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Distinctive Mechanisms of Adversity and Socioeconomic Inequality in Child Development: A Review and Recommendations for Evidence-Based Policy

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Abstract

This review proposes separate and distinct biological mechanisms for the effects of adversity, more commonly experienced in poverty, and socioeconomic status (SES) on child development. Adversity affects brain and cognitive development through the biological stress response, which confers risk for pathology. Critically, we argue that a different mechanism, enrichment, shapes differences in brain and cognitive development across the SES spectrum. Distinguishing between adversity and SES allows for precise, evidence-based policy recommendations. We offer recommendations designed to ensure equity in children's experiences to help narrow the achievement gap and promote intergenerational mobility.

Keywords

development, poverty, SES, achievement gap, intergenerational mobility, executive functions, policy

Tweet

Distinguishing the effects of adversity and SES on child development is critical for improving health and narrowing the achievement gap

Key Points

- The impacts of adversity and SES disparities are separate issues requiring separate evidence-based policy recommendations for child development.
- Prolonged early-life adversity affects the biological stress response and confers risk for pathology.
- Social buffering and exercise reduce stress and buffer again the effects of adversity.
- Wealth, *more than poverty*, may be driving SES-based disparities in brain and cognitive development, and educational achievement outcomes.
- Cognitive enrichment and early exposure to linguistic complexity can narrow the achievement gap.

Introduction

We argue that the effects of stress caused by adverse experiences that occur more often for impoverished families—including neighborhood violence, parental substance abuse, maltreatment, and homelessness—should not be conflated with the effects of socioeconomic status (SES) that exist *independent of adversity*. The distinction between effects of adversity and effects of SES is critical for science, society, and policy. For science, understanding individuals' everyday experiences offers us an opportunity to specify mechanisms more precisely. For policy, mechanistic precision offers opportunity for effective investment in programs that work to alleviate the specific problems children and families experience, preventing long-term economic waste. For society, reducing inequalities in intergenerational mobility, with respect to adversity and SES, allows for a more equitable and productive society. Of particular concern is that equating relatively lower SES with adversity risks creating a societal power construct around wealth, and risks erroneously stereotyping children from low- and middle-income families, *who make up the vast majority of the American population*, as at high risk for suffering a negative biological fate.

First, we briefly characterize the American income distribution. We then review scientific literature on early adversity more commonly experienced in poverty, followed by a review of scientific literature examining the effects of SES across the spectrum. We finally offer evidence-based recommendations on what arguably are separate mechanisms that require different interventions.

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Income Distribution in America

According to the National Center for Children in Poverty (http://www.nccp.org/), more than 22% of American children live in poverty, meaning that the needs of 16 million children cannot be met with their family income. This wealth gap mirrors the achievement gap. SES reflects the social standing of a family, and is typically measured as parent education, occupation, and income (McLoyd, 1998). Across the *entire income spectrum*, children from higher income communities are substantially more likely to attend college (Chetty, Hendren, Kline, & Saez, 2014). Attendance rates greatly improve if children living in lower-income neighborhoods move to higher-income neighborhoods, where they receive the full benefit of better schools and more opportunities for enriching experiences (Chetty & Hendren, 2017). This neighborhood effect applies to those living in poverty versus immediately above the poverty line in the same way that it applies to the affluent versus extremely wealthy.

To properly interpret the literature on adversity, poverty, and SES, we first report the typical range of income in America. According to the Census Bureau website (www.census.gov), in 2015, only about 12% of Americans made greater than US$150,000 per year, and 6.1% greater than US$200,000. Most American households made between US$50,000 and US$75,000 per year. Poverty is specifically defined by income and household size. In 2015, a family of four (with two children) making less than US$24,036 would be considered living in poverty, whereas a family of nine people, with seven children, making less than US$45,822 would be considered living in poverty.

Effects of Adverse Experiences More Common in Poverty

Children growing up in poverty experience more frequent, cumulative stressors including maternal depression and stress, family transitions, neighborhood violence, household crowding, maltreatment, and exposure to toxins (Mills-Koonce & Towe-Goodman, 2012). These exposures early in life, *with or without poverty*, increase the risk that a child will later develop mental and physical health problems, including depression, substance abuse, and suicidality (Green et al., 2010). With respect to cognitive and affective development, early-life adversity impairs three neural systems: the hippocampus, amygdala, and prefrontal cortex (PFC; McEwen, 2008).

The hippocampus is involved in learning and memory, and its functions are negatively influenced by early-life adversity across late childhood through adolescence (Carrión, Haas, Garrett, Song, & Reiss, 2010; Güler et al., 2012). Early-life adversity also affects the structure and function of the amygdala, associated with fear and threat detection (Tottenham & Sheridan, 2009), increasing the risk for anxiety and mood disorders (Dvir, Ford, Hill, & Frazier, 2014). Finally, early-life adversity affects both the structure and function of the PFC (Bick et al., 2015; Carrion & Wong, 2012; Mclaughlin et al., 2014), which is involved in executive control over thoughts, actions, and emotions. Our work with young children shows maltreatment affects executive functions and emotional control by age 6 (Werchan et al., 2017).

How Adversity Affects Development: The Biological Embedding of Stress

The prevailing hypothesis is that adversity affects development through the biological embedding of chronic, toxic stress in the brain (McEwen, 2008). Survival depends on identifying and reacting to threats to safety. Adversity activates the neurobiological stress response, which is measured as changes in the steroid hormone cortisol, which is measurable in blood and saliva. The first system activated in this stress response is the immediate "fight or flight" response. The second system is the slower response by the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis leads to the release of cortisol, which temporarily increases energy usage, enhances cognitive abilities, and strengthens immune reactions (McEwen, 2002). Long-term exposure, however, may reverse these effects, leading to metabolic disorders, worsened cognitive functions, and weaker immune responses (McEwen, 1998).

Identifying a stressor in a child's environment need not indicate negative outcomes. It is only when the stressor becomes chronic or toxic that the developing system begins to adapt to it as the "new normal." This can change the biology of the stress response (Mills-Koonce & Towe-Goodman, 2012), causing neural remodeling that has negative long-term consequences on how well that individual can function in a biologically and psychologically demanding world, thus posing a risk for psychopathology (McEwen, 2008). The hippocampus, amygdala, and PFC are rich in receptors that bind to cortisol, making these regions particularly susceptible to structural and functional changes as a result of toxic stress (McEwen, Nasca, & Gray, 2016).

A shift in the stress response is a consequence of the early adversity experienced by children living in poverty (Blair, Raver, Granger, Mills-Koonce, & Hibel, 2011). Children who have disrupted caregiving experiences have lower levels of cortisol, as well as less change in cortisol over the course of the day, than is typically measured in children without these disruptions (Tarullo & Gunnar, 2006). These effects have a cumulative impact on the developing brain. Assessing the impact of cumulative exposure to physical and social stressors in young teens, cortisol elevation correlated with the number of years living in poverty and exposure to adverse experiences (Evans & Kim, 2007). In a similar relation between cortisol levels and poverty duration, this cumulative exposure was inversely related to working memory, a PFC-dependent executive function, in young adults (Evans &

Schamberg, 2009). The cumulative impact of stress on developing systems is also evident in children with prenatal substance exposure and domestic violence (Lester et al., 2010) and children who have suffered abuse (for review, see Mills-Koonce & Towe-Goodman, 2012).

Summary

Prolonged early-life adversity affects the biological stress response, measured by variations in cortisol, and affects memory, emotion, and executive functions development. Adversity also puts a child at risk for later pathology. What this literature does not say is that all children from very low-income homes are experiencing maltreatment or poor care. A family need not be living in poverty to impose significant stress on a child, such as from divorce (Kraft & Luecken, 2009). Stressful experiences can happen to anyone, but a higher number is more commonly associated with living in poverty, and the effects seem to be cumulative.

Effects of SES Span the Poverty-to-Wealth Spectrum

Studies have examined the effects of SES on both brain and cognitive development. Here, we argue that stress is not a likely causal mechanism for SES effects seen along the entire spectrum. Wealth's advantages, *more than poverty*, are driving many SES-based differences in brain and cognitive development, and educational achievement outcomes.

SES influences both structural brain (Hanson et al., 2013) and cognitive (Amso, Haas, & Markant, 2014; Markant, Ackerman, Nussenbaum, & Amso, 2016) development as early as infancy. SES influences developmental outcomes in the domains of language, memory, and executive function (Hackman & Farah, 2009; Noble, McCandliss, & Farah, 2007; Noble, Norman, & Farah, 2005). SES affects executive functions in the transition from childhood to adolescence (Amso, Haas, McShane, & Badre, 2014), and memory (Markant et al., 2016) and face processing (Amso et al., 2014) beginning in infancy. Importantly, long-term achievement outcomes are shaped by children's executive functions (e.g., Blair, 2002).

In 1995, Hart and Risley showed that children from high-income families have much more rapid vocabulary growth than children from middle-income families, who in turn have more rapid vocabulary growth than children from families living in poverty. They argued that this affected language skills, IQs, and subsequent academic success. The majority of American children (which, as detailed above, are from low- and middle-income families) had vocabularies that grew at steady rates. However, there was a larger gap between them and the highest-income children, who made up a small proportion of the U.S. population that year. Many others have since shown similar effects of SES on language development (see Brito et al. for full review in this issue). For example, across a wide range of SES (as measured by parent education), differences in language have been found before age 2, and language and literacy at home partly explained this effect (Noble et al., 2015). These data reflect more enriched language environments with higher parent education.

SES has been linked to individual differences in executive function development in childhood and adolescence. Both positive and negative environmental experiences have been shown to affect the structure and function of the PFC (Hackman, Gallop, Evans, & Farah, 2015). For example, the effect of SES on executive functions in preschool-age children is mediated by positive practices in a child's home environment. The complexity of spoken language in the home and participation in literacy activities with parents mediate the relation between SES and executive functions (Hart & Risley, 1995; Noble et al., 2007). A study using a group of children from a wide SES range found that SES was the strongest predictor of language performance and that this relationship most strongly mediated the relationship between SES and working memory, an executive function (Noble et al., 2007). Other variables that mediate SES effects on child development include parental support, neonatal health, and maternal sensitivity, among others (Blair, Granger, et al., 2011; Evans & Schamberg, 2009; Hackman et al., 2015; Sarsour et al., 2011).

Striking findings come from studies that examine the relationship between SES and structural brain development. Two studies have used the Pediatric Imaging, Neurocognition, and Genetics (PING) Study to examine this relationship. The PING study is a multisite structural neuroimaging study of more than 1,000 participants aged 3 to 20 years. Family income ranges from US$4,500 to US$325,000, with a mean of US$97,617 (*SD* = US$76,719). Parent education ranges from 6 to 18 years, with a mean of 15 years (*SD* = 2.25 years). One study used this sample to determine that age-related changes in cortical thickness, a gross measure of brain development, vary by SES (Piccolo, Merz, He, Sowell, & Noble, 2016). In particular, family income and parent education separately interacted with age-related changes in cortical thickness. Children growing up in families with the lowest (US$4,500-US$25,000) and middle (US$35,000-US$75,000) incomes showed a greater U-shaped trajectory between 3 and 20 years than children with the highest family income (US$125,000-US$325,000). Thus, during adolescence, low- and middle-income children had more rapid cortical thinning than children raised in high-income homes. The only group to deviate from the others was the high-income group (26% of American households). Another study tested a group of teenagers from "low-income" (US$22,665-US$70,041) relative to high-income (US$122,461-US$168,470) families (Mackey et al., 2015). They similarly found that cortical thickness was greater in the high-income group compared

with the low-income group in certain cortical regions. Higher standardized test scores were also associated with greater cortical thickness. Similar to Hart and Risley (1995), both studies show that the cortical thinning trajectory was only different in the high-SES group. These data indicate that at a population level, the highest-income group is developing out of the typical range. Perhaps *wealth affects cognitive development and cortical thickness to a greater extent than poverty.*

The processes underlying cortical thinning cannot be determined using these gross measures derived from imaging techniques. There are certainly neural events underlying thinning, but they are not measurable in humans. Some argue that cortical thinning over development reflects pruning away inactive connections (Mclaughlin, Sheridan, & Nelson, 2017). That means that thicker cortices reflect more learning experiences that maintain these connections in children from the highest-income homes (Mclaughlin et al., 2017).

A different neural structure measure, white matter development, is used to indirectly index the efficiency of communication between regions. White matter development has been considered through the lens of SES and specifically whether white matter explains SES-related differences in executive functions (Ursache & Noble, 2016). Family income, but not parent education, interacted with white matter volume. White matter volume was found to be positively correlated with executive functions in low-income individuals, but this correlation diminished as SES increased. Thus, higher SES might have a protective effect on brain development (Ursache & Noble, 2016). That is, children with lower white matter volume (ostensibly for genetic or biological reasons) raised in higher-SES homes may be protected against the impact of SES on executive functions, whereas those in lower-SES homes may not benefit from this environmental buffer.

Socioeconomic Impacts Are Independent of the Biological Embedding of Stress

Critically, SES can affect development independent of stress caused by adversity. A study that examined the impact of SES on working memory in children and adolescents found that parent education influenced working memory performance in childhood, but this effect was not cumulative and did not influence working memory development into adolescence (Hackman et al., 2014). They also found that SES-related neighborhood stressors had no impact on working memory performance and development. From this, they argued that SES effects on working memory cannot be due to adversity because the effect of parent education was consistent across a large education range and were not specific to very low educational levels. Consistently, another study asked whether childhood maltreatment and/or SES were predictive of adult hippocampal and amygdala volumes (Lawson et al., 2017). They found that only maltreatment predicted lower hippocampal volumes. SES was not related to either volume.

While early-life adversity has been linked to cortisol differences, studies examining the effects of SES on cortisol have largely found no evidence of this relation. Cutuli, Wiik, Herbers, Gunnar, and Masten (2010) examined cortisol differences in a group of children living in family emergency shelters. They asked whether morning cortisol levels varied by adversity and/or SES. Adversity predicted levels of morning cortisol and change in cortisol over the day, whereas SES was not associated with either cortisol measure. Another study of cortisol levels in a large group of teenagers found no association between SES and cortisol levels (West, Sweeting, Young, & Kelly, 2010). Indeed, a review of 26 articles examining this relationship found weak evidence, if any, to support the claim that SES affects brain development through cortisol (Dowd, Simanek, & Aiello, 2009).

One exception examined whether hair cortisol levels in children and their parents were related to differences in internalizing disorders (e.g., depression, anxiety; Ursache, Merz, Melvin, Meyer, & Noble, 2017). Lower parent education, but not lower income, was associated with higher hair cortisol levels for both parents and children. Moreover, the effect was linear along the entire education spectrum. A college graduate and his or her child have higher hair cortisol than those with a master's degree, who have higher hair cortisol than those with a PhD. Taken together with the lack of association with income, it is not clear what inference can be drawn about the biological embedding of stress from these data.

Summary

This brief review shows that (a) SES effects span the entire income and education spectra and are not relegated to poverty and (b) the stress response, as indexed by cortisol, is not clearly related to SES across either the education or income spectra. Alternatively, enrichment opportunities that are more available in higher-SES homes in the form of greater language complexity, education quality, travel experiences, and exploration with caregivers may progressively shape brain and cognitive development in powerful ways (Conger & Donnellan, 2007; Hackman et al., 2015; Mclaughlin et al., 2017). In effect, social and cognitive enrichment is nourishment for the developing brain, and more nourishment is better.

Thus, children who do not live in wealthy homes, which is the vast majority of American children, *are not at more risk for pathology because they suffer more stress and because they do not have as many resources.* The real problem this literature points to is that of the achievement gap. Children who have relatively more enrichment opportunities during early development will be more competitive for positions in upper-tier schools and score better on standardized tests than children who have fewer enrichment opportunities. This achievement gap has wide-ranging ramifications not just for these children, but for the growth of communities and nations.

Evidence-Based Policy Recommendations

Finally, we offer evidence-based policy recommendations that are made cost-effective by their specificity to the problems they aim to improve. We offer recommendations that are designed to buffer the stress response in children experiencing early-life adversity associated with extreme poverty. We then offer recommendations for alleviating the SES achievement gap in future generations.

Recommendations for Children Facing Early-Life Adversity

Evidence suggests that dampening the cortisol response to adverse experiences may diminish the neural remodeling that comes with a long-term shift in stress reactivity. The first factor that helps dampen this response is social buffering. The best evidence for this comes in the form of close relationships with caregivers (for review, see Hostinar & Gunnar, 2013). Hostinar, Johnson, and Gunnar (2015) define social buffering as "a reduction in acute physiological stress responses with the presence or assistance of a conspecific during an otherwise stressful event" (p. 281). Caregivers have been shown to be powerful social buffers, or external regulators of HPA activity, for their children as early as infancy (Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996) and well into early adolescence, but they are not as effective in late adolescence (Hostinar et al., 2015). Other studies have shown that sensitive parenting plays an important role in lowering cortisol levels in children, which affects executive functions by the age 3 (Blair, Granger, et al., 2011).

One recommendation is to invest in programs that educate caregivers on how their stress response affects their child's development and how they can display more sensitive parenting behaviors. A recently developed intervention for American foster parents, the Attachment and Biobehavioral Catch-Up for Toddlers (ABC-T), provides foster parents with an opportunity to support their foster children's self-regulation development (Lind, Lee Raby, Caron, Roben, & Dozier, 2017). Foster children who received this intervention showed similar self-regulation outcomes to children who were never in foster care, and they outperformed foster children whose parents never received the ABC-T intervention. A similar result was obtained when nurses visited disadvantaged pregnant mothers, and later their infants at home (Olds, 2006). Results showed a simple informative health-parenting interaction improved children's outcomes 12 years later, and resulted in reductions in substance use and mental health problems, relative to children from the same backgrounds but without early nurse visits.

A second important factor that helps to dampen the stress response is exercise. Studies using animal models demonstrate that physical activity has positive effects on the HPA-axis responsivity to stressors. Exercise results in both a shortened HPA response and reduced anxiety following a stressor (Hare, Beierle, Toufexis, Hammack, & Falls, 2014). As discussed, it is the body's response to the stressor, not the stressor per se, that begins the wear and tear process. A meta-analysis of the effects of children's aerobic activity found positive impacts on cognitive and psychological outcomes (Lees & Hopkins, 2013). Thus, exercise may serve as a buffer against the stress response even in the presence of a toxic stressor. In short, *exercise may be a mechanism of resilience*. This is a key finding from a policy perspective. Daily exercise of reasonable duration, incorporated into the school curriculum in the form of recess and physical education, may have lasting effects on both the mental and physical health of children at high risk for experiencing adversity. For these evidence-based reasons, American schools can enhance physical activity aspects of their curricula.

Recommendations for Narrowing the Socioeconomic Achievement Gap

The SES-based achievement gap creates two societal problems. At a national level, we risk the intergenerational perpetuation of a social-class structure. For the individual, the gap limits children with fewer enrichment opportunities from reaching their full potential. Early exposure to linguistic complexity and enriching experiences for learning and nourishing the developing brain are critical to narrowing this achievement gap.

Powerful data suggest that shifts in enriching learning experiences, through school, home, or neighborhood environments, have a strong positive effect on achievement outcomes. Data from millions of children were used to examine income in adulthood as a function of where children grew up (Chetty & Hendren, 2017). Results showed that, for the lowest 25% of the income distribution, each year living in a 1 *SD* better county than the one they were born into resulted in a 10% increase in their adult income. These effects were similar for other outcomes, including college attendance, teenage birth rates, and marriage rates. In most cases, these better neighborhoods had equal or lower rents, indicating that the results cannot be attributed to family income per se. Rather than income, data illustrate the importance of infrastructure, less segregation and income inequality, and better play spaces and schools (Chetty et al., 2014) for intergenerational mobility, putting the onus on policies for improvements. These data show that moving to a better neighborhood will result, for the same child, parents, and family income, in long-term higher SES in proportion to the amount of time spent in the better neighborhood. An investigation of whether public spending in 25 countries affected test scores among teens found that increased public spending on health, housing, education, and family support resulted in less inequality in test scores and less of an emphasis on family background in determining these achievement outcomes (Aizer, 2014).

The key to the effects observed above is, the earlier the intervention, the better the outcome. Better schools, in particular, offer important avenues for enrichment opportunities that can power learning, and brain and cognitive development. In a 2011 study, researchers implemented the Chicago Readiness School Project in 35 Head-Start-funded classrooms (Raver et al., 2011). This project, which taught children how to engage in self-regulation, had a positive effect on children's vocabulary, math, and early literacy skills. Indeed, involvement in structured sequential activities, through play, arts, and skills development, is key to executive functions development (Diamond & Lee, 2011), known to be critical for academic achievement and success in math and reading (Lawson & Farah, 2017). A tremendous amount of change occurs from age 3 to 5 in executive functions (Davidson, Amso, Anderson, & Diamond, 2006). Yet, according to the Education Commission of the States (http://www.ecs.org), 35 states do not require children to attend kindergarten. The majority of states require children to enroll in school by age 6 (26 states) or even 7 (14 states). State-funded pre-K programs, which have been shown to be effective for long-term academic outcomes (Ansari & Winsler, 2016), are still not available in all states or to all children within a state. These policies are inconsistent with evidence that SES affects development beginning in infancy (Amso et al., 2014; Markant et al., 2016).

A delay in schooling or offering enrichment opportunities outside the home will additively affect children from the highest SES least and the lowest SES most. Schools offer opportunities not only for learning language, early literacy, and social interaction but also for exploring different books, toys, and outdoor play equipment that are not commonly experienced at home. Investments in improving school quality are thus imperative. To narrow the achievement gap, schools in lower-income communities must proportionally *compensate* for the resources not otherwise present in the child's home, including museums trips, educational toys, projects for exploration, exposure to music classes, and organized team sports. Narrowing the achievement gap also means schools in lower-income communities must be proportionally *better than*, not just on par with, schools in higher-income communities. Evidence from a large study of children in Tennessee suggests that teacher quality and size of kindergarten classrooms, *in the same school*, affected college attendance and earnings in adulthood (Chetty et al., 2014). In 2011, the U.S. Department of Education reported that low-income communities are receiving less, not more, of their share of financial support.

Conclusion

Here, we argue that the mechanisms influencing development as a result of early-life adversity, experienced more often in poverty, are different from the mechanisms shaping the SES-based achievement gap. Children in poverty are therefore experiencing a double hit—a high risk for stressors affecting mental health and a low probability for intergenerational mobility. We offered precise, evidence-based policy recommendations for both of these influences on developmental outcomes. As shown, this level of precision is both economically responsible and a wise investment in human capital.

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