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Dimensions of early experience and neural development: deprivation and threat

Margaret A. Sheridan[1*] and Katie A. McLaughlin[2*]

[1] Division of Developmental Medicine, Boston Children's Hospital, Harvard Medical School, Boston, MA, USA
[2] Department of Psychology, University of Washington, Seattle, WA, USA

Over the past decade, a growing area of research has focused on adverse childhood experiences (ACEs) and their impacts on neural and developmental outcomes. Work in the field to-date has generally conceptualized ACEs in terms of exposure to stress while overlooking the underlying dimensions of environmental experience that may distinctly impact neural development. Here, we propose a novel framework that differentiates between deprivation (absence of expected cognitive and social input) and threat (presence of a threat to one's physical integrity). We draw support for the neural basis of this distinction from studies on fear learning and sensory deprivation in animals to highlight potential mechanisms through which experiences of threat and deprivation could affect neural structure and function in humans.

Dimensions of early adverse experience: deprivation and threat

The past decade has witnessed a proliferation of research on ACE and developmental outcomes. The term 'ACE' has been used to refer to a range of negative exposures during childhood that powerfully affect mental health, cognitive, and educational outcomes [1]. The strong relation between ACEs and developmental outcomes has generated considerable interest in identifying the neurodevelopmental mechanisms that explain these associations. A small but rapidly growing body of work has examined the impact of ACEs on neural structure and function [2,3]. However, prior work on this subject made little attempt to identify the underlying dimensions of environmental experience that might influence neural development. Here, we propose a novel conceptual framework for understanding the impact of ACEs on neural development that differentiates between experiences of deprivation and threat. Our intended contribution is to identify potential pathways through which deprivation and threat come to impact neural structure and function using basic neuroscience principles from animal research. We highlight pathways beyond the most commonly hypothesized mechanism of stress exposure to suggest additional ways in which ACEs influence brain development. Our aim is not to comprehensively review existing evidence on ACEs and neural development in humans or animals, but to provide a conceptual framework to guide future research.

The long-term negative effects of ACEs on developmental outcomes have been documented for decades. This research historically focused on single types of adversity, such as abuse and neglect. Recent studies have examined associations between the number of ACEs and developmental outcomes [4], based on evidence that different types of ACEs frequently co-occur [1]. An unintended consequence of this approach has been an oversimplification of the boundaries between distinct types of environmental experience. One example of this problem involves use of the term 'early-life stress' (ELS), which is used to refer to disparate experiences ranging from institutionalization to maternal depression and marital conflict [5,6], and obscures differences between these experiences that are likely to have important implications for understanding their impact on neural development. Characterizing underlying dimensions of environmental experience associated with diverse forms of adversity is critical for identifying their distinct effects on neural development, which is an essential first step in identifying mechanisms linking ACEs to developmental outcomes.

Here, we propose a novel conceptual framework for studying the effects of ACEs on neural development. The central distinction we make is between experiences of deprivation and threat (Box 1). We suggest that these

Box 1. Definitions of threat and deprivation

Threat
Experiences of threat involve the presence of an atypical (i.e., unexpected) experience characterized by actual or threatened death, injury, sexual violation, or other harm to one's physical integrity. Our definition of threat is consistent with the definition of a traumatic event in the *Diagnostic and Statistical Manual of Mental Disorders* [56].

Deprivation
Experiences of deprivation involve the absence of expected environmental inputs in cognitive (e.g., language) and social domains as well as the absence of species- and age-typical complexity in environmental stimulation.

Corresponding author: Sheridan, M.A. (Margaret.Sheridan@childrens.harvard.edu, sheridan.margaret@gmail.com).
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[*] These authors contributed equally to this work.

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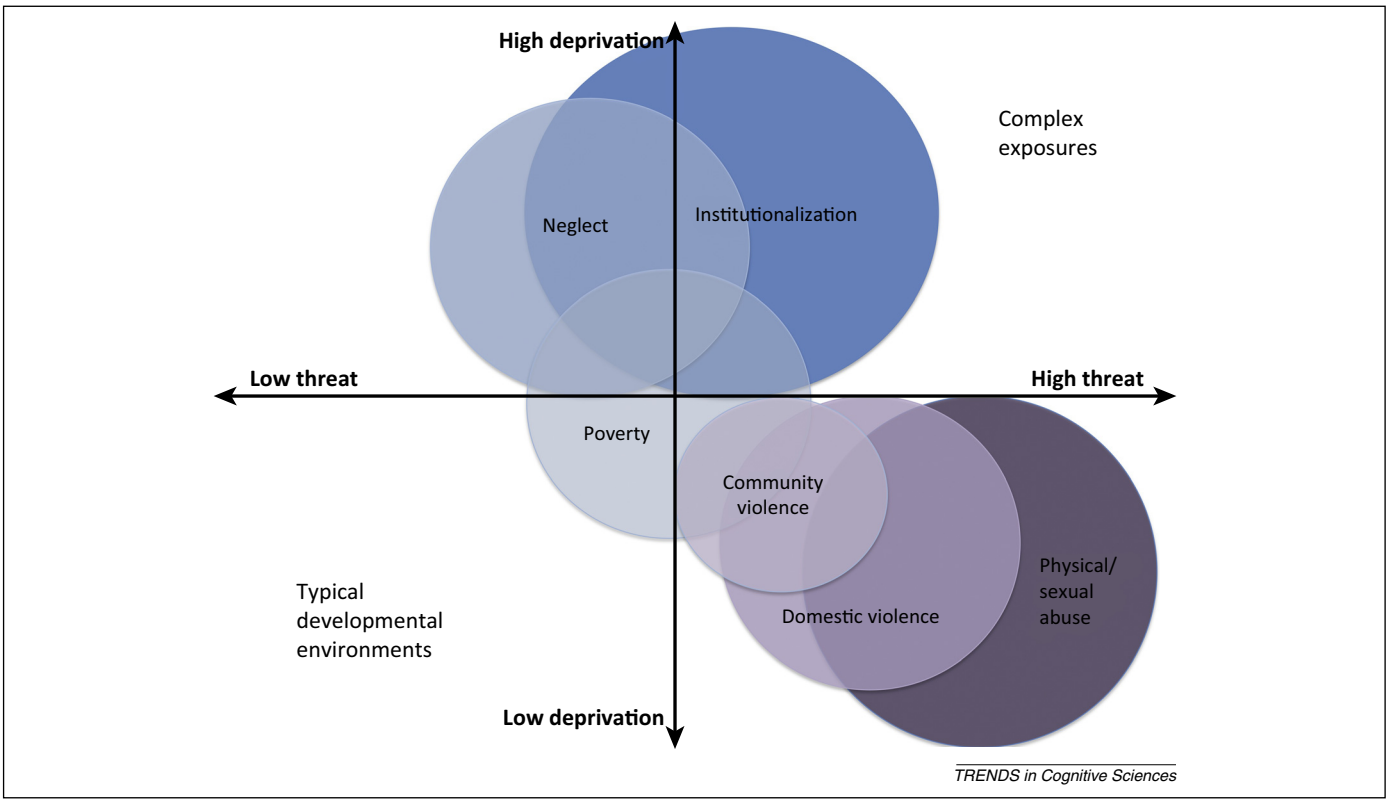

Figure 1. Dimensions of threat and deprivation associated with commonly occurring adverse childhood experiences (ACEs). This figure illustrates our argument that threat and deprivation are dimensions of experience that can be measured among children exposed to a range of adverse childhood experiences, both those that occur in isolation (e.g., a single incident of community violence exposure) and those that are co-occurring (e.g., physical abuse and physical neglect). We use the term 'complex exposures' to refer to experiences that, in most cases, involve aspects of both threat and deprivation.

dimensions differentially influence neurodevelopment. We do not propose that exposure to deprivation and threat occurs independently for children, because many ACEs co-occur. Instead, we propose that they can be measured separately (Figure 1) and have unique effects on neurodevelopment. Below, we separately describe deprivation and threat. Within each section, we first review mechanisms of neural development from animal neuroscience and describe how deprivation and threat influence these mechanisms. Next, we highlight emerging work in humans examining the neural consequences of ACEs. We end by proposing directions for future research that will help to determine the utility of our proposed framework.

The contribution of this perspective within the larger literature on ACEs and neurodevelopment is to highlight the importance of conceptualizing and measuring underlying dimensions of environmental experience reflected in frequently studied exposures, such as abuse, neglect, and poverty, because those dimensions may differentially influence neural outcomes. Critically, because fine-grained measurement of these dimensions has not been undertaken in human studies of neurodevelopment and because prior studies have focused on specific types of exposure (e.g., abuse) often without measuring or reporting co-occurring exposures (e.g., neglect), any conclusions regarding the consistency of existing human work with our proposed framework are necessarily tentative. Moreover, some exposures inherently involve high degrees of both deprivation and threat. For example, institutionalization involves the complete absence of an attachment figure in early development [7], an experience that not only involves deprivation in expected inputs, but can also represent a significant threat to survival for an infant. Importantly, we do not suggest that deprivation and threat are the only dimensions of experience that are important or that all ACEs can be conceptualized solely along these dimensions. Rather, we propose that these are two dimensions of experience that have not previously been differentiated with regard to their distinct influences on neural development.

Deprivation

The dimension of deprivation refers to the absence of species- or age-expectant environmental inputs, specifically a lack of expected cognitive and social inputs. We argue that the animal neuroscience literature examining the effects of sensory deprivation on sensory cortex development can be used as a model for understanding the neural consequences of deprivation in complex cognitive and social inputs in humans. Specifically, we suggest that an early environment without cognitive enrichment will yield a neural structure designed to deal with low complexity environments. Thus, exposure to cognitive and social deprivation in children would result in: (i) age-specific reductions in thickness and volume of association cortex, due in part to early overpruning of synaptic connections, lower numbers of synaptic connections, and reduced dendritic branching; and (ii) reduced performance on tasks that depend on these areas (e.g., complex cognitive tasks). Reductions in cortical thickness should be most pronounced in regions of association cortex that are recruited