The Developmental Consequences of Child Emotional Abuse: A Neurodevelopmental Perspective

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SUMMARY. This article provides an empirical and theoretical foundation to support increased attention to neurodevelopmental processes in understanding the developmental sequelae of child emotional abuse (CEA). After reviewing the socioemotional consequences of CEA, an overview of the mammalian stress response system is provided, the deleterious impact of early psychosocial adversity on the organization and integration of this system is discussed, and the applicability of these findings for considering CEA and its developmental consequences within a multi-level, integrative, developmental psychopathology framework is explained. Building on evidence that CEA is likely to result in significant and enduring alterations in the neurobiology of stress response systems and, by extension, in neurodevelopment more broadly, specific suggestions for future research and practice are offered. This article encourages greater attention to CEA as a salient developmental experience

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and to neurophysiological processes as a heretofore overlooked source of information about the relation between CEA and adaptation.
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INTRODUCTION

Since Kempe's landmark article on battered child syndrome in 1962 (Kempe, Silverman, Steele, Droegemueller, & Silver), child maltreatment has emerged from the shadows as a major public health epidemic (Margolin & Gordis, 2000). Despite a burgeoning literature on the developmental sequelae of child physical and sexual abuse, however, less attention has been directed to the study (and treatment) of child emotional abuse (CEA; Behl, Conyngham, & May, 2003). In reviewing the empirical and theoretical literature on CEA, this article calls attention to the relevance of developmental neuroscience for understanding CEA and its consequences across multiple levels of analysis.

With the entrée of special issues and forums dedicated to understanding CEA in the late 1980s and early 1990s (Cicchetti & Nurcombe, 1991; Garrison, 1987), and the founding of this journal in 1998 (Geffner & Rossman, 1998), CEA has become a legitimate area of empirical and theoretical inquiry. However, a lack of conceptual and operational clarity as to what constitutes CEA has hampered efforts to identify and ameliorate its detrimental effects (Cicchetti & Nurcombe, 1991; Iwaniec, 1995). In this paper, CEA includes behaviors alternately referred to as "emotional maltreatment/abuse," "psychological maltreatment/abuse," and "nonphysical harm," that describe a caregiving pattern that conveys to children "that they are worthless, flawed, unloved, unwanted, endangered, or of value only in meeting another's needs" (American Professional Society on the Abuse of Children [APSAC], 1995, p. 2). Although I discuss both hostile/controlling and neglectful/unresponsive caregiving behavior under the broad umbrella of CEA, different subtypes of CEA are likely to have different effects on development, though this hypothesis remains to be tested empirically.

The purpose of this paper is to present evidence of the impact of CEA on child development and adaptation with particular emphasis on putative neurophysiological processes that may inform our understanding of pathways toward and away from psychopathology in the aftermath of CEA. To this end, I begin by summarizing the psychosocial and behavioral consequences of CEA. I then review relevant data drawn from research on the neurobiological effects of childhood trauma to provide a venue for considering how experience and biology may transact to eventuate in specific maladaptive (or positive) developmental outcomes following CEA. Here, I focus on the mammalian stress response system, particularly the coordinated actions of the limbic-hypothalamic-pituitary-adrenal (L-HPA) and the norepinephrine-sympathetic-adrenal-medullary (NE-SAM) systems. I review evidence that adversity and caregiving quality influence the mammalian stress response system in ways that affect development and adaptation. I encourage the adoption of a theoretically informed, multiple-levels-of-analysis approach to future research and practice on CEA. Specifically, I argue that the integrative paradigm of developmental psychopathology provides a conceptual framework to orient future investigations and interventions. In conclusion, I discuss the empirical and clinical implications of a developmental psychopathology approach for future research and practice aimed at understanding and mitigating the deleterious consequences of CEA.

THE SOCIOEMOTIONAL CONSEQUENCES OF CEA

Prospective and retrospective investigations implicate CEA in the etiology of significant and enduring deviations in socioemotional development (see Hart, Binggeli, & Brassard, 1998 for review). In their longitudinal study of a high risk poverty sample, Egeland and colleagues demonstrated prospective relations between CEA and insecure attachment to caregivers, noncompliance, low persistence, low enthusiasm, poor concentration, and declines in cognitive and motor competence across the first several years of life (Egeland, Weinfield, Bosquet, & Cheng, 2000). By school age, CEA was associated with high levels of negativity, impulsivity, poor social competence, low academic achievement, and increased psychopathology (see Erickson, Egeland, & Pianta, 1989 for review). Among the first to recognize and document the negative effects of CEA, the seminal work of Egeland and colleagues has been followed by other studies that clearly demonstrate specific associations between CEA and negative outcomes (e.g., Herrenkohl, Herrenkohl,

Egolf, & Wu, 1991; Solomon & Serres, 1999). Retrospective research has extended these findings into adulthood, demonstrating associations between CEA and anxiety, depression, personality disorders, suicidality, low self-esteem, and health problems (Briere & Runtz, 1988; Johnson et al., 2001; Mullen, Martin, Anderson, Romans, & Herbison, 1996; Spertus, Yehuda, Wong, Halligan, & Seremetis, 2003). Moreover, in several studies, the negative effects of CEA have been equivalent to, or greater than, those following other kinds of abuse or trauma (Briere & Runtz, 1988; Claussen & Crittenden, 1991; Gross & Keller, 1992; Mullen et al., 1996; Spertus et al., 2003; Vissing, Straus, Gelles, & Harrop, 1991).

Clearly, CEA is associated with serious and negative emotional and behavioral consequences. Indeed, some have suggested that CEA is *the* core factor underlying the deleterious effects of child maltreatment broadly (Hart et al., 1998; Navarre, 1987). As is the case in the broader literature on child maltreatment (Cicchetti & Toth, 2000), however, extant research on CEA has focused on psychological and behavioral consequences and mechanisms of psychopathology to the relative exclusion of biological processes. This, despite the growing body of empirical research indicating that child maltreatment may influence neurodevelopmental processes to alter the structure, organization, and function of the brain and its neurobiological systems (De Bellis, Baum et al., 1999; De Bellis, Keshavan et al., 1999; De Bellis & Putnam, 1994; Glaser, 2000; Perry & Pollard, 1998). Efforts to understand the neurobiological and neurodevelopmental consequences of early adversity have yet to examine if and how CEA may shape developmental pathways at the level of physiology.

A NEURODEVELOPMENTAL PERSPECTIVE

Over the past 20 years, our understanding of the development and functioning of the mammalian brain has increased dramatically. The enduring capacity for plasticity at the level of form and function is a central feature of the brain with processes related to cell proliferation, migration, differentiation, and death enabling both recovery from injury and untoward deviations following adversity (Kolb, 1989). While many systems are affected by, and are integrally involved in, stress responsivity (see Bremner & Vermetten, 2001; Chrousos, 1998; De Bellis, Baum et al., 1999 for reviews), here I focus on the physiology and neurobiology of

the core mammalian stress response systems that have been subject to the most empirical attention.

The Mammalian Stress Response

The mammalian stress response consists of two primary systems: the limbic-hypothalamic-pituitary-adrenal (L-HPA) axis regulates slower acting responses to stress and the norepinephrine-sympathetic-adrenal-medullary (NE-SAM) system underlies acute stress responses (Gunnar & Cheatham, 2003; Lopez, Akil, & Watson, 1999). In response to a perceived threat or stressor, the central nucleus of the amygdala activates the L-HPA and NE-SAM systems via connections with the hypothalamus and brainstem, respectively (Roozendal, Koolhaas, & Bohus, 1997). Operating at various sites throughout the central and peripheral nervous systems, these networks modulate behavioral, emotional, cognitive, metabolic, immunological, autonomic, and endocrine aspects of the mammalian stress response (Owens & Nemeroff, 1991).

The limbic-hypothalamic-pituitary-adrenal axis. The L-HPA axis, which consists of the hypothalamus, anterior pituitary gland, and adrenal cortices, regulates the longer acting and slower reacting stress response (see Figure 1; Vasquez, 1998). Following stress-induced amygdalar innervation, neurons in the paraventricular nucleus of the hypothalamus (PVN) secrete corticotropin-releasing hormone (CRH) into the hypophysial portal system that connects the hypothalamus to the pituitary gland. CRH travels through this system to the anterior pituitary where it stimulates the formation and release of adrenocorticotropic hormone (ACTH). Acting at receptors in the adrenal cortex, ACTH stimulates the release of glucocorticoids (cortisol in humans and primates, corticosterone in rodents) into the bloodstream. In turn, glucocorticoids act at receptors throughout the brain and body to suppress immune functioning, increase glucose conversion, reduce fear responses, influence learning and memory, reduce digestion, and inhibit further CRH secretion via negative feedback to the hypothalamus, pituitary gland, and hippocampus (Nelson & Carver, 1998).

The norepinephrine-sympathetic-adrenal-medullary system. In addition to its role stimulating the pituitary to release ACTH, CRH acts in the locus ceruleus (LC) of the brainstem to increase norepinephrine (NE) release and activate the sympathetic nervous system (SNS; see Figure 2; Valentino, Curtis, Page, Pavcovich, & Florin-Lechner, 1998). The NE-SAM system, which is comprised of the SNS and the adrenal medulla, stimulates

FIGURE 1. Schematic of the limbic-hypothalamic-pituitary-adrenal stress response system (L-HPA). In response to perceived threat or stress, corticotropin-releasing hormone (CRH) is secreted by the paraventricular nucleus of the hypothalamus. Acting in the anterior pituitary gland, CRH stimulates the production and release of adrenocorticotropic hormone (ACTH). ACTH acts in the adrenal cortices of the adrenal glands to stimulate the synthesis and release of glucocorticoids (i.e., cortisol in humans) into the bloodstream. In addition to its role of modulating long-term stress responses, cortisol provides inhibitory feedback to the brain (e.g., hypothalamus, pituitary gland, hippocampus) to modulate the subsequent production and release of CRH and ACTH. Connections among the hypothalamus and the amygdalar and hippocampal limbic structures are not shown here.

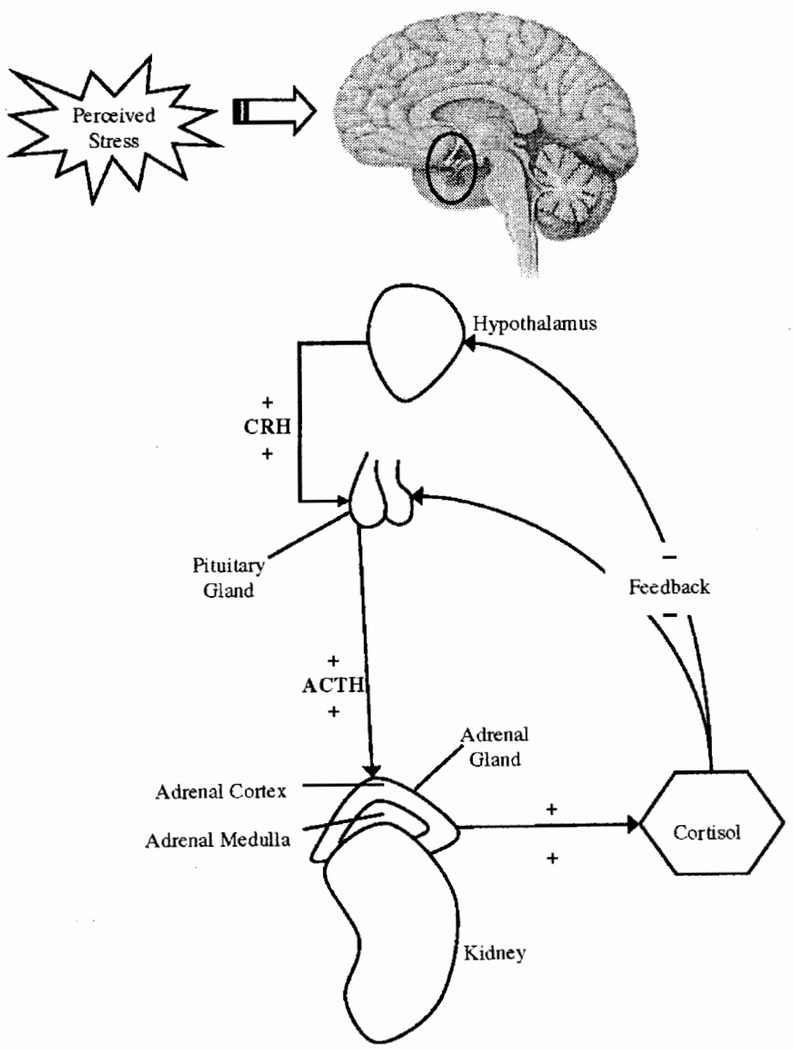
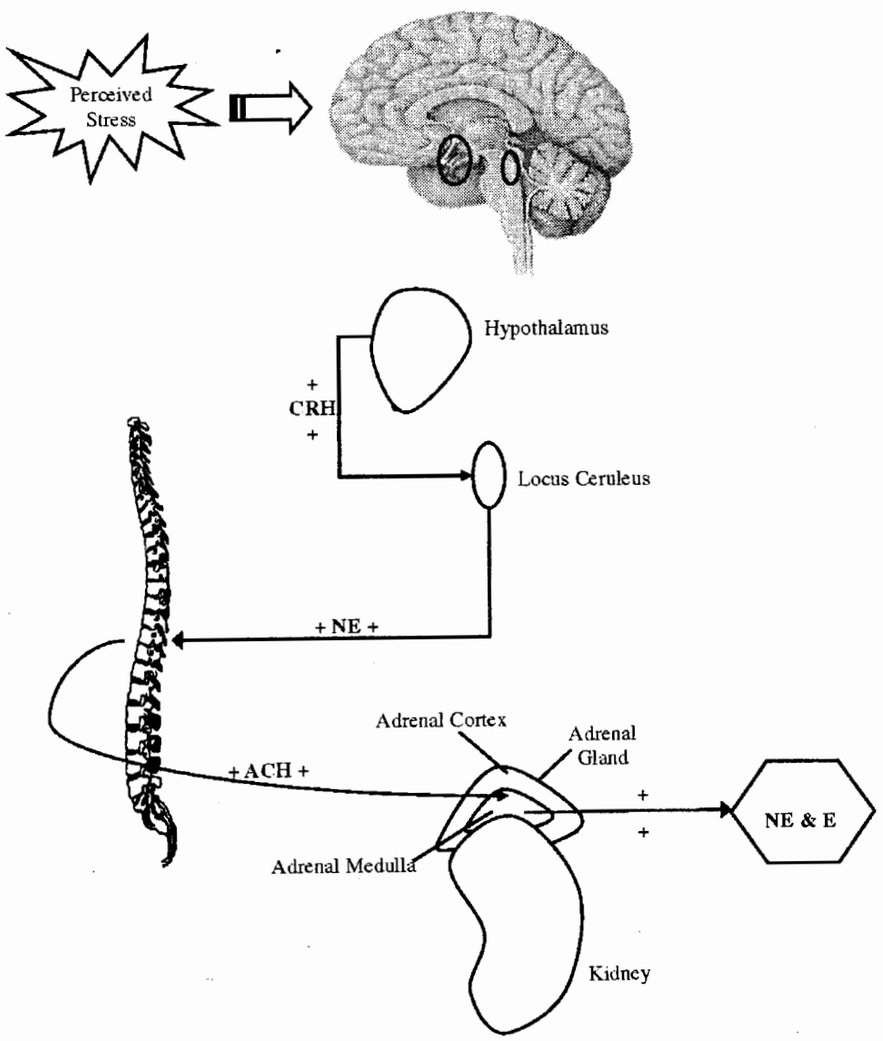


FIGURE 2. Schematic of the norepinephrine-sympathetic-adrenal-medullary system (NE-SAM). In response to perceived threat or stress, corticotropin-releasing hormone (CRH) is secreted by the paraventricular nucleus of the hypothalamus. Acting in the locus ceruleus, which is a nucleus in the brain stem, CRH stimulates the production and release of norepinephrine (NE). In turn, NE activates the sympathetic nervous system (SNS) and the release of acetylcholine (ACH) in the adrenal medulla of the adrenal glands. ACH stimulates the production and release of large amounts of epinephrine (E) and smaller amounts of NE into the blood stream to mediate acute stress responses. Connections among the hypothalamus and the amygdalar and hippocampal limbic structures are not shown here.



the production and release of NE and epinephrine (E) from the adrenal medulla into the blood stream where they act at receptors to elevate heart rate and blood pressure and ready the body for fight and flight responses to acute stressors (Koob, 1999). Together, the L-HPA and NE-SAM systems coordinate efficient and adaptive responses to stress via the peripheral release of adrenal steroids (i.e., glucocorticoids from the adrenal cortex) and catecholamines (i.e., NE and E from the adrenal medulla), respectively. Moreover, the coordinated action of these systems modulates processes related to neuronal migration, differentiation, synaptic proliferation, and, by extension, neurodevelopment (De Bellis, Keshavan et al., 1999).

Complementary coactivation. As primary stress mediators, glucocorticoids and catecholamines underlie pathways toward both positive adaptation and pathophysiology (Bremner, 1999; McEwen, 2000; Sapolsky, 1996). In the short-term, these systems are essential for effective responses to stressful stimuli, but dysregulation of these systems may contribute to enduring and pathological alterations as resources are directed away from long-term survival functioning in favor of short-term energy mobilization and response. Under normal circumstances, reciprocal connections within and between the L-HPA and NE-SAM systems serve to modulate the stress response (Nelson & Carver, 1998; Valentino et al., 1998). Simultaneous activation of the L-HPA and NE-SAM systems yields adaptive responding, but activation of one without the other may produce indiscriminate flight/fight reactions, depression, anxiety, and other symptoms of pathology (Yehuda, Southwick, Mason, & Giller, 1990). Furthermore, alteration of neurobiological stress systems may negatively influence other aspects of neurodevelopment (e.g., synaptic pruning, dendritic branching, neuronal death or endangerment; Sapolsky, 1996). Thus, alterations in L-HPA and/or NE-SAM stress response systems may mediate relations between early life stress and pathological outcomes (Bremner, Krystal, Sowthwick, & Charney, 1996; Cicchetti & Walker, 2003; Heim, Ehlert, & Hellhammer, 2000; Heim & Nemeroff, 2001; Kaufman, Plotsky, Nemeroff, & Charney, 2000).

PSYCHOSOCIAL ADVERSITY AND STRESS PHYSIOLOGY

Evidence from preclinical (i.e., animal) and clinical (i.e., human) studies converge on the assertion that adversity can instantiate neurophysiological alterations that undermine the adaptive organization and operation of mammalian stress response systems. Moreover, social factors, particularly the quality of early caregiving, have significant effects on

the development, organization, and enduring efficacy of these systems. In this section, I review preclinical and clinical studies that point to probable neurodevelopmental effects of CEA and their implications for adaptive functioning.

Preclinical Studies

Animal research consistently indicates that adversity in early development has a negative impact on the organization and efficacy of neurobiological stress response systems (see Francis, Caldji, Champagne, Plotsky, & Meaney, 1999; Sanchez, Ladd, & Plotsky, 2001 for reviews). Moreover, studies suggest that the quality of the early caregiving environment is a major influence on observed relations between adversity and stress regulation (see Francis & Meaney, 1999; Levine, 2001 for reviews). Brief separations between rodent pups and dams in handling paradigms yield very different effects than maternal separation paradigms, which expose the pup to prolonged separation from the dam. Separated rats exhibit larger and longer glucocorticoid responses to stress, reduced glucocorticoid receptor density in the hippocampus and prefrontal cortex, increased CRH activity, and larger central NE responses to threat via the LC. In contrast, handled rats exhibit decreased CRH activity, increased glucocorticoid receptor density in the hippocampus, and generally reduced stress reactivity (Ladd, Owens, & Nemeroff, 1996; Plotsky & Meaney, 1993). In sum, a brief stressor (i.e., handling) appears to enhance neurophysiological stress modulation, while a prolonged stressor (i.e., separation) undermines it.

Interestingly, studies indicate that the quality of maternal care upon reunification is a key mechanism underlying these divergent responses to handling and separation paradigms. After brief handling, there is an increase in maternal grooming and arched-back nursing, whereas longer separations lead to disorganized caregiving behavior. Early handling alters maternal grooming and nursing behavior in ways that protect the structure and functioning of the rodent pup's stress response system, while extended separations undermine maternal caregiving in a way that compromises the development of the pup's stress response systems (Caldji, Diorio, & Meaney, 2000). Extending these findings to nonhuman primates, recent studies demonstrate that exposure to unpredictable resource availability in a variable foraging paradigm stresses Bonnet macaque monkey mothers, degrades the quality of caregiving to infant monkeys, and increases the offspring's stress reactivity (Coplan, Paunica, & Rosenblum, 2004). Additional studies have shown that normative individual

differences in caregiving can influence stress response systems such that rodent mothers who groom and arched-back nurse their pups more tend to have more stress resistant offspring (Caldji et al., 1998; Liu et al., 1997). Finally, cross-fostering studies indicate that postnatal caregiving experience (e.g., taking a pup from a low grooming and arched-back nursing mother and cross-fostering it to a high grooming and arched-back nursing mother) modifies the pup's stress response systems, which demonstrates that these effects do not exclusively reflect genomic similarities (Francis, Diorio, Liu, & Meaney, 1999). These findings point to the importance of early caregiving quality for the development and operation of stress response systems.

Clinical Studies

Consistent with preclinical findings, clinical research indicates that early adversity may alter the neurobiology of stress response systems with enduring implications for human neurodevelopment and adaptation. Although the majority of research in this area has focused on populations exposed to traumatic stressors in childhood, growing evidence from broader samples supports preclinical findings that more subtle variations in the quality of early caregiving have salient effects on the organization of stress response systems. Together, these studies provide a strong evidentiary base for the assertion that CEA is likely to undermine the development and operation of human stress response systems with enduring negative implications for adaptation.

Research consistently demonstrates that child maltreatment contributes to marked deviations in normative neurobiological and neurodevelopmental processes related to the operation of the L-HPA and NE-SAM stress response systems. However, the specific direction of these effects (i.e., hypo versus hyperactivation) varies in ways that are not yet fully understood (see Cicchetti, 2003 for review). For example, in a large low-income sample of maltreated and nonmaltreated school-age children, Cicchetti and Rogosch (2001a) observed a pattern consistent with *hyper*cortisolemia among children with sexual or multiple abuse histories, but a pattern suggestive of *hypo*cortisolemia among physically abused children. Other researchers have suggested that initial patterns of hyperactivation may be followed by a progressive shift toward hypoactivation over time as a function of changing receptor densities (e.g., downregulation of glucocorticoid receptors in the anterior pituitary; De Bellis & Putnam, 1994). Despite changes as a function of maltreatment subtype, time elapsed since exposure, or other variables, it is important

to recognize that both hypo and hyper stress reactivity have negative implications for development and adaptation (Heim et al., 2000).

While studies of abused children suggest that marked deviations in early caregiving contribute to pathological alterations in developing stress response systems, researchers have recently begun to study more subtle variations in parenting processes through the lens of developmental neuroscience (see Bugental, Olster, & Martorell, 2003 for discussion). For example, Gunnar and colleagues have shown that the quality of maternal care shapes the normative development of stress reactivity over the first year of life. Infants with responsive caregivers exhibit age-expected declines in L-HPA axis activity between 6 and 15 months of age, whereas infants with less-responsive caregivers display an atypical increase in cortisol reactivity between 6 and 15 months of age (Gunnar, Broderson, Krueger, & Rigatuso, 1996). Similarly, secure attachment relationships, which typically follow from a history of sensitive and responsive caregiving, are associated with more adaptive stress responsivity (e.g., lower cortisol elevations in response to a stressor) than insecure attachment relationships, which are associated with less responsive and consistent caregiving (Gunnar, Brodersen, Nachmias, Buss, & Rigatuso, 1996; Nachmias, Gunnar, Mangelsdorf, Parritz, & Buss, 1996). Particularly pronounced deviations in normative stress responses (e.g., marked elevations in stress-induced cortisol and heart rate) have been observed among children with disorganized attachment relationships, which typically follow from the kind of unsafe, unpredictable, or severely misattuned caregiving that may characterize CEA (Hertsgaard, Gunnar, Erickson, & Nachmias, 1995; Spangler & Grossman, 1993).

As observed in animal studies of cross-fostering, these effects are not mediated exclusively by genomic processes. Investigations of substitute care strongly suggest that observed relations between parenting quality and child stress responses transcend the contribution of genetic similarity. For example, in a study of 9-month-old infants, Gunnar and colleagues (1992) found that the child's response to a 30-minute separation from the caregiver was moderated by the quality of the substitute care provided (Gunnar, Larson, Hertsgaard, Harris, & Broderson, 1992). Infants did not evidence a stress response when placed with a responsive babysitter, but they did exhibit increased cortisol when placed with an insensitive babysitter. Similar findings have been observed in investigations of the relation between the quality of out-of-home child care and stress reactivity in young children, with high quality care setting associated with reduced levels of L-HPA dysregulation (e.g., Dettling, Parker, Lane, Sebanc, & Gunnar, 2000).

CHILD EMOTIONAL ABUSE AND NEURODEVELOPMENT

Preclinical and clinical research studies demonstrate that enduring alterations in the activity of the L-HPA and NE-SAM stress response systems may follow from adverse experiences in childhood. Evidence suggests that the quality of the early caregiving environment may moderate or mediate these relations. Moreover, even relatively subtle variations in the quality of early care appear to affect developing stress response systems. To date, however, few studies have examined whether these patterns generalize to CEA, whether effects differ across subtypes of CEA (e.g., unavailable versus intrusive care), and what the implications of these potential alterations may be for later development and adaptation.

The available literature suggests that recurrent patterns of hostile, indifferent, degrading, and unpredictable emotional exchanges in the caregiving milieu, as may typify instances of CEA, will have negative and enduring effects on emerging stress response systems and adaptation. Of the few studies that have included assessments of CEA in their investigations of stress responsivity, the majority indicate that CEA is associated with dysregulation of both the L-HPA and NE-SAM stress response systems. Bugental and colleagues (2003) found that young children who had been emotionally abused in the first year of life exhibited atypical elevations in basal levels of cortisol suggesting L-HPA axis dysregulation (Bugental, Martorell, & Barazza). A similar relation between CEA and elevated L-HPA axis activity has been found in a sample of adult children of Holocaust survivors who report a history of CEA (Yehuda, Halligan, & Grossman, 2001). With respect to noradrenergic functioning, Jones and colleagues (1997) found that intrusive parenting was associated with elevated catecholaminergic transmission suggesting hyperactivation of the NE-SAM system. In contrast to the studies reviewed thus far, Cicchetti and Rogosch (2001a) did not find differences in cortisol regulation patterns between children with a history of CEA and a high-risk nonmaltreated comparison sample. However, these authors note that the prevailing context of risk in this study may have overshadowed meaningful differences between children with and without histories of CEA.

In addition to CEA-induced alterations in stress responsivity, a growing body of evidence indicates that CEA may contribute to dysregulated stress response patterns indirectly. For example, CEA may moderate relations between physical or sexual abuse and stress-response alterations. In a study of depressed maltreated, depressed nonmaltreated, and nonmaltreated children, Kaufman and colleagues (1997) observed

differences in L-HPA functioning following intravenous administration of CRH. As expected, depressed abused children evidence higher levels of ACTH release than both nonmaltreated groups; however, all the depressed maltreated children who exhibited increased ACTH release were living in homes with ongoing CEA.

In addition to direct effects on neurodevelopment via the introduction or mitigation of arousing stimuli, CEA may influence psychological processes that, in turn, affect the child's stress responsivity. Intrapsychic mechanisms, such as perceived control and predictability, can regulate the activity of stress response systems (Granger, Weisz, McCracken, Ikeda, & Douglas, 1996; Sapolsky, 1994). Thus, CEA may indirectly affect stress responsivity via representational processes (e.g., reductions in self-esteem; Gross & Keller, 1992) that might contribute to altered stress reactivity. As the mechanisms by which CEA undermines effective stress regulation come into focus, attention should shift toward the study of the adaptive consequences of CEA-induced alterations in stress physiology.

IMPLICATIONS FOR ADAPTATION

The specification of neurodevelopmental mechanisms in the pathophysiology of mental disorders is a prominent focus of contemporary experimental psychopathology (see Cicchetti & Walker, 2003 for review). Research suggests that alterations in the neurobiology of stress responsivity may contribute to contemporaneous and prospective adaptational difficulties that have been associated with CEA. Among school-aged children, for example, early adversity and consequent alterations in stress physiology have been associated with reduced social, cognitive, and emotional competence (Gunnar, Tout, deHaan, Pierce, & Stansbury, 1997; Hart, Gunnar, & Cicchetti, 1995). In adult samples, dysregulation of stress response systems has been associated with anxiety disorders and depression (Heim, Owen, Plotsky, & Nemeroff, 1997; Nemeroff, 2004). Available evidence suggests that CEA may cause deviations in normative stress response development that contribute to disorders of adaptation that have been associated with CEA.

In addition to the physiological consequences of CEA, recent findings suggest that there may be physiological causes of CEA. Animal research indicates that the intergenerational transmission of parenting behaviors may occur via experience-induced alterations in stress response physiology (Francis, Diorio et al., 1999). In an unpublished study

by Martorell and Bugental (as summarized in Bugental, Martorell et al., 2003), parents who endorsed low levels of perceived power were more likely to show cortisol elevations in response to stressful interactions with their toddlers, which, in turn, contributed to punitive parenting. Thus, parents who respond to challenges in the caregiving relationship with physiological activation may be more likely to engage in punitive parenting practices. To the extent that CEA increases stress reactivity and decreases levels of self-esteem and self-efficacy, it may increase the probability of compromised parenting in the next generation. These findings support the assertion that, as a chronic relational adversity in childhood, CEA carries a high probability for inducing neurobiological deviations in development that are implicated in the pathophysiology of subsequent maladaptation, including the intergenerational transmission of child maltreatment.

A FRAMEWORK FOR FUTURE RESEARCH AND PRACTICE

In this paper, I encourage greater attention to putative neurodevelopmental processes that may be affected by CEA and that, in turn, influence social, emotional, and behavioral adaptation. To this end, I provide specific suggestions for future research and practice that incorporate psychobiological processes across multiple levels of analysis and intervention. As observed by Gottlieb and Halpern (2002), and as supported by the research reviewed here, the cause of development is neither biology, nor the environment, but rather the relation within and among developmental systems and their components. I contend that a multiple-levels-of-analysis approach to future research and practice is essential to our understanding of the specific relations within and among the psychosocial, behavioral, and biological systems involved in CEA and its developmental sequelae.

Directions for Future Research

Over the past decade, scholars have highlighted the need for interdisciplinary research efforts across multiple levels of analysis (Cacioppo & Berntson, 1992; Cicchetti & Blender, 2004). However, a relational view of causality in research requires more than information from multiple levels of analysis; it requires theoretically informed hypotheses that specifically consider relations across systems (Gottlieb & Halpern, 2002). Thus, interdisciplinary, multi-level research requires a conceptual

framework that can accommodate multiple sources of information and appreciates that "there are psychological phenomena that derive from events at one level of analysis and that are only or more distinctly observable across levels of analysis" (Cacioppo & Berntson, 1992, p. 1023).

As an integrative conceptual framework that draws on the principles of core developmental theories and models, developmental psychopathology is especially well-suited for orienting future research and practice related to CEA within an interdisciplinary, multi-level systems approach (Cicchetti, 1993; Masten, 2006; Sroufe & Rutter, 1984). Developmental psychopathology localizes positive and pathological adaptation in the transactional relations between individuals and their internal and external environments, rather than as inherent to the individual or the environment (Cicchetti & Toth, 1997). This transactional view of development readily encourages the integration of biological and psychological levels of analysis within a common conceptual framework. Building on a developmental psychopathology framework and adopting multi-level paradigms, future research on the developmental consequences of CEA will advance us toward an integrative understanding of the neurophysiological and psychosocial transactions that follow from CEA to eventuate in particular adaptive outcomes. To this end, I offer the following suggestions for future research on CEA.

First, there is a need for greater clarity in defining CEA, and for improved measures to enable its reliable assessment across the developmental continuum (see Hart, Brassard, Binggeli, & Davidson, 2001 for a review of these issues). Similarly, given the tremendous value of experimental manipulations in animal research, there is a marked need for the development of animal paradigms that can approximate the human experiences of degradation, humiliation, and betrayal that typify much of child maltreatment, particularly CEA. Although there are significant limitations to the translation of findings across species, animal studies remain an important resource for initial hypothesis testing to inform clinical research and for identifying neurophysiological mechanisms of change that cannot be directly observed in human samples.

Second, just as the conceptualization and assessment of CEA will change across time and context, so, too, must future research trace patterns of adaptation and transaction over time. As discussed previously, the physiological and behavioral effects of CEA will vary as a function of the developmental status of the individual at the time of exposure and of the time that has elapsed since exposure (Nelson & Carver, 1998; Teicher, 2002). Similarly, there is a need for longitudinal research designs to test if and how CEA-induced physiological changes affect

long-term adaptation. A longitudinal, process-level approach to the study of CEA and adaptation will permit the specification of causal relations, as well as the identification of intervening factors that may moderate pathways towards and away from pathological outcomes.

Third, future investigations must explicitly address the dynamic nature of development and adaptation as outgrowths of transactions among multiple, embedded, overlapping, and interacting systems. Specific features of CEA, including age of the child at time of onset, gender of the child victim, gender of the perpetrator, frequency of abuse, presence of other forms of abuse, and how the child perceives and makes meaning out of the abuse may influence the impact of CEA on the child's physiology, psychology, and adaptation (Cicchetti & Rogosch, 2001a; Manly, Kim, Rogosch, & Cicchetti, 2001; Mullen et al., 1996). Moreover, specific investigations must examine if and how hostile/controlling caregiving might differentially affect neurophysiological development relative to emotionally neglectful/unresponsive caregiving. Across levels of ecological influence, the presence of protective or vulnerability factors related to socioeconomic status, parenting quality, social support, and culture may moderate the relation between adversity and adaptation (Yates & Masten, 2004). Finally, the child's genetic constitution, developmental history, and the quality of her/his current adaptation (e.g., comorbid psychopathology) may influence the relation between CEA exposure and response (Cicchetti & Rogosch, 2001b; Kaufman et al., 1997; Sapolsky, 1994). As our understanding of the impact of adversity on neurobiological development and adaptation advances, we must achieve similar gains in our recognition of factors that moderate observed relations among adversity, neurodevelopment, and socioemotional adaptation.

Fourth, in keeping with the call for greater attention to moderating variables in future research, a developmental psychopathology framework encourages attention not only to processes that engender risk, but also to those that confer strength in the face of vulnerability. Just as psychobiological processes may contribute to maladaptation, so, too, may they underlie the better-than-expected processes and outcomes that typify resilience (Curtis & Cicchetti, 2003; Davidson, 2000). Research aimed at identifying both positive and pathological pathways following CEA will further our understanding of specific processes underlying observed patterns. Indeed, the salience of parenting quality for the developing stress response system was revealed only by the study of the better-than-expected outcomes that followed from early handling paradigms. As observed by Curtis and Cicchetti (2003), such research may

profitably explore whether positive adaptation in the face of adversity derives from greater resistance to adversity, greater resources for recovery, and/or greater capacity for compensation.

As a macroparadigm, developmental psychopathology is uniquely equipped to bridge artificial dualisms between different lenses of empirical inquiry, between behavioral and biological science, and between basic and applied research. Building on this framework, future research must adopt interdisciplinary, integrative paradigms that can be readily translated to real-world practice with children and families. In addition to the adoption of a developmental psychopathology framework, there must be an appreciation for interdisciplinary collaborations at the level of funding agencies and professional evaluative networks. In short, an individualistic science of psychology cannot uncover the dynamic multisystem transactions that underlie development and adaptation. Future research must transcend single-level designs (e.g., including both biological and socioemotional assessments) and incorporate multiple methods and measures within levels (e.g., including several indicators of physiological functioning, such as cortisol, catecholamines, neuroelectrical activity and neuroimaging). Undoubtedly, this work rests at the precipice of psychology's growing edge (see Nelson et al., 2002 for a discussion of issues and examples of this kind of work). However, understanding the relations between adversity and neural development, and identifying factors that moderate these relations, holds tremendous promise for intervention efforts aimed at reducing the deleterious impact of early adversity.

Directions for Future Practice

Relative to interventions targeting physically and sexually abused children, and to a lesser degree neglected children, there has been little attention directed toward helping emotionally abused children. Psychotherapeutic and/or pharmacological interventions may prevent or reverse the deleterious effects of early stress exposure at both behavioral and physiological levels (Curtis & Nelson, 2003; Kandel, 1998; Nelson, 2000). Just as plasticity renders the organism vulnerable to deviations in adaptive processes, so, too, does it confer a capacity for resistance, self-righting, and recovery. Adopting a developmental psychopathology perspective in research on both positive and pathological pathways following exposure to CEA has important implications for the design and implementation of effective intervention efforts, especially in terms of identifying particular populations, and systems within them, to target.

One implication of this approach is that periods of rapid development harbinger greater vulnerability to both positive and negative influences. As such, childhood, with its attendant elevation in neural growth and modification, may be an especially vulnerable period, either to the degenerative effects of maltreatment or to the restorative effects of intervention. That being said, the potential for psychobiological plasticity extends well into adulthood such that the human brain continues to respond to both positive (e.g., training) and negative (e.g., injury) experiences over the life course (Nelson & Bloom, 1997). Although it is important for intervention efforts to target core developmental systems in early childhood, the inclusion of follow-up supports will be important for the maintenance of positive gains over time.

Preclinical and clinical studies suggest that the caregiving system may be especially influential in the prevention, amelioration, or reversal of negative consequences related to CEA. Animal studies demonstrate that environmental factors (e.g., social enrichment, improved caregiving) can modulate neurogenerative and stress response processes (Francis, Diorio et al., 1999; Liu et al., 1997). In humans, attachment security, which is a proxy for sensitive and responsive caregiving, buffers human stress response systems (Gunnar, 1998; Nachmias et al., 1996). The attachment system is a profitable target for intervention because it carries the possibility for both protective and restorative processes at multiple levels of action (see Egeland, Weinfield, Bosquet, & Cheng, 2000 for review). In a recent intervention study, Fisher and colleagues (2000) demonstrated the efficacy of early intervention (EI) efforts focused on the quality of the child-caregiver relationship for both behavioral and physiological aspects of development. In this study, a group of maltreated preschoolers (sexual abuse, physical abuse, exposure to partner violence and other trauma) were placed in an EI foster care program that consisted of foster parent education, child and family therapy, and parent support groups intended to encourage consistent (nonabusive) discipline, positive reinforcement, and close monitoring of the child. After 12 weeks of treatment, behavioral effects were found for both foster parents and children. The EI group exhibited improved parenting practices relative to regular foster care parents. In addition, the children in the EI group evidenced notable declines in symptom endorsement on an inventory of child behavior problems, while children in the regular foster care group evidenced progressive increases in behavioral maladaptation. Salivary cortisol measures indicated that the EI children exhibited a significant shift toward normative circadian cortisol release over the course of treatment, whereas children in regular foster care exhibited

increasingly atypical patterns. Although preliminary in nature, these findings suggest that early intervention and prevention efforts may prove integral to socioemotional *and* neurophysiological adaptation and recovery (see Dozier, Lindheim, & Ackerman, 2005 for review).

As evident in the EI program described above, successful intervention with high-risk families requires a multi-faceted approach to service provision that aims to support and restore core adaptational systems, such as the attachment relationship (see Erickson, 1998 for discussion). To this end, efforts to prevent CEA and support positive parenting might include services to reduce caregiver strain (e.g., economic, educational, occupational resources), improve caregiver understanding of child development (e.g., parent education, parent sensitivity training), and foster social networks that can maintain positive change beyond the parameters of a particular intervention (e.g., home visitation, support groups). Just as pathology derives from multiple levels of influence, so, too, must intervention efforts transcend these levels to foster positive developmental outcomes.

Developmentally appropriate, systems-oriented, multi-pronged prevention and intervention efforts will emerge out of interdisciplinary collaborations between scholars and practitioners, between animal and human researchers, and between scientists and the communities they serve. Historically, interdisciplinary and translational endeavors of this kind have been stymied by overemphasis on specialization and individual achievement in training and funding organizations. However, there is a growing recognition that the inclusion of diverse sources of information, such as evaluation research or biological measures of development and adaptation, not only informs interventions, but also affirms, expands, and challenges extant theories about adversity and adaptation (Cicchetti & Hinshaw, 2002; Yates & Masten, 2004).

CONCLUDING COMMENTS

CEA disrupts development across multiple domains, including social, emotional, self, cognitive, and biological processes. Although research has heretofore focused almost exclusively on psychological mechanisms in understanding pathways from CEA to various outcomes, evidence indicates that CEA has the capacity to initiate persistent alterations in neurophysiological stress response systems that lead to increased vulnerability for stress, anxiety, depression, and other problems of adaptation. In order to better understand these processes and identify meaningful

ways to intervene, research and practice must draw on multiple levels of analysis across theoretical, empirical, and applied domains. The integrative paradigm of developmental psychopathology provides a conceptual framework that can bridge prior factions between science and practice and between developmental psychology and neurobiology.

Understanding the psychobiological correlates and consequences of child maltreatment broadly, and CEA in particular, has significant importance for future research and programming aimed at mitigating or reversing its negative impact on development, as well as for potentially preventing its transmission to subsequent generations. As reviewed here, a preponderance of evidence indicates that "adequate nurturance and the absence of intense early stress permits our brains to develop in a manner that is less aggressive and more emotionally stable, social, empathic and hemispherically integrated" (Teicher, 2002, p. 75). This article encourages and informs the development and implementation of multi-faceted, developmentally informed interventions that encompass multiple levels of change and adaptation to foster basic adaptational systems that buffer and scaffold physiological and psychological development.

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