

Poverty Grown Up: How Childhood Socioeconomic Status Impacts Adult Health

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ABSTRACT: Socioeconomic status and health status are directly related across the world. Children with low-socioeconomic status not only experience greater health problems in childhood but also aspects of their socioeconomic status become biologically incorporated through both critical periods of development and cumulative effects, leading to poor health outcomes as adults. We explore 3 main influences related to child's socioeconomic status that impact long-term health: the material environment, the social environment, and the structural or community environment. These influences illustrate the importance of clinical innovations, health services research, and public policies that address the socioeconomic determinants of these distal health outcomes.

(*J Dev Behav Pediatr* 31:154–160, 2010) **Index terms:** socioeconomic status, poverty, life course.

CLINICAL SCENARIO

Dylan is a 4-year-old boy who has been followed in primary care clinic since he was born at 34 weeks to an 18-year-old mother, who had moved into a shelter before his birth. Although he had a relatively short and uneventful course in the neonatal intensive care unit, his medical history has been complicated by multiple visits for wheezing, first in the context of bronchiolitis, now with a pattern suggestive of a diagnosis of asthma. He lives with his mother and his younger sister, Nevaeh, in public housing. On his third visit for wheezing in 6 weeks, his mother asks for a second round of steroids, noting that it is the only thing that seems to keep him from wheezing. She dreads having him on steroids because it makes his already hyperactive personality worse and asks the physician whether he should be evaluated for attention-deficit hyperactivity disorder (one of the teachers at his new pre-K had suggested that his learning problems might be related to inattention). In addition, she is wondering what, besides traps, she might do about the cockroaches in the unit, which seems to persist because of the trash in the hallway.

SOCIOECONOMIC STATUS AND HEALTH: OVERALL CONNECTIONS

Socioeconomic status and health are directly correlated across the world.¹ Children living in poverty are more likely to experience low birth weight, learning disabili-

ties, mental health problems, iron deficiency anemia, asthma, burns and injuries, obesity, and hospitalization than their more affluent peers.^{2–6} Likewise, adult health outcomes follow a spectrum that closely matches social status, and this gradient of health status persists at each step on the social ladder, including the highest steps.^{7,8}

There are myriad mechanisms to explain the connection between social status and health. The individual's material environment mediates exposure to toxins, infectious disease, and inadequate nutrition. For example, among low-income families, poor housing conditions may increase exposure to toxins and allergens, leading to intelligence quotient losses due to lead poisoning⁹ or lost-school days due to increased asthma morbidity.⁵ The surrounding social environment brings varying degrees of social support, stimulation, and stress. An overworked or very stressed single parent may use harsher discipline and speak with her child less, reducing verbal acquisition and readiness for school.¹⁰ Finally, community and social structure influence the quality of and access to health care and education that leads to poor health, poor education, and inadequate social capital. Social marginalization is also a breeding ground for community violence, a risk factor for serious injuries, posttraumatic stress disorder¹¹ and other sequelae of mental illness.

Examining Childhood Socioeconomic Status as an Independent Predictor of Adult Health

Strong connections link child health and adult health, as well as childhood socioeconomic status (SES) and adult SES. Considerable research also demonstrates a direct connection between childhood SES and adult health, regardless of whether a child manifests health consequences during childhood or changes social class from childhood to adulthood. Figure 1 depicts the multiple interactions among childhood SES, childhood health,

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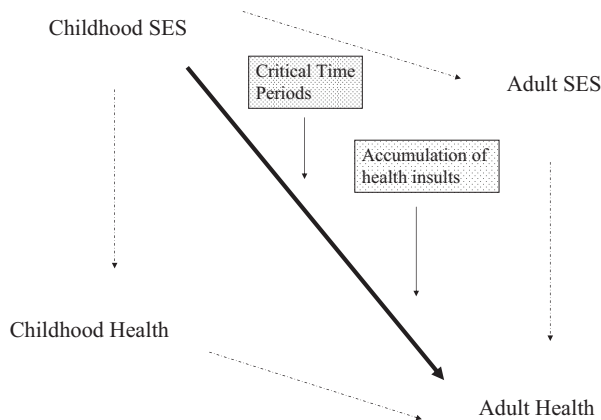


Figure 1. Pathways connecting the childhood socioeconomic status and adult health. SES, socioeconomic status.

adult SES, and adult health and highlights several of the foundational theories of how these states are connected.

The Whitehall I and Whitehall II studies conducted in Britain in the 1960s to 1980s demonstrate that risk of mortality from heart disease is correlated with social class even after controlling for known risk factors for mortality—such as smoking status, hypertension, and serum cholesterol.^{7,8} Said differently, the risks of mortality incurred by living in a lower social class cannot be explained solely by increased exposure to known predictors of mortality. Low social class itself—independent of the measured confounding factors—contributes to poor health outcomes. Data from the Whitehall studies also assessed the relative impacts of adult and childhood SES on adult health: although adult circumstances are critical, higher social class in adulthood does not completely erase the health impacts of living in a lower social class in childhood.^{12,13} Similarly, a longitudinal study of 1000 children in New Zealand concluded that low SES in childhood was predictive of differences in cardiovascular health, substance abuse, and periodontal disease at 26 years, regardless of SES at age 26.¹⁴ These findings suggest that elements of the early life experience associated with poverty have become “biologically imbedded¹⁵” and contribute to poor adult health.

“Life course” analysis examines how circumstances over a lifetime contribute to eventual health outcomes. Halfon and Hochstein,¹⁶ in describing the framework, state that health is determined by multiple “nested” factors, including physical, social, family, psychological, and cultural environments; the risk factors inherent in these environments become biologically incorporated into the individual and determine lifelong health trajectories.

Researchers in the MacArthur Foundation Network of SES and Health have described 2 complementary frameworks for understanding how childhood SES impacts adult health.¹⁷ The first theory proposes that there are “critical periods” for the development of certain organ systems or physiologic processes; changes that happen during these periods are irreversible,¹⁸ and thus the

physical and social environments experienced by the child during that critical time period have lasting effects on subsequent health. For example, low birth weight, known to be related to poor maternal nutrition,¹⁹ teenage pregnancy,²⁰ and maternal smoking²¹—all of which are more prevalent in low SES households—is a risk factor for cardiovascular disease among adults,^{22–25} possibly due to poor development of the pancreas (linked to later type 2 diabetes) and of the kidney (predisposing to hypertension).^{26–31} Low SES at birth is, then, a risk factor for adult cardiovascular disease, regardless of the SES of the individual at the age that problems occur. Poor health behaviors disproportionately seen among low SES adults further exacerbate early critical period change. The concept of “critical periods” is often described as the “latency model” of developmental health, highlighting that the health effects of these critical periods may not be recognized until much later.¹⁵

A second theory regarding how physical and social environments become biologically imbedded emphasizes the cumulative effects of multiple experiences. Evidence of this theory is the finding that while low-SES children recover as well from early health insults as higher SES children, they suffer more health insults over time, which leads to poorer health in adolescence and adulthood.¹⁵ McEwan,³² in his work on the connection between stress and health outcomes, calls the impact of multiple stressors “allostatic load.” In this framework, allostasis is the process of reestablishing equilibrium after a stressor. Each time the body goes through allostasis it pays a small price to reestablish equilibrium; the cumulative cost to the individual of managing stressors is allostatic load. Increased allostatic load is associated with poor health outcomes³² suggesting that some of the impact of low SES may become incorporated over time.

This cumulative effects of SES on health has also been called the “pathways” model (as opposed to the latency model) because the experiences of individuals that are damaging to their health are correlated or even causative of one another, “in a pathway or chain of risk.”^{18,33} For example, the “pathway” in the previous example from maternal smoking to low birth weight^{19,21} to adult cardiovascular disease involves both the inadequate organogenesis mentioned,^{26–31} and also follows a pathway through the effects of smoking on infant brain development,³⁴ contributing to learning and school problems, which are precursors to poor health behaviors such as subsequent smoking³⁵ that further contribute to adult cardiovascular disease.

Critical periods and cumulative experiences are complementary theories, and both take place within the life course of a given individual. In the above example, cigarette smoking by a mother during her pregnancy may have a critical effect on the brain of the developing fetus, but the impact on the child’s own subsequent smoking on his risk of cardiovascular disease exemplifies a cumulative risk factor.

CHILDHOOD SOCIOECONOMIC STATUS AND ADULT HEALTH OUTCOMES: SPECIFIC INFLUENCES

With the substantial pediatric and public health advances of the past century—vaccines, newborn screening, milk pasteurization, and clean water, to name a few—children are healthier, and many manifestations of the health consequences of the physical and social environment have moved from childhood to adulthood. The term “developmental health” has been used to describe the dynamism of this process, in which multiple forces within the environment interact with the individual’s physiology and genes, influencing the lifelong health trajectory.¹⁵ We will review some of the best-studied examples of how socioeconomic status (SES) becomes subsumed in the individual by exploring 3 different influences: the material environment, the social environment, and the community or structural environment in which the child lives.

Influence 1: The Material Environment

A growing body of evidence has called attention to the impact of the intrauterine environment—the child’s first physical environment—on adult obesity, type II diabetes, and hypertension, independent of adult-level risk factors for CV disease such as smoking, employment, alcohol consumption, and exercise.^{22,36–40} Clinical and animal studies support these epidemiologic findings and suggest potential mechanisms linking prenatal factors to low birth weight and to coronary artery disease. For example, fetal undernutrition or lower birth weight leads to fewer beta cells within the pancreas at birth,²⁶ predisposing for insulin “burn out.” In addition, fetuses living in low-glucose environments appropriately adapt by increasing insulin resistance,^{27,28} thus maintaining adequate serum glucose concentrations for the developing brain at the expense of other tissues. This adaptation can, however, lead to increased risk for type 2 diabetes in later life, especially if glucose is abundant in the environment.²⁶

Low birth weight is also associated with increased blood pressure in the adult.^{29,30} The likely mechanism for this hypertension—in addition to possible renal damage inflicted through diabetes—is that low birth weight infants have fewer nephrons at birth,³¹ leading to nephron-overload glomerulosclerosis⁴¹ and subsequent hypertension.⁴² The highest blood pressures among former low birth weight adults are found in those who were small as infants but overweight as adults, suggesting that the frequently linked adult diseases of obesity, diabetes, and hypertension may have a common origin in the neonatal period.⁴³

The development of asthma has also been tied to the early life material environment. Although there are multiple genes affecting the development of asthma, it seems that those regulating IgE synthesis and allergic inflammation require activation by the environment.⁴⁴ In addition, cockroaches and dust mites, more prevalent in

low-income homes, are risk factors for the development of airway hypersensitivity and subsequent asthma, likely through modulation of the early immune system in the direction of a more allergenic Th-2 like response.^{45–48} Naturally, once sensitivity to allergens such as cockroaches and dust mites occurs, further exposure will lead to increased asthma morbidity.⁴⁸ This interaction between the physical environment of the child and the developing pulmonary system has lifelong effects. When examined prospectively, young adults who experienced poverty as children have sharper declines in the lung function than their peers who were not raised in poverty (controlling for current SES).⁴⁹

Influence 2: The Proximal Social Environment

Similar to the material environment, the young child’s social environment becomes biologically imbedded in the growing child. Early infancy is characterized by intense brain development, which is exquisitely sensitive to interactions with caregiving adults.⁵⁰ Infants and children experiencing chronic stress or social deprivation demonstrate specific patterns of neurotransmitter release, leading to structural alterations in the brain development, and ultimately impacting memory, educational attainment, and ability to cope with subsequent stressors.^{50–52}

Specific physiological alterations caused by stress during early childhood highlight how the social environment becomes biologically subsumed. Rat pups not groomed frequently by their mothers in infancy have increased activation of the hypothalamic-pituitary-adrenal axis in response to subsequent mild stresses.⁵³ Studies indicate that this is due to methylation of the gene encoding for expression of the glucocorticoid receptor, which ultimately participates in feedback inhibition of the stress response. Those groomed show less methylation, express more glucocorticoid receptor, and then have greater feedback inhibition of the stress response and subsequent lower hypothalamic-pituitary-adrenal axis responses to stresses in adulthood.⁵⁴ Notably, methylation is found to be a stable response not altered in adulthood.⁵⁵ The same epigenetic control of the stress response is also described in humans: those abused in early childhood have been shown to have methylation at a key promoter site encoding the gene for the glucocorticoid receptor.⁵⁶ Fewer glucocorticoid receptors in the hippocampus reduces feedback inhibition as adults, leading to greater hypothalamic-pituitary-adrenal responses to stresses than their nonabused peers.

Prolonged cortisol release in response to stress is thought to result in hippocampal neuron damage and loss by increasing vulnerability to calcium and glutamate,⁵⁷ thus causing neural damage and death. This mechanism may explain the finding that increased cortisol levels are detrimental for learning and memory.^{58,59} In support of this explanation are magnetic resonance imaging scans showing atrophy of the hippocampus with stress-related conditions, including recurrent de-

pression and posttraumatic stress disorder arising from severe childhood physical and sexual abuse.⁶⁰

Children who experience abuse or neglect in the early social environment also display more high-risk health behaviors, such as cigarette smoking, alcohol abuse, and multiple sexual partners,⁶¹⁻⁶³ which are associated with alcoholism, sexually transmitted disease, unintended pregnancy, and suicide.^{61,64,65} This relationship was found to be graded: those who had experienced more adverse experiences or household dysfunction displayed more high-risk behaviors as adults.

Exposure to stress at a young age has consequences outside of the central nervous system. Low SES has been linked to long-term increased inflammatory markers (particularly C-reactive protein, fibrinogen, and white blood cells),⁶⁶⁻⁶⁸ which may be additional factors on the causal pathway between low SES in childhood and adult cardiovascular disease.^{67,68}

Influence 3: Structural and Community-Associated Forces

Community and structural forces influence health outcomes directly. The educational system plays a critical role in the lives of low-income children. Some portion of early school success is a function of the home environment: high-SES children have been shown to live in a more language-rich environment,⁶⁹ and therefore, to have better language skills at the start of school that contribute to success there.¹⁰ On the other hand, school quality likely relates to school success. Despite reforms in school financing, school district funding in many states is dependent on the income of those living in the district, and it has been shown that inequality in funding from district to district contributes to differences in achievement among children of different family backgrounds.⁷⁰ Although it is very challenging to separate out educational outcomes from other socioeconomic determinants of health, poor educational attainment has been consistently shown to be a risk factor for poor overall health quality as an adult.^{71,72} The duration of education has the strongest relationship to adult mortality.⁷³ In addition to the connection among education success, ultimate income, and access to healthier lifestyles,⁷⁴ education may also become “biologically imbedded” in the brain. Poor educational achievement is a risk factor for Alzheimer disease—a geriatric disease characterized by neurofibrillary tangles.^{75,76} It has been proposed that education promotes the development of neural connections that are protective against Alzheimer disease.⁷⁷

Race and ethnicity are also structural factors known to contribute to all-cause mortality. Although in this country there is considerable overlap of poverty with certain racial and ethnic groups, research consistently demonstrates that race and ethnicity play a role in health outcomes that cannot be explained solely through considerations of poverty, material resources, or access to health care.⁷⁸ Social marginalization and concentration of poverty may play a structural role in mediating these

disparate health outcomes among racial groups in the United States.^{79,80}

IMPLICATIONS FOR CLINICAL PRACTICE AND HEALTH POLICY

The impact of the material, social, and community environments experienced by poor children on their subsequent adult health has clinical, research, and policy implications. In attempting to address any of the socioeconomic determinants of disease from a clinical perspective, physicians need knowledge of the social determinant and its prevalence in their community of practice, a screening tool with appropriate test characteristics, and access to an effective intervention, as well as clinical time in which to affect this screening and address these needs.

Partnerships between health care professionals and other community resources have given physicians access to the knowledge and resources to address some social determinants of disease. The Medical-Legal Partnership, an innovation of the last decade, links physicians with legal aid lawyers who are effective at addressing legal barriers to reducing social determinants of disease.⁸¹ For example, a recalcitrant landlord can refuse to exterminate cockroaches, threatening the family with unfair eviction if they pursue enforcement. An immigrant mother can be asked for documentation of her legal residency when applying for food stamps for her citizen children, which is a violation of her rights and becomes a barrier for obtaining the needed foods stamps. Medical-Legal Partnership lawyers train physicians in identifying needs and then assist families in receiving benefits for which they are legally eligible.

Project HEALTH is another such clinical innovation, partnering physicians with college student volunteers trained to assist families in meeting basic needs like locating an available child care slot, applying for food stamps, or aiding in housing searches.⁸² Finally, myriad nurse-staffed home visiting programs have addressed a variety of child development outcomes among high-risk families⁸³; these programs have undergone more rigorous evaluation than either of the programs listed earlier, but these evaluations have shown mixed results.

These clinical innovations should come hand-in-hand with research questions. Do these clinical interventions actually meet families' basic needs, and, furthermore, do they result in desired health outcomes? Existing literature suggests direct child health benefits from food and income subsidies, but research has yet to establish direct health benefits from housing interventions.⁸⁴⁻⁸⁷ Observational research in this area faces numerous threats to validity because those families participating in clinical interventions or receiving existing social welfare benefits may, in fact, be different from those qualifying but not enrolled. One randomized clinical trial demonstrated that a clinically integrated screening program for basic social needs increased the likelihood that families would be connected with community resources; however, no

research has demonstrated a connection between screening for these needs in clinical settings and actual patient health outcomes.⁸⁸ In addition, although improvements in the social history have been suggested,⁸⁹ no validated clinically useful screening tool has been developed for housing, food, or income issues. This research will be necessary to prompt uptake and further funding of these clinical innovations.

Finally, the connections between childhood socioeconomic status and adult health suggest how critical social policies can be to health outcomes. Social programming such as Women, Infants, and Children (WIC), food stamps, public housing, Section 8 vouchers, and income-supplementing programs such as Transitional Aid to Families with Dependent Children work to protect low-income families against the health impacts—both proximal and distal—of poor socioeconomic conditions. Reframing these as interventions as necessary to health may be important to sustaining or growing public expenditures.

CONCLUSION

Growing evidence for the connection between childhood socioeconomic status and adult health puts pressure on medical practitioners, health service researchers, and social policy experts to collaborate in developing effective interventions for the childhood antecedents of adult health. Clinical partnerships may aid physicians in screening for and addressing the social determinants of health, when existing interventions exist. Researchers are charged with rigorously evaluating existing and future clinical innovations and social programming to determine its impact on the health of children and their subsequent health as adults. Finally, practitioners and researchers must collaborate in the policy arena to help reframe social policy as health policy and arm communities with interventions that work far upstream of adult health problems.

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