Neurobiological Models of the Impact of Adversity on Education

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Abstract

Poverty and exposure to adversity have been linked with decreased educational success. Various environmental and neurobiological pathways have been proposed for these associations, however, existing models have several clear drawbacks. Here we outline existing models, and propose an alternate model linking exposure to adverse experiences in childhood to education success. Specifically, we propose that measured dimensions of experience (e.g., decreased cognitive enrichment or increased exposure to violence), instead of named exposures (e.g., poverty) impact neurobiology through neurodevelopmental processes of neuroplasticity. Our model results in testable hypotheses and clear intervention strategies. We predict that exposure to trauma will have a distinct neurobiological impact from exposure to a lack of cognitive stimulation and that these distinct exposures will benefit from different interventions. Specificity in this arena is thus likely to better support educational achievement for disadvantaged children.

Extensive evidence links childhood poverty to decreased educational success [1]. Children raised in poverty have lower school achievement, greater academic problems, and are less likely to graduate from high school than children who never experience poverty [2]. These associations have been observed consistently for decades and have generated considerable interest in developing strategies for reducing socio-economic disparities in educational outcomes. More recently, a wider range of adverse childhood experiences (ACEs)— including child abuse, community violence exposure, parental psychopathology and loss of a parent—has also been associated with educational outcomes (Bethell, Newacheck, Hawes, & Halfon, 2014; Jimenez, Wade, Lin, Morrow, & Reichman, 2016). Together, these findings indicate clearly that adverse social and environmental experiences early in life exert a lasting influence on educational success. In this paper, we explore neurobiological pathways that might explain these relationships.

We first review two leading conceptual models that posit specific pathways through which early experience influences educational outcomes. The literature linking poverty with

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educational success has developed largely independently from the literature linking ACEs with developmental outcomes. As a result, the hypothesized pathways through which early experience are conceptualized to influence child development are different in each of these models. Next, we present an alternative and integrated view of how diverse environmental experiences may come to shape educational success. This integrated approach brings together and extends the poverty and ACEs models, providing testable hypotheses about the neurobiological pathways impacted by different early experiences. In relaying this model, we hope to provide novel targets for interventions aimed at reducing educational disparities.

Poverty, Learning Experiences, and Education

The link between poverty and educational outcomes has often been attributed to differential exposure to cognitively-stimulating experiences and opportunities for learning as a function of socio-economic status (SES). Children born to wealthier parents with more education are likely to have better formal and informal educational opportunities beginning at an early age than children from families with less education and fewer resources. Children from high-SES families live in houses with more books where parents speak more often and in more complex ways to their children, and when they enter school they are more likely to experience an enriched educational environment than children in low-SES families. Children from high-SES homes are more likely to visit museums, engage in extracurricular activities, and spend greater time in the company of an invested adult than their lower-SES peers [2,3]. Variation in these types of early experiences is thought to influence neurocognitive development, including language, memory, attention, and both implicit and explicit learning processes [4,5]. Lack of learning opportunities is thought to directly drive atypical neurocognitive development; for example, low linguistic complexity in parental speech predicts poor child language development [6]. Disruptions in these domains of neurocognitive development might explain why children from low-SES families enter school at a disadvantage compared to their higher-SES peers [7]. Further, the impact of these early learning opportunities may further impact a child's ability to learn in school once they enter formal education [5].

Adversity, Stress, and Education

Leading conceptual models of childhood adversity argue that negative child development outcomes are the result of exposure to stress rather than a lack of exposure to cognitively-stimulating experiences [8]. The most prominent model of adversity, hereafter referred to as the ACEs model, focuses on the number of adverse childhood experiences: as the number of ACEs increases, risk for negative outcomes, including poor educational success, increases [9,10]. Within this model, poverty is either controlled for as a confounder or is just one of a number of adversities that can influence developmental outcomes. Importantly, childhood adversity is associated with detrimental outcomes across virtually every indicator of healthy development, ranging from mental and physical health to labor market success (Dube, et al., 2001; Edwards, Holden, Felitti, & Anda, 2003; Green et al., 2010; Johnson, Crosnoe, & Elder, 2011; Johnson & Schoeni, 2011; McLaughlin et al., 2010; Wickrama, Simons, & Baltimore, 2012). The broad nature of the impact of adversity on child development is thought to result, at least in part, from neurobiological mechanisms or

biological embedding of environmental experience (Hertzman & Boyce, 2009). The most commonly hypothesized pathway asserts exposure to environmental adversity chronically activates neurobiological stress response systems. Greater exposure to these adversities results in "wear and tear" on the neurobiological systems supporting the stress response. This wear and tear is termed allostatic load [11] The processes and consequences of allostatic load have been widely studied in animal models [12] and attempts have been made to replicate many of these findings in humans (with mixed success, see [13]).

Poverty model vs. ACEs model

These models of the impact of adversity on educational success have different strengths. First, the ACEs model encompasses a variety of adverse experiences that influence educational success, acknowledging that it is not just a lack of resources that hinders child development, but also exposure to violence, parental psychopathology, and parental absence. Second, the ACEs model acknowledges that these adversities are highly co-occurring, whereby the presence of one adversity increases the likelihood that a second will exist, and that this co-occurrence yields greater risk than the presence of a single adversity [10,14]. In addition, specifying a neurobiological mechanism affords a precise description of how adversity impacts child development that can be assessed across a wide variety of populations. For example, in our research we have used identical tasks and physiological measurements of stress response system function to assess the impact of diverse adversities, including poverty maltreatment, and institutionalization[13,15,16]. Similarly, we and others have used neuroimaging to assess neural structure across widely different adversities and settings[17,18]. These assessments overcome the numerous problems inherent in child or parent-report survey measures. Despite these attractive aspects of the ACEs model, there are several shortcomings. The proposed neurobiological pathways of the ACEs model are precise and suggest that prevention of ACEs would have substantial downstream impacts on health and education. However, while the ACEs model is useful for identifying children in need of intervention, it does not yield a specific intervention strategy to prevent downstream developmental consequences of stress dysregulation for children exposed to adversity beyond stress reduction programs, such as mindfulness-based meditation interventions. While there is some evidence that such therapies can be useful, and they are increasingly widely implemented, they are understudied in non-clinical populations of children and youth or with groups exposed to adversity [21]. Finally, the assumption of the ACEs model that all exposures contribute equally to developmental outcomes through a single neurobiological mechanism is questionable and ignores the importance of adversity type, timing, or chronicity on child development, including educational outcomes.

The poverty model addresses several of the gaps in the ACEs model. Primarily, the poverty model focuses on a specific pathway through which poverty impacts education success: insufficient learning experiences [7]. This pathway lends itself admirably to very specific intervention strategies in which children are provided enhanced access to learning opportunities through early educational programs, increased access to learning materials such as books, and greater parent-child interactions. Indeed, several randomized control trials have conclusively demonstrated that these interventions have positive long-term educational effects for children in poverty [22–25], providing experimental support for the

learning pathway linking poverty and educational success. However, the poverty model falls short in several ways. First, it ignores the wide variety of adverse experiences that influence child development and educational success. Living in poverty clearly increases the probability that a child will experience other adversities. In a population-representative sample of U.S. youths, 75% who experienced economic hardship were also exposed to at least one other ACE [26]. However, the kinds of co-occurring adversities varied, and ranged from parental divorce to sexual abuse. Relatedly, in a different population-representative sample, parental SES was associated with use of physical discipline, but the correlation was small (r = .10) after accounting for covariates [27]. Thus, it is clear that while SES is associated with greater exposure to other forms of adversity, it is not so completely overlapping with other ACEs that it can be assumed that the mechanisms underlying their impact on child development are identical. Second, the resource pathway, while specific, is limited in scope. For example, a lack of educational resources cannot account for the breadth of impact of poverty on child development. Poverty in childhood is associated with a range of negative outcomes including increased mental and physical health problems that are not an obvious consequence of lack of educational resources [28,29]. Finally, the lack of a neurobiological pathway through which lack of resources may impact child development makes this pathway less precisely defined than the ACEs pathway and not measureable across diverse settings and adversity types. Perhaps most importantly, the lack of a neurobiological pathway is inconsistent with the mounting evidence of an association between poverty and neural structure and function [17,20,30–32].

Deprivation and Threat

We propose an alternate approach to investigating the impact of adversity on educational success and child developmental outcomes more generally [33,34]. This alternative is based on two principles. First, across the range of adverse childhood experiences (e.g., maltreatment, community violence, lack of educational resources) it is possible to extract *dimensions* of adversity that encompass numerous types of exposures. Two initial dimensions of exposures are proposed: the absence of cognitive and social stimulation, termed deprivation, and the presence of experiences involving harm or threat of harm, termed threat. Conceptually, these dimensions cut across numerous exposures. For example, threat is a core feature of sexual abuse, physical abuse, and community violence and deprivation is a core feature of poverty, neglect, and institutionalization. Second, unique emotional, cognitive, and neurobiological pathways underlie the association of these dimensions of experience with developmental outcomes. Specifically, the absence of cognitive enrichment—deprivation—will influence the development of higher-order cognitive processes such as linguistic processing and executive function. We expect that exposure to threat in childhood provides a specific type of learning experience that will influence mechanisms involved in the acquisition and extinction of fear, with downstream consequences on emotion processing. These hypotheses are derived from animal models of early threat and deprivation exposure [35,3637,38]. See Figure 1 for an overview of our model.

Specifically, we hypothesize that the effects of deprivation and threat impact neurodevelopment through the typical developmental processes of neuroplasticity. In the case of deprivation, rodent and human studies suggest that a lack of environmental

stimulation leads to dramatic increases in synaptic pruning; when rodents are raised in low enrichment environments, global decreases in cortical volume are observed [38]. Similarly, when cognitive enrichment is low during early human development, for example among children raised in institutions with limited caregiver contact, cortical volume and thickness are reduced throughout the cortex [19,18]. In cases where cognitive deprivation is more mild, as in poverty, we would expect the effects to similar but attenuated; recent evidence is consistent with this prediction [17,39]. General reductions in cortical volume are not isolated phenomena but are likely to yield deficits in higher-order cognitive functions, such as language and executive functions, because they require coordinated function of multiple areas of association cortex and rely on late-developing areas of the brain such as the prefrontal cortex. This model thus expands upon the learning mechanisms outlined in the poverty model to encompass a neurobiological pathway underlying deprivation-related deficits in neurocognitive function that, in turn, will decrease educational success. Exposure to threat during periods of developmental plasticity, in contrast, shifts development of cortical and subcortical structures involved in coordinating fear responses and processing other negative emotions. Specifically, the presence of learning experiences involving high degrees of threat will bias these systems towards early detection of other environmental threats. While this initial prediction is a functional one, such functional neural changes must be instantiated structurally to some degree; thus in the case of threat we may expect specific structural changes only in cortical and subcortical structures which support emotional control. With regard to educational outcomes, children exposed to threat will be hindered in their ability to control their response to negative emotions in the classroom resulting in disruptive behavior and negatively impacting their learning opportunities.

The influence of threat and deprivation on developmental outcomes are mediated through basic neurodevelopmental processes involved in pruning and potentiation of synapses. Pruning is the mechanism underlying the impact of many childhood environmental experiences on neural development (e.g., phonemic retention in the context of multiple language exposure, visual cortex organization) [40–42]. Long-term potentiation is the basic neurobiological mechanism through which learning occurs throughout development [43,44]. The hypothesized pathways described here are in contrast to the stress pathway proposed in the ACEs model which relies on the existence of *developmentally atypical* processes of synaptic re-organization through abnormal dendritic changes for their hypothesized effect on a myriad of outcomes. The fact that synaptic pruning and potentiation, which we propose here, are the mechanisms responsible for typical acquisition of skills and abilities during development make them likely pathways through which neural development is shaped by adverse experiences.

This alternate conceptual model addresses many of the limitations in the ACEs and poverty models when considering the impact of adversity on educational outcomes. First, our model encompasses a variety of adverse experiences. Second, we propose neurobiological mechanisms that are specific to particular kinds of experiences at a testable level of specificity. Although adverse experiences are co-occurring, they can and should be measured separately to determine whether specificity exists in the cognitive, emotional, and neurobiological processes that they influence. Importantly, although threat and deprivation each impact educational success, they are likely to do so through distinct cognitive,

emotional, and neurobiological pathways. Thus, the neurobiological impact of deprivation can be assessed while statistically controlling for violence exposure, and vice versa. Third, this model has clear implications for interventions to improve the education experiences of children who have experienced adversity. Given our hypothesized differences linking deprivation and threat to educational outcomes, we likewise expect that children exposed to different types of adversity might benefit from distinct interventions. For example, a child with emotion regulation difficulties following exposure to traumatic violence and a child with executive function deficits following a lack of cognitive stimulation at home both may display disruptive behavior in the classroom. However, interventions for these two hypothetical children could be very different. In the former case, the child may benefit from a trauma focused therapy designed to increasing self-regulatory capacity via emotional awareness whereas in the latter case the child may benefit from increased scaffolded learning opportunities in the classroom.

Importantly, the deprivation and threat model builds on existing models of the impact of adversity on child development. The concept of deprivation is derived from the poverty literature identifying decreased cognitive stimulation as one pathway through which poverty influences education, and the concept of threat is related to stress exposure postulated in the ACEs model. Even the neurobiological pathways we propose to account for the impact of these dimensions on child development have been articulated by previous authors [45]. Our model brings together multiple dimensions of adversity and pathways through which these adversities could be embedded to yield novel testable hypotheses. This approach is an alternate to existing models of the impact of adversity on education that promises to generate novel and more targeted intervention strategies which have the potential to enhance education opportunities for the most disadvantaged children.

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Highlights

- Poverty and childhood adversity are associated with decrements in educational success

- Current neurobiological models for these effects are limited

- We provide an alternate model for the impact of adversity on academic performance.

- This model emphasizes *developmental* neurobiological pathways
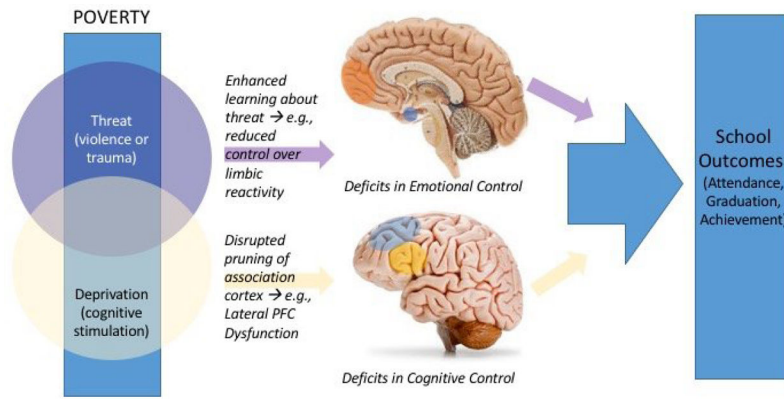
Figure 1.
Within adversity exposure, we hypothesize that dimensions of experience can be measured which will differentially impact neurodevelopment leading reductions in academic success. First panel: While exposure to cognitive enrichment (Deprivation dimension) is *likely* to be reduced as a function of poverty, it may not be, thus the deprivation circle is shown as not completely overlapping with poverty. Similarly, exposure to violence or trauma is likely to be, but is not always, increased for children living in poverty. These two circles are shown as overlapping because children are sometimes exposed to both deprivation and threat. Second panel: Next we show a cartoon of the hypothesized cognitive, emotional, and neurobiological pathways through which these kinds of exposures may impact school outcomes. Third panel: In the final part of the figure we show that school outcomes are an equifinal outcome for both dimensions of exposure. Thus, examining the neurobiology is central to distinguishing the impacts of various dimensions of exposure.