

Neglect as a Violation of Species-Expectant Experience: Neurodevelopmental Consequences

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ABSTRACT

The human brain requires a wide variety of experiences and environmental inputs in order to develop normally. Children who are neglected by caregivers or raised in institutional environments are deprived of numerous types of species-expectant environmental experiences. In this review, we articulate a model of how the absence of cognitive stimulation and sensory, motor, linguistic, and social experiences common among children raised in deprived early environments constrains early forms of learning, producing long-term deficits in complex cognitive function and associative learning. Building on evidence from animal models, we propose that deprivation accelerates the neurodevelopmental process of synaptic pruning and limits myelination, resulting in age-specific reductions in cortical thickness and white matter integrity among children raised in deprived early environments. We review evidence linking early experiences of psychosocial deprivation to reductions in cognitive ability, associative and implicit learning, language skills, and executive functions as well as atypical patterns of cortical and white matter development—domains that should be profoundly influenced by deprivation through the learning and neural mechanisms we propose. These patterns of atypical development are difficult to explain with existing models that emphasize stress pathways and accelerated limbic system development. A learning account of how deprived early environments influence cognitive and neural development provides a complementary perspective to stress models and highlights novel pathways through which deprivation might confer risk for internalizing and externalizing psychopathology. We end by reviewing evidence for plasticity in cognitive and neural development among children raised in deprived environments following interventions that improve caregiving quality.

Keywords: Brain development, Childhood adversity, Deprivation, Early life stress, Learning, Neglect

<http://dx.doi.org/10.1016/j.biopsych.2017.02.1096>

The human brain requires a wide variety of experiences and environmental inputs, some during sensitive periods, in order to develop normally. The simplest demonstration of this principle can be observed in sensory systems; access to patterned light and complex sounds during the first months of life is required for normal visual and auditory function to develop. Similar sensitive periods exist for the development of more complex behaviors and competencies, including language and the formation of an attachment to a caregiver. The wide-ranging domains of functioning that require environmental input for normal development are referred to as experience expectant (1). In this review article, we examine what happens when these expected environmental inputs are absent. We present a conceptual model of how an absence of expected inputs from the environment influences learning and neurodevelopmental processes in children, and we review existing literature on youths raised in deprived early environments in light of this model. We highlight how atypical cognitive and neural development might serve as a mechanism linking environmental deprivation to psychopathology, and we end by reviewing evidence for plasticity in cognitive and neural outcomes among children raised in deprived environments following interventions that improve caregiving quality.

SCOPE OF THE PROBLEM

Neglect involves failure of a caregiver to act in ways that are necessary to meet the basic needs of a child (2–4). Neglect encompasses inadequate provision for physical needs, poor protection from harm, and failure to provide for emotional or educational needs (see Table 1) (2–4). Neglect is the most common form of maltreatment reported to child protective services in the United States (2,5). Worldwide, millions of children have lost their parents due to armed conflict, forced migration, or infectious diseases; a common response is to raise these children in institutions. Although most institutions provide for physical needs, institutional care is often characterized by limited interaction with caregivers, resulting in a failure to provide for children's emotional and developmental needs. Despite the high prevalence of neglectful early environments, the developmental consequences of neglect are understudied as compared with other forms of adversity (6).

NEGLECT AS ENVIRONMENTAL DEPRIVATION

Environmental deprivation is a central feature of child neglect and institutional rearing. This deprivation spans numerous inputs the human brain expects, often at particular points in

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Table 1. Key Domains and Examples of Child Neglect

Neglect Involves Failure of a Caregiver to Provide for:	Examples
Physical Needs	Nutrition, clothing, shelter, access to medical care
Protection From Harm	Inadequate supervision
Emotional Needs	Presence of a stable caregiver, sensitive and responsive caregiving, emotional nurturance
Educational Needs	School attendance

development. Deprivation is the core feature of neglect that distinguishes it from other forms of adversity, such as trauma and abuse, where the most prominent feature is harm or threat of harm to the child. Although experiences of deprivation often co-occur with experiences of threat (i.e., abuse), the developmental consequences of deprivation and threat are at least partially distinct (7–9). Here, we focus specifically on neurodevelopmental consequences of deprivation resulting from neglect and institutional rearing.

At the most fundamental level, neglected children are deprived of a stable, sensitive, and responsive caregiver, which is a species-expectant experience. Caregivers are necessary to ensure survival in early human development by providing nutrition and ensuring safety from threats (10). Infants are born with a behavioral repertoire designed to ensure caregiver protection and proximity (e.g., crying) (11). Children develop a secure attachment when caregiving is sensitive, responsive, and predictable (12–15). Caregivers impose regularity onto children's environment by regulating sleep–wake cycles and feeding and by responding contingently to distress with physical proximity and nurturance. Neglected children are not afforded sensitive, supportive, and stable caregiving on a consistent basis. Parents with documented histories of neglect generally show low levels of emotional warmth, positive behaviors, and empathy (16–18). Neglectful families also exhibit caregiving that is irregular and unstable (17). A similar absence of emotionally supportive caregiving occurs in institutional environments, where caregiver interactions with children are infrequent and contingent responding is low (19).

Early in life, most forms of learning occur in the context of caregiver interactions. The sensory, motoric, linguistic, and social experiences provided by caregivers determine the complexity of children's environment and the degree of cognitive stimulation children receive. Caregivers regulate exposure to environmental inputs of numerous kinds, including language and auditory stimulation in the form of caregiver vocalizations, social interaction through play, and sensory and motor stimulation through physical contact and the provision of objects for children to manipulate. In some domains (e.g., language), exposure to environmental input must occur in the context of social interaction to generate learning (20,21). The absence or unavailability of a primary caregiver results in gross reductions in sensory, cognitive, and social stimulation. Indeed, reductions in cognitive stimulation, provision of learning opportunities, supervision by adults, and parent–child interactions have been observed among children who are neglected (16,18,22). Similarly, children raised in institutions experience dramatic reductions in exposure to language, less frequent and predictable interactions with adults, limited variation in daily routines and experiences, and less access to novel and age-appropriate enriching cognitive stimuli than do children raised in families (19,23,24).

Importantly, the severity of deprivation experienced by neglected children exists along a continuum. Most studies do not measure specific types of deprivation directly (e.g., degree and complexity of linguistic experiences) but rather assess the presence of neglect or institutional rearing. Determining how the neurodevelopmental mechanisms outlined below vary as a function of the severity of deprivation is a critical goal for future research.

EXISTING PERSPECTIVES

A variety of brain regions and circuits are influenced by early deprivation. The absence of a caregiver to provide protection from harm and to regulate arousal and distress represents a pervasive stressor that can produce lasting changes in emotional development. Most existing models emphasize atypical limbic system development resulting from prolonged early-life stress as a central mechanism underlying developmental outcomes associated with caregiver deprivation (25–31). Strong evidence supports this view. Children deprived of a stable and responsive caregiver exhibit high levels of insecure and disorganized attachment and atypical affective development characterized by heightened emotional reactivity, accelerated functional development of the amygdala, poor emotion regulation, and atypical stress reactivity (25,32–37). These disruptions in attachment and affective development contribute to high levels of internalizing psychopathology among children raised in deprived early environments (31,38–40).

But is this the only mechanism involved in neglect? One of the most consistent observations of neglected children is that they exhibit deficits in numerous areas of cognitive development (27,28), and these deficits are more extreme than those observed in other forms of adversity (e.g., abuse) (41). Are disruptions in limbic system development sufficient to explain these widespread cognitive effects? We argue that additional mechanisms are involved.

Deprivation as an Absence of Learning

Environmental deprivation that characterizes child neglect and institutional rearing has a pervasive and lasting influence on development. Disruptions in early learning may underlie the far-reaching developmental consequences of neglect, including those not readily explained by atypical limbic development (e.g., low cognitive ability). Children who experience neglect are raised in an environment characterized by the absence or limited availability of a caregiver, which curtails the complexity of their sensory, motor, and linguistic experiences and reduces learning opportunities.

Early deprivation constrains basic forms of learning that depend on rich sensory and social inputs early in development, including associative and implicit learning. Caregivers play a critical role in the development of these learning

processes by directing children's attention to relevant stimuli in the environment through repetitive vocalizations, facial displays, and tactile stimulation (42). Child-directed language has unique acoustic properties (43–45) that shape early learning by increasing infant attention to external stimuli and enhancing associative learning (46–48). In the absence of caregiver speech directed to the child—a feature of neglectful environments (17,19)—associative learning and implicit learning are likely to be constrained. Indeed, child-directed speech produced by caregivers with depression fails to promote infant associative learning (49) due to reduced perceptual salience of caregiver vocalizations that lack the typical acoustic properties of child-directed speech (50).

Associative and implicit learning processes are the foundation on which more complex forms of cognition and learning are built. Atypical early development of these types of learning may propagate throughout myriad domains of development, producing deficits in general cognitive abilities, language, and executive functioning. Language development relies on implicit learning of regularities in speech and their pairing with visual cues in the environment (51–56). Associative learning also provides the scaffolding for executive functioning, including conflict adaptation, response inhibition, cognitive flexibility, and attentional control (57). Early disruptions in associative learning may produce difficulties in multiple domains of executive functioning by reducing associations between goal representations and relevant environmental stimuli in a particular context.

Neglect involves reduced inputs in sensory, linguistic, cognitive, and social domains. Here we propose a common learning mechanism—constrained associative learning—that might explain the consequences of deprivation in each of these domains on cognitive development, although it is important to highlight that other forms of learning are also reduced for children who have inconsistent interactions with caregivers (e.g., imitation) and that domain-specific mechanisms (e.g., visual input producing columnar organization of visual cortex) also influence developmental outcomes. Altogether, a learning account of deprivation predicts that neglect will be associated with atypical cognitive development in many domains, including poor associative and implicit learning, global declines in cognitive functioning, and deficits in language and executive functioning.

Neurodevelopmental Mechanism of Environmental Deprivation

Early deprivation exerts profound influences on neurodevelopmental processes that are shaped by learning and experience, particularly experience-expectant processes (1). Experience-expectant refers to processes whereby the human brain expects certain inputs to acquire a skill or competency, typically during sensitive periods in development. If such inputs are present, development proceeds in a typical fashion. When a child passes through the sensitive period without such inputs, development will be compromised in those domains (58).

An overproduction of synaptic connections early in development that are pruned as a function of experience provides the biological basis for experience-expectant learning (1). Pruning is an activity-dependent developmental process that selectively eliminates synaptic connections that are used infrequently (59–61). When two cells coactivate frequently,

the synaptic connection between them strengthens and becomes more efficient, resulting in long-term potentiation and increases in the density and number of dendritic spines on postsynaptic neurons. Long-term potentiation underlies numerous forms of learning, including experience-expectant learning (62,63). In contrast, when two cells coactivate infrequently, dendritic spines shrink or disappear and the synaptic connection weakens and is likely to be eliminated (64,65). Synaptic pruning is a central force in the remodeling of the brain across development in response to experience (60).

We propose that environmental deprivation hijacks the developmental process of synaptic pruning, resulting in accelerated and extreme synapse elimination (see Figure 1). Animals deprived of visual input early in development exhibit dramatic reductions in synapses, dendritic branching, and the number and density of dendritic spines in visual cortex (66–70). These changes produce measurable reductions in the thickness of visual cortex in animals deprived of visual input (70).

What about environmental deprivation that is more global? The environment of neglected and institutionally reared children is characterized by an absence of inputs and complexity across multiple domains. Animal models of global deprivation compare animals raised in isolation in an empty cage with those reared in a complex environment with access to conspecifics, toys, and novel stimuli; this type of deprivation leads to dramatic changes in synaptic organization similar to those observed in sensory deprivation but that are more widespread across the cortex. Animals raised in a deprived environment exhibit reductions in the number of synapses per neuron (71), the density of cortical dendritic spines (72), the branching and length of dendrites (73,74), and cortical thickness (75,76). These neural changes are accompanied by deficits in numerous forms of learning and memory (77–81).

Although synaptic changes are a primary mechanism of experience-dependent plasticity, poor white matter integrity resulting from reduced myelination and axon sprouting also occurs in animals exposed to early environmental deprivation, particularly in the posterior corpus callosum (82) and prefrontal cortex (PFC) (83). Other mechanisms are also involved, including changes in epigenetic regulation (84), although we do not review those mechanisms here.

Animal models of global deprivation resemble neglect in that they reflect an environment characterized by a lack of complexity and an absence of sensory, cognitive, and social stimulation. Findings from animal models provide clues about how deprivation will influence learning and neural development in children. Specifically, we expect deprivation to be associated with exaggerated synapse elimination, reduced dendritic branching and density throughout the cortex, and reduced myelination in the corpus callosum and PFC; although these molecular processes cannot be studied directly in humans, they should produce reductions in cortical thickness (see Figure 2) and fractional anisotropy measurable by magnetic resonance imaging.

NEURODEVELOPMENTAL CONSEQUENCES OF ENVIRONMENTAL DEPRIVATION

Does existing evidence support a learning model of environmental deprivation? In this section, we review evidence on the association of environmental deprivation with domains of

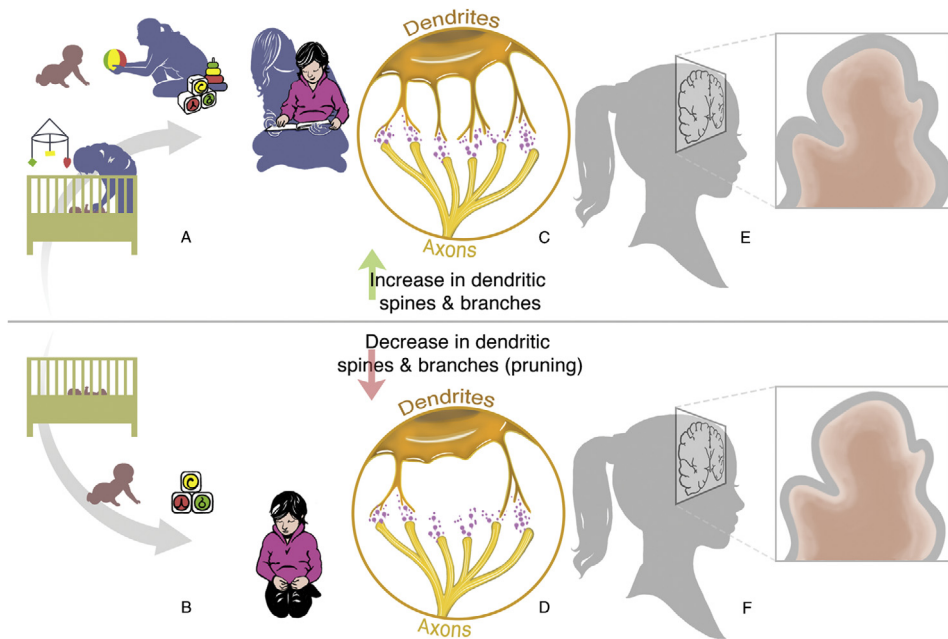


Figure 1. A neurodevelopmental mechanism of early deprivation. **(A)** A typical early environment is rich in sensory, linguistic, and social experiences that occur in the context of interactions with a caregiver and that provide a rich source of cognitive stimulation. Caregivers foster cognitive stimulation and shape early learning by directing children’s attention to important cues in the environment through vocalizations, facial displays, and tactile stimulation. **(B)** Child neglect and institutional rearing constitute a deprived environment with dramatic reductions in the quantity and quality of caregiver interactions. This type of deprived environment constrains cognitive and social stimulation as well as learning opportunities. **(C)** Coactivation of presynaptic and postsynaptic neurons promotes postsynaptic dendritic branching, dendritic spine formation, and stabilization and strengthening of existing synaptic connections. **(D)** Limited coactivation of presynaptic and postsynaptic neurons ultimately leads to pruning of the synaptic

connection. **(E)** A typical early environment should produce a pattern of greater cortical thickness throughout both primary sensory and association cortices as compared with children in a deprived environment. **(F)** Accelerated synaptic pruning that occurs as a result of deprived sensory, linguistic, cognitive, and social stimulation will lead to age-specific reductions in cortical thickness for children raised in deprived environments that are widespread across the cortex.

development that should be strongly influenced by the learning and neural mechanisms outlined in our model and that are difficult to explain based solely on stress and limbic system pathways. Specifically, we examine the associations of deprivation with global cognitive ability, associative and implicit learning, language, executive functioning, cortical structure, and white matter integrity. Our model predicts that children exposed to deprivation will exhibit poor performance in these cognitive

domains, widespread reductions in cortical thickness, and reduced white matter integrity, particularly in the corpus callosum and PFC. We constrain this review to studies of children with documented histories of a) neglect or b) institutional rearing beginning early in life (i.e., where children were institutionalized from birth or shortly thereafter). We exclude studies that assess neglect or developmental outcomes in adults, that assess neglect based on retrospective appraisals (85,86), or that focus on children institutionalized later in development.

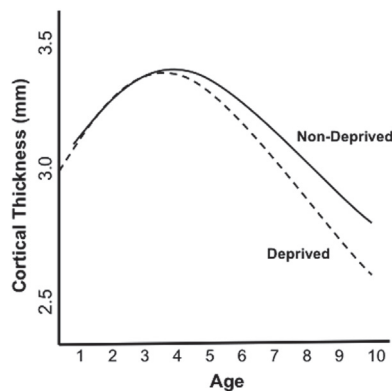


Figure 2. Predicted patterns of cortical thickness across development for children raised in typical and deprived early environments. Exaggerated synaptic pruning occurring throughout the cortex among children from deprived environments will produce a pattern of accelerated cortical thinning as compared with children from nondeprived environments. This pattern will produce age-specific reductions in cortical thickness in both primary sensory cortex and association cortex.

Cognitive Ability

If environmental deprivation produces learning deficits, global cognitive ability should be affected. Indeed, children exposed to neglect and institutional rearing experience dramatic reductions in cognitive ability. Neglected children have lower IQ and academic performance than children raised in typical caregiving environments (41,87–91). A similar pattern has been observed among children reared in institutional settings. Tizard and Rees’s seminal study of children raised in institutions characterized by relatively mild deprivation nonetheless demonstrated that these children had lower IQ than children raised in families (92). Critically, the degree of environmental stimulation children received—including literary experiences, interactions with adults, and experiences outside the institution—was positively associated with cognitive ability (92). Dramatic reductions in IQ among children reared in deprived institutional settings have been widely replicated and are associated with the duration of institutional care (93–95). These findings are consistent with a learning account of

deprivation in which the degree and duration of cognitive and social stimulation in the early environment are strongly associated with global cognitive abilities.

Associative Learning

Associative learning creates connections between co-occurring stimuli or between a stimulus and a response, mediated by coordinated cell assemblies whose synaptic connections are strengthened on coactivation (96,97). Although we expect environmental deprivation to be associated with broad impairments in associative learning, existing research has focused primarily on stimulus–response learning. Children raised by responsive caregivers learn that exhibiting signs of distress will produce caregiver proximity, soothing behaviors, food, or removal of a source of distress. Through contingent responding, children learn that certain behaviors elicit reward (e.g., food, soothing). An absence of contingent responding teaches children that their behaviors are unlikely to produce reinforcement, creating weak stimulus–reward associations that may shape the neural circuits underlying reward learning, producing lasting alterations in reward-directed behavior.

Growing evidence supports these predictions. Poor stimulus–response learning has been observed in children raised in institutions and is associated with the duration of institutional care (98). Children reared in institutions do not alter behavioral responses to stimuli as a function of reward value, whereas typically developing children are faster and more accurate in responding to rewarded versus nonrewarded stimuli (99; see also M.A. Sheridan, Ph.D., *et al.*, unpublished data, 2017). Institutionally reared children also exhibit reduced activation in the ventral striatum—a region centrally involved in reward processing—during reward anticipation and in response to positive cues (100,101). Existing evidence supports the prediction that early deprivation is associated with atypical stimulus–response associative learning.

Implicit Learning

Implicit learning is an unconscious learning process that creates abstract knowledge through detection of structure in a complex sensory environment (102–105). Implicit learning depends on a rich sensory environment and observable regularities in the environment. Infants rapidly learn about statistical regularities in the environment across multiple sensory domains (51,106,107) that form lasting representations of the environment that facilitate skill development, problem solving, and predictions about the future (102,105). Implicit learning is likely to be constrained in environments lacking sensory and linguistic complexity.

Two studies have examined implicit learning in neglected children. The first examined children adopted into the United States internationally who performed no worse than nonadopted children on an implicit learning task (108). These children displayed cognitive abilities that were no different from those of comparison children, suggesting a less deprived institutional experience. In contrast, a recent study documents poor implicit learning in children raised in deprived institutions. Children exposed to institutional deprivation were less likely to learn a pattern of numbers embedded in a serial reaction time task, as indicated by slower reaction time and lower accuracy than

comparison children on patterned trials relative to nonpatterned trials (M.A. Sheridan, Ph.D., *et al.*, unpublished data, 2017). More research on implicit learning following early deprivation is clearly needed.

Language Development

Extensive evidence supports the prediction that language development is influenced by environmental deprivation. Poor language skills are likely influenced directly by inconsistent exposure to caregiver language—common in neglected children—and indirectly through poor associative and implicit learning. In non-deprived family environments, the degree of environmental stimulation in the home as well as the amount and quality of maternal language predicts children's language skills (109,110). Poor expressive language and receptive language have been consistently observed in neglected children, who exhibit language difficulties that are more pronounced than those associated with abuse (87,111,112). Children raised in institutional settings also exhibit meaningful reductions in language ability that are associated with the duration of institutional care (108,113–115).

Executive Functioning

Executive functions (EFs) comprise a set of cognitive processes that support the ability to learn new knowledge and skills, hold in mind goals and information, and create and execute complex, future-oriented plans. EFs encompass working memory, inhibition, and switching/cognitive flexibility (116,117). These skills, and the frontoparietal networks that support them, exhibit a protracted developmental trajectory that extends throughout adolescence (118,119), suggesting ongoing plasticity in EFs across development. Yet, one of the most consistently observed patterns in children exposed to early deprivation is lasting and intractable EF deficits. Children raised in institutions exhibit poor performance on tests of working memory, inhibition, planning, sustained attention, and cognitive flexibility (98,108,120–126) and exhibit a less efficient pattern of dorsolateral PFC recruitment in tasks requiring executive control (126). EF performance is worse in children with more severe and longer-lasting deprivation (98,108,123,124). EF deficits often persist after removal from a deprived environment, which is surprising given the extended development of brain regions that support these functions. Associative learning is thought to play a central role in the development of EFs by facilitating the association of goal representations with relevant environmental stimuli in a particular context (57). Early problems in associative learning may explain, in part, these lasting difficulties with EFs, although future research is needed to evaluate this possibility.

Neural Structure and Function

Consistent with the first proposed neurodevelopmental mechanism of deprivation—accelerated synaptic pruning, leading to age-specific reductions in cortical thickness—children reared in deprived institutions exhibit smaller total brain volume (127), pronounced reductions in cortical gray matter (128), and widespread cortical thinning throughout both primary sensory and association cortices in the parietal, temporal, and frontal lobes (129). Reduced cerebellum volume was also reported in one study of children raised in institutions (130). Smaller left

amygdala volume and larger right amygdala volume were reported in a small study of previously institutionalized children (127), but these patterns have not been replicated in larger studies (33,128).

Institutionally reared children exhibit patterns of neural function that reflect tonic cortical hypoactivation consistent with these structural findings, including reduced power in high-frequency electroencephalogram bands (alpha) and increased power in low-frequency bands (theta), increased short-distance electroencephalogram coherence, and reduced gamma cross-frequency coupling, each of which is associated with duration of institutional care (131–134).

White matter changes have also been observed, consistent with evidence from animal models, including reduced volume of the posterior corpus callosum (128,135) and reduced fractional anisotropy in white matter tracts linking the PFC with the temporal lobe (e.g., uncinate fasciculus, superior longitudinal fasciculus) and the striatum (e.g., internal and external capsules) (135–137).

NEURODEVELOPMENTAL MECHANISMS LINKING DEPRIVATION TO PSYCHOPATHOLOGY

Children raised in deprived environments exhibit elevations in many forms of psychopathology, including anxiety, depression, attention-deficit/hyperactivity disorder (ADHD), aggression, and substance abuse (38,40,138). What role do disruptions in cognitive and neural development play in the etiology of deprivation-related psychopathology? One domain for which these mechanisms appear to be particularly important is externalizing psychopathology, including ADHD—a disorder characterized by impulsivity, inattention, and poor EF. ADHD is strongly associated with institutional rearing and persists following removal from institutional care after the age of 6 months (38,40,139). EF deficits (particularly in working memory and inhibition), reductions in cortical thickness, and patterns of neural function reflecting tonic cortical hypoactivation are mechanisms that explain the association of early deprivation with ADHD (121,129,140). Changes in neural structure and cognitive function related to early deprivation may be a core mechanism underlying the development of externalizing psychopathology in children exposed to early deprivation.

In contrast, internalizing psychopathology following early deprivation improves following removal from a deprived environment (38,40) and involves mechanisms consistent with stress models focusing on atypical affective development. Improvements in anxiety and depression following early deprivation are explained, in part, by the development of secure attachment to new caregivers among adopted children (39).

Future research is needed to evaluate whether atypical associative learning is a mechanism in the link between early deprivation and psychopathology. Both depression and externalizing problems have been associated with poor reward learning and atypical neural response to reward (141,142), patterns also observed among children exposed to early deprivation. It seems plausible that disruptions in reward learning are an additional pathway explaining the link between deprivation and multiple forms of psychopathology.

PLASTICITY

Are the neural and behavioral consequences of deprivation reversible? Understanding ongoing plasticity is essential for preventing the onset of psychopathology in children raised in deprived environments. Although numerous methods exist in animal models for examining plasticity and critical periods, there are obvious constraints on what one can do with human children. One approach that has yielded important insights into neural plasticity is intervention, where the aim is to manipulate a system at different points in development and examine the impact on developmental processes. One example is the Bucharest Early Intervention Project, the first randomized controlled trial of foster care as an intervention for early institutionalization (23,143). After screening for developmental and neurological issues, 136 infants aged 6 to 30 months were randomly assigned to a high-quality foster care intervention or to care as usual—continued institutional care (23). These children have been followed through age 12 years, and a 16-year assessment is ongoing.

Relevant to our proposed model of deprivation, of the developmental domains reviewed here, intervention effects have been found for IQ, language development, reward learning, neural function assessed with electroencephalogram, and white matter volume and integrity (93,113,128,132,134,135; see also M.A. Sheridan, Ph.D., *et al.*, unpublished data, 2017); similar effects on cognitive development have also been observed in intervention studies designed to improve caregiving quality in institutions (144). Additional domains that improved in children randomized out of institutional care include physical growth, attachment, stress reactivity, and internalizing symptoms (34,38,40,145). Earlier removal from the institution led to more dramatic improvements in IQ, language, neural function, stress reactivity, and attachment. No intervention effects were found for EFs, ADHD, and cortical thinning, which are affected by institutionalization but not by foster care intervention.

CONCLUSIONS

Child neglect and institutional rearing deprive children of numerous environmental experiences the human brain expects to develop normally. This deprivation produces lasting alterations in many domains of cognitive development, including general cognitive ability, associative and implicit learning, language, and EFs as well as reductions in cortical gray matter volume and thickness and white matter integrity. We propose that deprived environments constrain early forms of experience-expectant learning, accelerate the neurodevelopmental process of synaptic pruning, and limit myelination, ultimately producing these atypical patterns of cognitive and brain development. These developmental disruptions, in turn, may confer risk for psychopathology, which is common among neglected and institutionally reared children. Interventions aimed at improving developmental outcomes in children raised in deprived environments would benefit from increased attention to the importance of cognitive and social stimulation.

ACKNOWLEDGMENTS AND DISCLOSURES

This research was supported by the National Institute of Mental Health (Grant Nos. R01-MH103291 [to KAM], R01-MH106482 [to KAM], and

R01-MH091363 [to CAN]), a Jacobs Foundation Early Career Research Fellowship to KAM, and a Jacobs Foundation research grant to CAN.

All authors report no biomedical financial interests or potential conflicts of interest.

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Received Oct 13, 2016; revised Feb 03, 2017; accepted Feb 21, 2017.

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