

# The Developmental Neuroscience of Emotional Neglect, Its Consequences, and the Psychosocial Interventions That Can Reverse Them

**F**or at least the past 40 years, we have learned how devastating child maltreatment can be to an individual's development (1). Early child maltreatment, when the child survives its immediate effects, confers risk for significant physical and psychiatric morbidity and mortality throughout the remaining lifespan—from physical ailments, including cardiovascular disease, autoimmune conditions, and cancer, to substance abuse (2) and suicide (3).

Most of the research on this topic has focused on physical and sexual abuse, albeit with which emotional neglect is often comorbid. Yet as René Spitz (4) noted in reference to orphaned infants in the postwar period, emotional neglect in and of itself is also damaging and potentially fatal. Only recently have developmental neuroscientists focused specifically on emotional neglect.

In contemporary institutions such as the Romanian orphanages described by Smyke et al. in this issue of the *Journal* (5), infants generally receive adequate nutrition, physical care, and protection from violence, but little consistent, dependable social interaction with an adult caregiver, and consequently they have no apparent attachment to another person. The longer institutionally deprived infants go without intervention, the more difficult it can become for their development to advance in the many areas—such as cognitive functioning, language acquisition, and social behavior—for which an attachment relationship seems to be a prerequisite (6).

Recent advances have permitted researchers to increase our understanding of genomic and nongenomic (i.e., epigenetic) factors that interact with the caregiving environment to contribute to individual outcomes and to favor intergenerational transmission of psychopathology or resilience. Yet generally these gene and environment studies, with few exceptions, have not explored how emotional neglect affects the developing child and his or her brain via any particular gene-environment interaction during sensitive periods of development. And researchers have only recently begun to explore what we can do to limit or even reverse at least some of the deleterious effects of emotional neglect early in development so as to foster greater plasticity and richness in the social-emotional and intellectual life of the affected child.

The study reported by Bogdan et al. in this issue (7) is thus a pioneering effort to integrate the effect of an emotionally neglectful caregiving environment with the effect of a gene on neuronal activation in the limbic system and other subcortical areas that interdependently contribute to the stress response via the hypothalamic-pituitary-adrenal (HPA) axis. The HPA axis makes glucose available to fuel the stress response with a feedback loop to modulate the process. The HPA axis's mineralocorticoid and glucocorticoid hormones and receptors play an important role in the onset and offset of the stress response.

Bogdan et al. describe how the val allele of the high-affinity mineralocorticoid receptor, which regulates the onset of the HPA axis stress response and has been associated with depressive and anxiety symptoms and specific learning difficulties, interacts with a deprived caregiving environment to produce heightened neural activation within the amygdala. The resulting “endophenotype” described provides new opportunities for focusing interventions that might target the underlying mechanisms that produce this heightened amygdala reactivity—as well as therapeutic strategies that might prevent subsequent related psychopathology. Notably, the study was conducted within the specific developmental period of late childhood/early adolescence, when the corticolimbic system’s regulation of the endocrine portion of the HPA axis is coming online.

Some individuals may be at risk for heightened depressive and anxiety symptoms with even mild to moderate emotional neglect by virtue of their carrying the val allele, whereas others who have a different gene variant will be relatively resilient. Might it be possible that those with the val allele in the presence of an enriched rather than a deprived caregiving environment could exhibit a superior adaptation to their environment without its presence? While this is not known for the val allele, it has been suggested in the case of at least one other genetic polymorphism, which thus challenges our categorical thinking of risk versus resilience with a more complex, dimensional model of gene variants that promote different degrees of plasticity (versus risk) that is suited for unpredictable, changing environments (8). Another caveat in reading the Bogdan et al. article and other reports of a single gene-environment interaction is that despite the very sophisticated science applied in such studies, the reported findings account for only a very limited percentage of variance (7% in the case of the Bogdan et al. study) that is related to one particular receptor and one particular area of the brain in the context of one particular form of adverse experience during one particular period of development. We are only at the beginning of our voyage—gene-gene-environment and gene-environment-environment interactions lie ahead.

Although the science is just starting to set sail, interventions to address the effects of maltreatment on the individual and to interrupt the intergenerational transmission of maltreatment and related psychopathology are possible.

The article by Smyke et al. (5) shows how children who suffer more pervasive emotional neglect due to institutional care can show clinically significant reductions in the severity of at least one form of attachment disorder after early psychosocial intervention in the form of enhanced child-centered foster care. This landmark study, which is the first randomized controlled trial of foster care versus institutional care as usual, also shows that while one can reverse the symptoms of one form of attachment disorder—the withdrawn type—one is harder pressed to diminish the symptoms of the disinhibited type. Given that the latter form of psychopathology itself carries a risk for subsequent psychiatric morbidity, the authors raise the important question of whether clinicians’ efforts are best directed toward trying to eradicate disinhibited social behavior. The authors suggest that perhaps a more realistic approach would be to train the affected individuals with these limitations to “read and respond” better to social cues as an adaptation to their social

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environment. Although the Romanian situation is striking on first reading because it recapitulates the extreme deprivation first identified by Spitz (4) in institutionalized children, its more enduring value is that it reaffirms Spitz's second observation, namely, that individualized, consistent human contact has a profound therapeutic role for the infant.

Both these studies are relevant to many clinical practices. Emotional neglect may arise more commonly from parental psychiatric conditions, such as major depression and posttraumatic stress disorder, both of which are associated with attachment disturbances and corresponding patterns of corticolimbic dysregulation on functional neuroimaging (9, 10). At least one study (11) has documented enlarged amygdala volume in children of depressed as opposed to nondepressed mothers, suggesting the need for further research on intergenerational transmission of developmental and relational psychopathology in the wake of early adverse experience. As developmental neuroscience becomes ever more sophisticated, it validates with even greater certainty the importance of a good foster family to a neglected child, as early as possible in the child's life.

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