Stressing Out the Poor

Chronic Physiological Stress and the Income-Achievement Gap

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It is well known that economic deprivation early in life sets children on a trajectory toward diminished educational and occupational attainment. But why is early-childhood poverty so harmful? If we can‘t answer that question well, our reform efforts are reduced to shots in the dark.

In this article, we offer a new perspective on this question. We suggest that childhood poverty is harmful, in part, because it exposes children to stressful environments. Low-income children face a bewildering array of psychosocial and physical demands that place much pressure on their adaptive capacities and appear to be toxic to the developing brain. Although poor children are disadvantaged in other ways, we focus our analysis here on the new, underappreciated pathway depicted in Figure 1. As shown in this figure, children growing up in poverty demonstrate lower academic achievement because of their exposure to a wide variety of risks. These risks, in turn, build upon one another to elevate levels of chronic (and toxic) stress within the body. And this toxic stress directly hinders poor children’s academic performance by compromising their ability to develop the kinds of skills necessary to perform well in school.
accumulation occurs in various ways; for example, children who score poorly at age six may be tracked into low-achievement school groups, which in turn exposes them to lower expectations, to less rigorous curricula, and to less capable peers, all of which further disadvantage them and generate ever more substantial between-group gaps. The Risk–Stress Model, to which we turn later, suggests that such splaying may also be attributed to the cognitive deficits and poorer health that chronic stress generates. Both cognitive deficits and ill health then repeatedly disadvantage poverty-stricken children in one educational setting after another.

Pathway #1: Parenting Practices

What types of forces have social scientists conventionally understood as explaining the achievement gaps illustrated in Figure 2? One reason poor children lag behind their more affluent peers is that their parents interact with them in ways that aren't conducive to achievement. For example, psychologist Kathryn Grant and her colleagues have documented a strong and consistent relation between socioeconomic disadvantage and harsh, unresponsive parenting. In one national dataset, 85 percent of American parents above the poverty line were shown to be responsive, supportive, and encouraging to their children during infancy and toddlerhood, whereas only 75 percent of low-income parents had the same achievement-inducing parenting style. While most low-income parents (i.e., 75 percent) do provide adequate levels of support and encouragement, these data reveal, then, a nontrivial difference across income levels in the chances that children will experience a problematic parenting style. There is considerable evidence that at least a portion of the cognitive developmental consequences of early childhood poverty is due to this difference.

Pathway #2: Cognitive Stimulation

It's also well known that children from low-income households tend to receive less cognitive stimulation and enrichment. For example, a child from a low-income family who enters first grade has been exposed on average to just 25 hours of one-on-one picture book reading, whereas an entering middle-income child has been exposed on average to more than 1,000 hours of such reading. Likewise, during the first three years of life, a child with professional parents will be exposed to three times as many words as a child with parents on welfare.

We will unpack this new Risk–Stress Model in the balance of our article. However, before doing so, it's useful to first go over the evidence regarding the relation between poverty and achievement and then to present some of the well-known pathways through which this relationship is generated. With that background in place, we can then describe the Risk–Stress Model, as represented in Figure 1.

**Poverty and Achievement**

It is well known that children born into low-income families lag behind their middle- and upper-income counterparts on virtually all indices of achievement. To provide one example, a national study of elementary school children shows that children in the poorest quarter of American households begin kindergarten nearly 10 percent behind their middle-income and affluent classmates in math (Figure 2). Six years later, as they are about to enter middle school, the poorest quarter of American children have fallen even further behind, with the gap between themselves and their most affluent schoolmates nearly doubling.

The splaying pattern revealed here, a general one that holds across various outcomes, may be attributed to the tendency for advantage and disadvantage to accumulate over time. This
tive development. The key concern here: Children from impoverished households face a wide array of physical and psychosocial stressors. Their homes, schools, and neighborhoods are much more chaotic than the settings in which middle- and upper-income children grow up. Such conditions can, in turn, produce toxic stress capable of damaging areas of the brain known to underlie cognitive processes—such as attention, memory, and language—that all combine to undergird academic success. In the pages that remain, we document each of the steps in the Risk–Stress Model.

**Poverty and Cumulative Risk Exposure**

The stressors that poor children face take both a physical and psychosocial form. The physical form is well documented; poor children are exposed to substandard environmental conditions including toxins, hazardous waste, ambient air and water pollution, noise, crowding, poor housing, poorly maintained school buildings, residential turnover, traffic congestion, poor neighborhood sanitation and maintenance, and crime. The psychosocial form is also well documented; poor children experience significantly higher levels of family turmoil, family separation, violence, and significantly lower levels of structure and routine in their daily lives.

An important aspect of early, disadvantaged settings may be exposure to more than one risk factor at a time. A powerful way to capture exposure to such multiple sources of stress and strain is the construct of cumulative risk. Although there are various ways to quantify cumulative risk, one common approach is to simply count the number of physical or psychosocial risks to which a child has been exposed. In one UK study, the authors counted how often children were exposed to such stresses as (a) living with a single parent, (b) experiencing family discord, (c) experiencing foster or some other form of institutional care, (d) living in a crowded home, and (e) attending a school with high turnover of both classmates and teachers. It was found in this study that inner-city children experienced far more of these stresses than did the better-off working-class children. The same result holds in the United States (see Figure 3). In rural New England, only 12 percent of middle-income nine-year-olds experienced three or more physical and psychosocial risk factors, whereas nearly 50 percent of low-income children crossed this same threshold (of three risk factors).

In a national U.S. sample of premature and low birth weight infants, Brooks-Gunn and colleagues similarly found that infants born into low-income families experienced nearly three times more risk factors than their middle-income counterparts by the time they were toddlers. These same low-income toddlers were seven times more likely than their affluent counterparts to experience a very high number of risk factors (≥ 6). The pattern is overwhelmingly clear: Being born into early poverty often means exposure to many more physical and psychosocial risk factors.
Cumulative Risk Exposure and Chronic Stress

But does such differential exposure indeed result in higher stress levels among poor children? The simple answer is that it does. In cross-sectional analyses of 9- and 13-year-old children, Evans and colleagues found that the risk exposure described in Figure 3 elevated baseline, resting blood pressure as well as overnight indices of such stress hormones as cortisol. At age 13, when challenged by mental arithmetic problems, children with higher levels of cumulative risk exposure did not show a typical healthy response, instead exhibiting a muted rise in blood pressure. These same children also didn’t recover as successfully from the mental challenge posed by these arithmetic problems (as indexed by the longer time it took their blood pressure to return to pre-stressor baseline levels). The evidence thus suggests that children exposed to high levels of cumulative risk are less efficient both in mobilizing and then shutting off physiological activity.

The Risk–Stress Model, as represented in Figure 1, implies that the effect of family poverty on stress is mediated by risk exposure. Although one would ideally like to test that mediation, it’s also important to simply document the association between poverty and stress (thereby ignoring the mediating factor). Many investigators have indeed documented that disadvantaged children have higher chronic physiological stress levels, as indicated by elevated resting blood pressure. A smaller number of studies have also uncovered higher levels of chronic stress hormones, such as cortisol, among disadvantaged children. To provide just a few examples, Figures 4 and 5 show elevated resting blood pressure as well as higher overnight urinary stress hormones in a sample of nine-year-old rural children.

The foregoing data, which pertain to nine-year-olds, don’t tell us when such stress symptoms emerge. Do poverty-stricken children show evidence of elevated stress early on in their lives? Or do such symptoms only emerge later? With support from the Stanford Center for the Study of Poverty and Inequality, we sought to answer this question by reanalyzing a national data set of very young at-risk children. The Infant Health and Development Program (IHDP) is a representative sample of low birth weight (≤ 2500 grams) and premature (≤ 37 weeks gestational age) babies born in 1985 at eight medical centers throughout the country. This sample of nearly 1,000 babies is racially and economically diverse (52 percent Black, 37 percent White, 11 percent Hispanic).

We assessed resting blood pressure and child’s height and weight at 24, 30, 36, 48, 60, and 78 months of age. The collection of physical health data at such young ages and over time provided us with an unprecedented opportunity to examine the early trajectories of chronic stress among a high-risk sample of babies. Both baseline blood pressure levels and Body Mass Index (BMI) reflect wear and tear on the body and are precursors of lifelong health problems. The former is indicative of cardiovascular health and the latter of metabolic equilibrium. BMI, which reflects fat deposition, is measured as height divided by weight (kg/m²).

We sought to assess whether these two measures of stress are elevated in poverty-stricken neighborhoods. Low-income neighborhoods, as defined in our study, have median household incomes below $30,000 (in 1980 dollars), while middle income neighborhoods have median income levels exceeding $30,000 per household. As is evident in Figures 6 and 7, babies growing up in low-income neighborhoods have health trajectories indicative of elevated chronic stress. Additional statistical controls for infant birth weight, health, and demographic characteristics did not alter these trajectories. These figures also reveal, even more importantly, that elevated stress emerges very early for children growing up in low-income neighborhoods. BMI,
for example, proves to be unusually low among poor children under five years old, but it then takes off as these children grow older. The blood pressure measure, by contrast, registers high among low-income children from almost the very beginning of our measurements (i.e., 24 months). This research confirms, then, that low-income children are more likely than others to develop dangerous stress trajectories very early on in their childhood. As we discuss below, this has profound consequences for their likelihood of success in school and beyond.

**Chronic Stress and the Achievement Gap**

The next and final step in our chain model pertains to the effects of chronic stress on achievement. Here we turn to an important longitudinal program on poverty and the brain at the University of Pennsylvania conducted by Martha Farah and her colleagues. In a series of studies with multiple samples drawn from lower- and middle-class Black families in Philadelphia, Farah and colleagues show that several areas of the brain appear vulnerable to early childhood deprivation. Using batteries of neurocognitive tests of brain function and brain imaging studies, Farah and other neuroscientists can map the areas of the brain that are recruited by neurocognitive tasks. As shown in Figure 8, among the areas of the brain most sensitive to childhood SES are language, long-term memory (LTM), working memory (WM), and executive control. What the graph depicts is the separation, in standard deviation units, between a low- and middle-SES sample of 11-year-old Black children from Philadelphia. For this sample, one standard deviation represents about one-fifth of the total distribution of scores. Samples differing by 3.5 or more standard deviations are virtually non-overlapping. Given that the samples differ by about 3.5 standard deviations for all four areas of brain functioning, this means that there is virtually no overlap between poor and middle-class Black children when it comes to language, long-term memory, working memory, or executive control. Eleven-year-old Black children from lower SES families reveal dramatic deficits in multiple, basic cognitive functions critical to learning and eventual success in society. These results reveal the starkly cognitive foundation to the poor performance of low-income children.

But is this achievement gap attributable to cumulative risk and chronic stress? With a recent follow-up of the sample depicted in Figures 4 and 5, Evans and colleagues have now provided the first test of the final link in the Risk–Stress Model. The baseline finding from their research is that working memory in early adulthood (i.e., age 17) deteriorated in direct relation to the number of years the children lived in poverty (from birth through age 13). If, in other words, a child lived in poverty continuously, his or her working memory was greatly compromised. The main result of interest, however, was that such deterioration occurred only among poverty-stricken children with chronically elevated physiological stress (as measured between ages 9 and 13). That is, chronic early childhood poverty did not lead to working memory deficits among children who somehow avoided experiencing the stress that usually accompanies poverty.

**Conclusion**

Childhood socioeconomic disadvantage leads to deficits in academic achievement and occupational attainment. It's long been argued that such deficits arise because poor children are exposed to inadequate cognitive stimulation and to parenting styles that don't encourage achievement. We don't dispute the important role of these two variables. But we have outlined here evidence for a new, complementary pathway that links early childhood poverty to high levels of exposure to multiple risks, which in turn elevates chronic toxic stress. This cascade can begin very early in life. Even young babies growing up in low-income neighborhoods already evidence elevated chronic stress. This stress then accounts for a significant portion of the association between poverty and working memory, a critical cognitive skill involved in language and reading acquisition.

The Risk–Stress Model suggests that the poverty–achievement link can be broken by addressing (a) the tendency of poverty to be associated with physical or psychosocial risks (e.g., environmental toxins, family turmoil), (b) the effects of such risks on stress, and (c) the effects of stress on achievement. If this model bears up under further testing, it would be useful to explore which of these pathways is most amenable to intervention.

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