} The ability to form and maintain strong and diverse social ties and the access to social support are potent predictors of child and adult health.³⁶

Social characteristics of the neighborhood in which a child lives also can influence his or her psychological development. As discussed earlier, the likelihood of living in a violent neighborhood or in a neighborhood with low social capital increases with decreasing SES.⁷² Being witness to or victimized by high levels of neighborhood, school, or home violence is associated with poor emotion regulation and maladaptive social information processing,^{77,78} with increased feelings of fear and decreased feelings of control,⁷⁹ and with poor social adjustment.⁸⁰ Children observing violence have been found to display immune dysregulation and greater risk for asthma morbidity^{81,82} that are thought to set a trajectory for poor adult respiratory health. Living in neighborhoods with low social capital is associated with poorer self-rated health and higher mortality rates, perhaps in part by impeding psychosocial resources such as support and respect.^{83–85} Similarly, attending schools with less overall order and cohesiveness and higher levels of disruption and violence could contribute to increased perceived stress and the development of feelings of helplessness, isolation, and fear and in turn behavioral and physiological effects that are detrimental to health.

Psychosocial exposures and health behavior development

Psychosocial exposures also can influence children's long-term health through mechanisms involving behaviors enacted by the children themselves or those enacted by their parents. Research conducted with adults shows that the likelihood of being a smoker or heavy drinker decreases and of consuming a healthy diet and engaging in regular physical activity increases with increasing SES.^{24,86}

Accordingly, children of higher SES parents are more likely to be exposed to health-promoting behaviors and less likely to be exposed to risky behaviors. Parents serve as models for the appropriateness of both harmful and beneficial health behaviors. Thus, parental health practices can significantly influence the behaviors children choose to adopt. Probability of smoking, excessive alcohol consumption, and consumption of high fat foods is greater in adolescents whose parents partake in each of these health-risk behaviors.⁸⁷ In contrast, adolescents whose parents engage in regular exercise are more physically active than adolescents with more sedentary parents.⁸⁸

The prevalence of adult depression increases with decreasing SES.⁸⁹ Thus, the likelihood that a child will have one or more depressed parents also increases the further one moves down the socioeconomic hierarchy. Children of depressed mothers are less likely to visit their family physician for routine well-child care than are children of nondepressed mothers.^{90,91} This relative lack of parental attention to preventive care may influence children's long-term health in two ways: first, those who do not receive adequate well-child care may become vulnerable to diseases and conditions that may otherwise have been prevented; and second, children may interpret their not receiving well-child care as an indication that preventive care is not important. Insofar as such a misconception persists throughout adulthood, the likelihood of future vulnerability to poor health increases.

Neighborhood SES has also been found to play a role in critical health practices. Living in a neighborhood populated by comparatively few professional or managerial workers is associated with an increased risk of adolescent and out-of-wedlock childbearing.^{92,93} Teenage pregnancy carries with it a higher risk of maternal health complications than does pregnancy in adult women. For example, teenage mothers have been found to be at greater risk than adult mothers of developing preeclampsia during their pregnancies,⁹⁴ which has been associated with an increased risk of hypertension, stroke, and cardiovascular death later in life.^{95,96} Pregnant adolescent girls also are more likely than pregnant adult women to have acquired a sexually transmitted disease (STD), as well as other genital-urinary infections such as bacterial vaginosis and cystitis.^{97,98} If left untreated, infection with an STD

can have negative implications for long-term health, especially among women. For example, the connection between human papilloma virus (HPV) and risk for cervical cancer in women is well established. Chlamydial infections—which often remain undiagnosed for months or even years—can increase a woman's risk of developing pelvic inflammatory disease and subsequent chronic pelvic pain and infertility. In rare cases, infection with hepatitis B can become chronic and place individuals of both sexes at risk for potentially fatal liver disease.

Psychosocial exposures and physiological development

Psychosocial exposures, via their effects on certain aspects of psychological development, also can influence the quality of children's long-term health through physiological mechanisms. Chronic psychological stress associated with belonging to a risky family may promote dysregulation of certain biological systems, ultimately leading to preclinical and clinical disease.⁶⁸ For example, prolonged or repeated activation of the HPA and the sympathetic-adrenomedullary system (SAM) in response to an unpredictable family environment creates the potential for allostatic load, a loss of adequate regulation of a broad range of physiological systems.⁹⁹ Allostatic load may contribute to the pathophysiology of many chronic diseases, and may act as a pathway through which psychosocial factors such as familial instability might influence susceptibility to and exacerbation of disease.¹⁰⁰ For example, stressful domestic situations that increase with decreasing SES, such as maternal depression and the presence of an unrelated adult male in the household, are associated with hormonal disruptions and accelerated puberty in girls.¹⁰¹

Less than optimal psychosocial environments are also associated with alterations in cortical structure and function. Early deprivation of enriched environments, including social relationships and parental investment, are associated with abnormal cortical synaptogenesis in young children due to insufficient stimulation.¹⁰² Adult offspring from risky families demonstrate atypical amygdala activation in response to emotional stimuli, suggesting that the emotion dysregulation frequently displayed by children from risky families may be observable at the neural level.¹⁰³

Psychosocial exposures during later childhood, particularly those that involve modeling of health risk behaviors, also can influence cortical maturation. Adolescence is a period during which individuals are most likely to experiment with risky behaviors.¹⁰⁴ Moreover, the state of cortical development during adolescence makes individuals at this stage of maturation exceptionally vulnerable to the long-term adverse effects of alcohol. Early and excessive alcohol consumption can result in “reprogramming” of the developing cerebral cortex such that the adolescent is at increased risk both for cognitive impairment and for future dependence.¹⁰⁵

Early psychosocial exposures associated with lower SES are also thought to alter biological systems in a manner that can influence health decades later. In the early years of life, when the developing nervous, endocrine, and immune systems are still plastic, the social (and physical) environment may alter these systems in persistent, and sometimes permanent, ways.¹⁰⁶ Comparative animal research conducted with rat pups shows that early life exposures to nurturing social environments, such as those that include regular handling by keepers or high levels of licking and grooming by their mothers, have been associated with reduced adult corticosterone responses to restraint stress,^{107,108} and greater adult hippocampal expression of genes encoding the glucocorticoid receptor.¹⁰⁹ Extrapolating these findings to humans, one might speculate that individuals who were reared in warm and supportive family environments, compared to those who grew up in neglectful or chaotic environments, may be more likely to evidence similarly enhanced regulation of HPA activity.

One intriguing hypothesis for how biological alterations might take place as a consequence of SES-related physical and psychosocial exposures involves the effects of environmental exposures on genomic processes. For example, environmental exposures may affect biochemical modifications to the nucleotides that comprise DNA or to the histone proteins that package DNA, a process known as epigenetic programming. Hence epigenetic programming refers to stable changes in the activity of a gene that arise without alterations to its DNA sequence. Epigenetic programming has been hypothesized to mediate the aforementioned effects of maternal licking and grooming on rat pups’ corticosterone responses to restraint stress,^{107,108} as adult rats who

received high levels of licking and grooming early in life showed less DNA methylation, and more histone acetylation in hippocampal regions associated with the glucocorticoid promoter.¹¹⁰ Other types of gene–environment relationships include those wherein genotype shapes the kinds of environmental experiences an individual has, and those wherein genotype influences *how* an individual will be *affected by* a given environmental exposure. The former relationship was exemplified by the findings of a recent study that showed a correlation involving a polymorphism of the dopamine transporter gene (DAT1) and adolescent boys’ tendencies to affiliate with delinquent peer groups.¹¹¹ The latter relationship has been demonstrated by the repeatedly observed moderating effect of a functional polymorphism in the serotonin transporter promoter region (5-HTTLPR) on the association of psychological stress with risk for depression.^{112,113}

When does SES exposure matter the most?

Three broad conceptual models hypothesize when during childhood and adolescence (i.e., ages or developmental periods) and for what duration SES-related physical and psychosocial exposures have the most important implications for adult health (cf. discussion of life course models by Refs. 7,114–116). We will refer to these as the *timing*, *accumulation*, and *change* models.

Timing model

According to the timing model, SES-related factors have the greatest influence on adult health if experienced during *specific developmental periods* defined as age ranges (e.g., birth to three years) or more broadly as periods of development (e.g., childhood, adolescence). The fit of this model to the actual processes linking early life SES with adult health depends upon the identification of *sensitive periods* when individuals are most vulnerable to SES exposures. In addition, identifying the developmental stage during which a sensitive period occurs may depend upon the outcome of interest (cf. ¹¹⁷). For example, early childhood may be a sensitive period during which exposure to SES-associated air pollution or environmental tobacco smoke significantly increases susceptibility to respiratory conditions by damaging or delaying the developing

respiratory system, whereas adolescence may be a sensitive period during which modeling of poor health behaviors such as smoking and poor dietary habits may increase lifetime risk for atherosclerotic disease.

Table 1 provides some examples of sensitive periods during which certain types of exposures increase the risk of specific health outcomes. As apparent from the table, very early exposures carry the potential to exert a powerful impact on adult health, which is the usual prediction of timing models. However, as also suggested by Table 1, the risk for several specific health outcomes may be more sensitive to exposures that are encountered later in childhood or adolescence.

Although we focus here on exposures that occur during childhood and adolescence, exposures that occur *in utero* also have been associated with vulnerability to future disease. The fetal origins hypothesis proposes that suboptimal maternal nutrition during gestation may have detrimental effects on fetal growth specifically by initiating persistent changes in metabolic, physiological, and structural parameters.¹¹⁸ Long-term effects associated with poor growth *in utero* include increased risk of CHD,^{119,120} insulin resistance and type 2 diabetes (see ¹²¹ for review), and noncardiovascular death in women.¹²² In addition to maternal nutritional deficiencies, other *in utero* exposures that become increasingly common with decreasing SES also have been found to place individuals at elevated risk for poor adult health. For example, exposure to allergens *in utero* is associated with increased risk of developing asthma and related allergic diseases among individuals with a hereditary disposition toward allergy (see Ref. 123, for review). Also, transplacental exposure to polycyclic aromatic hydrocarbons has been found to be associated with genetic mutations that place individuals at increased risk for cancer later in life.¹²⁴ Interestingly, because *in utero* exposures are often highly correlated with exposures occurring in the early years of life, studies reporting the importance of exposures during the first years of life may actually reflect the importance of prenatal environments.

Accumulation model

The accumulation model suggests that the detrimental effects of low SES accrue throughout the life

course such that risk for poor adult health increases with increasing *intensity* of socioeconomic disadvantage and with increasing *duration* of exposure to such disadvantage. In contrast to the timing model, the accumulation model is indifferent to when during childhood and adolescence the SES-related exposures occur. Rather, the accumulation model considers risk in proportion to the total *dosage* of exposure to SES-related adversities over the course of childhood and adolescence. In other words, the accumulation model predicts that those experiencing low SES throughout both childhood and adolescence are at greater risk for adult morbidity and mortality than are those experiencing low SES during only parts of their childhood or adolescence. The life course view of accumulation is that age is totally irrelevant. The only thing that matters is the magnitude and duration of exposure. It is important to emphasize that we are posing a different question than the life course researchers. This paper addresses whether early exposures have effects on adult health that are independent of adult exposures. Thus, we propose the possibility that accumulated exposures experienced during childhood and/or adolescence may be more important than accumulated exposure later in life.

Change model

The change model suggests that the *direction* of SES mobility across childhood and adolescence has important implications for adult health outcomes. Predictions of this model include that upward mobility, a change from lower to higher levels of SES, would result in better adult health. Put differently, it predicts that negative effects of low SES during early childhood would be partly or wholly remediated by higher SES later in childhood or adolescence. In contrast, downward mobility—a fall in SES over childhood and adolescence—would result in poorer adult health. That is, children who are born to high SES families would not be protected from the negative effects of low SES that might be experienced at a later developmental stage. This model does not predict different outcomes than one would predict from adult SES, provided that the upward or downward trajectory continues into adulthood. Less discussed (or tested) is the possibility that multiple changes in SES (whether upward or downward), particularly if they are drastic differences, throughout the

Table 1. Examples of sensitive periods during which certain types of exposures increase the risk of specific health outcomes

												Puberty									
Stage	Infancy		Childhood										Adolescence						Adulthood		
Age (years)	Birth to 1 year	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20...
Exposure																					
Psychosocial environment	Deprivation of enriched environments may result in decreased cortical neurogenesis																				
	Family stressors in the home may lead to accelerated puberty in girls, thus increasing their risk for future health problems																				
Physical environment	Exposure to lead may disrupt cortical development, resulting in impairments to brain function and IQ that may continue through adulthood.																				
	Exposure to inhaled toxicants may impair lung growth and maturation																				
Behavioral influences												Adolescent drinking may lead to permanent impairments in cognitive function, and to eventual alcohol dependence.									
	Over-nutrition places girls at risk for early puberty, thus bypassing the time of peak growth in height. Undernutrition can lead to girls' delayed puberty leading to deficits in bone growth due to the lack of hormonal influences usually present during the period of peak bone mass accrual.												Poor nutrition and lack of physical activity may impact accrual of peak bone mass, leading to an increase in future risk of fracture and osteoporosis.								

life course may negatively impact future health outcomes by creating unstable and unpredictable environments.

Evidence for the three models

What does the current evidence say about the validity of the three models with regard to childhood SES and adult physical health outcomes? The review by Pollitt *et al.*⁷ discussed earlier revealed consistent support for both the timing and accumulation models. Specifically, low early life SES as well as increasing duration of low SES exposures throughout the life course each was associated with increased adult cardiovascular risk factors and cardiovascular morbidity and mortality.

Although compelling, these findings do not directly address the question posed here. The objective of the Pollitt *et al.* review was to evaluate the influence of SES *throughout the entire life course* on adult cardiovascular disease outcomes. Accordingly, studies included in the review that provided support for the accumulation model generally aggregated SES across one time point during childhood (or adoles-

cence) and one or more time points during adulthood. Thus, whether the sum total of socioeconomic disadvantage across childhood and adolescence confers risk for poor adult health independent of adult SES conditions cannot be determined. In regard to timing models, the importance of SES during childhood was clearly demonstrated by the evidence reviewed by Pollitt *et al.* However, because most of the reviewed studies collected early life SES data for only a single point during childhood or adolescence, we cannot know whether the measured time point constitutes a sensitive period for adult risk; the beginning of an accumulation of low SES exposures; or a point of downward inflection from previously higher early life socioeconomic conditions.

Future directions

Fundamental to examining the validity of the three models is including multiple assessments of early life SES throughout childhood and adolescence. Ideally, information on the same or comparable SES indicators (e.g., parents' occupation, household income) would be obtained at each time point.

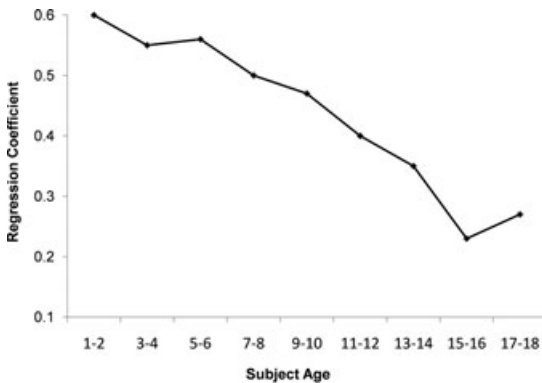


Figure 3. Adjusted effect size (regression coefficients) for the associations between parental home ownership and adult susceptibility to colds at different ages (two-year ranges) during childhood and adolescence. Reprinted with permission from Cohen, S., W.J. Doyle, R. Turner, *et al.* 2004. Childhood socioeconomic status and susceptibility to the common cold. *Psychosom. Med.* 66: 553–558.

Moreover, testing of the relative fit of the various models requires analyses comparing associations between adult health outcomes and SES at different ages during childhood and adolescence. For example, in a study we discussed earlier,¹¹ parental home ownership was assessed for each year of childhood and adolescence. Results of the study indicated that the fewer the years of parental home ownership during childhood and adolescence, the greater the risk of developing a common cold when exposed to a cold virus, thus providing evidence that seemed to support the accumulation model. However, study findings also showed that those whose parents did not own their home during their early life did not benefit from parental home ownership when they were adolescents. In fact, the size of the association of home ownership and adult susceptibility to colds decreased with increasing age of SES exposure (see Fig. 3), suggesting that home ownership during very early childhood was more important for adult health than ownership later in childhood and adolescence. In the final analysis, then, the data provided support for the timing model, while ruling out the accumulation model. Potential effects of upward and downward mobility were also excluded because changes in home ownership from childhood to adolescence were not associated with the risk of developing a cold. This level of detailed

analysis was made possible by the collection of SES data across multiple ages.

However, the Cohen *et al.* study¹¹ did not address other issues germane to fully understanding the influence of childhood socioeconomic circumstances on adult health outcomes. For example, home ownership does not reveal any information about each parent's contribution to the family's overall SES. It is possible that parents' unique SES may have differential implications for children's long-term health depending on whether it is the mother's or the father's status that is in question. For instance, if good health practices learned during childhood is an important pathway, one might imagine that the mother's education level would be as or more important than the father's. It is also possible that same-sex parent SES may have a greater impact in some cases (depending on mechanisms) than the opposite-sex parent.

Conclusions

SES exposures during childhood are powerful predictors of adult cardiovascular morbidity, cardiovascular mortality, all-cause mortality, and mortality due to a range of specific causes. Although there are scattered data on morbidities other than cardiovascular disease (e.g., upper respiratory infections, dental cavities), there is not enough evidence on other diseases at this point to make any strong inferences.

As we have outlined in this paper, there are many aspects of both physical and psychosocial environments that covary with early SES that could feasibly account for long-term effects on health. However, the existing empirical literature provides little evidence on the question of the mediation of early SES exposure on adult health. Being able to pinpoint the environmental characteristics associated with SES that contribute to long-term health would allow more efficient and effective preventive intervention. Moreover, being able to pinpoint the more proximal psychological, behavioral, and biological pathways that are driven by these environments could aid in designing interventions, but would also allow us to predict the types of health outcomes that should be influenced. For example, if lower SES children are at greater risk for coronary heart disease because they become lifetime smokers, than we might expect that they would have similar increased risk for

other diseases tied to smoking including lung cancer, respiratory problems, and strokes.

The models we examined present alternative and sometimes complimentary hypotheses about the ages at which childhood and adolescent SES matter most for adult health, and how long SES exposures of specific types need to last to influence health. To date, there are few empirical data testing the models against one another in the prediction of physical health. There are practical, conceptual, and analytical explanations for this lack of data. In the most practical sense, available studies of childhood SES and adult health are not designed to compare the relative fit of each model. First, there are very few studies that include more than one measure of SES during the period of childhood through adolescence. Second, even these studies seldom actually compare whether SES as measured at different ages in early life differentially predicts adult health. Finally, those studies that do test the accumulation model mostly ask a different question than we propose here. They generally average across all (early life and adult) assessments of SES, rather than comparing averaged early life SES to averaged adult SES.

On an analytic level, the interdependence of the three models presents several challenges that must be addressed in order for reasonable inferences to be made. For example, testing the accumulation model requires incorporating an appropriate control for SES *concurrent with* the outcome, as cumulative SES indices may in some cases be conflated with current SES. Testing the change model is especially challenging, as the only valid comparisons that can be made are between individuals of the same initial (or final) SES who either change levels of SES or have a stable SES between two time points. Other problematic statistical issues include determining when covariates should be measured; and whether controls should be included for family variables such as structure (e.g., single- vs. two-parent home; number of siblings) and stability. The latter two issues are complex in that child health and/or family variables may either contribute to or result from decreasing SES. A thorny issue is how to approximate childhood health. For example, in studies of heart disease in adulthood, should cardiovascular risk factors in childhood be included, even though the levels of risk are “normal”? In studies of adult mortality, what childhood measures are theoretically even relevant?

On a more conceptual level, it is important to emphasize that these models are more heuristics than independent testable hypotheses. First, more than one model could apply at the same time. For example, it is possible that there are cumulative effects of early SES exposure across childhood and adolescence (accumulation model), but that exposures at specific developmental periods are more impactful than those at others (timing model). Second, in some cases the models do not make independent predictions. For example, the upward mobility model may be viewed as a special case of accumulation with later rises in SES averaged together with initial low SES to result in a smaller dose of socioeconomic disadvantage relative to that experienced by those with consistently low SES.

Another conceptual issue concerns the possibility that both the mechanisms linking early SES with adult health outcomes and the models describing when during childhood and adolescence these mechanisms are most influential, vary across diseases or disease stages. For example, disease processes that are progressive, such as atherosclerosis, may fit the accumulation model, whereas those that involve exposure to a critical triggering event, such as early viral or antigen exposures associated with increased risk for adult respiratory disease, may fit the timing model. This hypothesis will require research examining a broader range of outcomes than assessed so far, as well as clear hypotheses about how such effects might occur at different ages.

Although these issues are challenging, they are, nonetheless, important to resolve. If the evidence continues to show associations of childhood SES and adult health, it is vitally important to know when the risk begins, what are the causal mechanisms in the physical and social environment correlated with low childhood SES, and which strategies based on understanding timing and mechanisms are most promising to reduce the long-reaching adversity of low childhood SES.

“The Child is the father of the Man.” 1802 William Wordsworth.

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Conflicts of interest

The authors declare no conflicts of interest.

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