Development and Psychopathology, **13** (2001), 539–564
Copyright © 2001 Cambridge University Press
Printed in the United States of America

Developmental traumatology: The psychobiological development of maltreated children and its implications for research, treatment, and policy

MICHAEL D. DE BELLIS
University of Pittsburgh Medical Center; and Western Psychiatric Institute and Clinic

Abstract
In this review, a developmental traumatology model of child maltreatment and the risk for the intergenerational cycle of abuse and neglect using a mental health or posttraumatic stress model was described. Published data were reviewed that support the hypothesis that the psychobiological sequelae of child maltreatment may be regarded as *an environmentally induced complex developmental disorder*. Data to support this view, including the descriptions of both psychobiological and brain maturation studies in maltreatment research, emphasizing the similarities and differences between children, adolescents, and adults, were reviewed. Many suggestions for important future psychobiological and brain maturation research investigations as well as public policy ideas were offered.

Child maltreatment may be the single most preventable and intervenable contributor to child and adult mental illness in this country. Adults with child maltreatment histories are more likely to manifest multiple health risk behaviors and serious medical illnesses (Felitti, Anda, Nordenberg, Williamson, Spitz, Edward, Koss, & Marks, 1998) and greater rates of psychiatric and medical utilization (Walker, Unutzer, Rutter, Gelfand, Saunders, VonKorff, Koss, & Katon, 1999) than adults without maltreatment histories.

Multiple, densely interconnected neurobiological systems are impacted by the acute and chronic stressors associated with childhood maltreatment. These neurobiological systems significantly influence physical and cognitive development and emotional and behavioral regulation. Results from recent research suggest that the overwhelming stress of maltreatment experiences in childhood is associated with alterations of biological stress systems and with adverse influences on brain development (De Bellis, Baum, Birmaher, Keshavan, Eccard, Boring, Jenkins, & Ryan, 1999; De Bellis, Keshavan, Clark, Casey, Giedd, Boring, Frustaci, & Ryan, 1999).

Developmental traumatology is the systemic investigation of the psychiatric and psychobiological impact of overwhelming and chronic interpersonal violence (child maltreatment) on the developing child. This is a relatively new area of study in child psychiatry that synthesizes knowledge from develop-

Some of the ideas presented in this manuscript are in *Child Abuse and Neglect: A Public Mental Health Problem,* by M. D. De Bellis, master's thesis submitted in April 2000 to the Graduate Faculty of the Graduate School of Public Health in partial fulfillment of the requirements for the degree of Master of Public Health.

Address correspondence and reprint requests to: Michael D. De Bellis, MD, MPH, Associate Professor of Psychiatry, University of Pittsburgh Medical Center, Director, Developmental Traumatology Neuroimaging Laboratory, Western Psychiatric Institute and Clinic, 3811 O'Hara Street, Pittsburgh, PA 15213; E-mail: DeBellismd @msx.upmc.edu.

mental psychopathology, developmental neuroscience, and stress and trauma research. The development of the brain is regulated by genes, which interact profoundly with life experiences, particularly early childhood experiences. Active areas of research investigate the consequences of child maltreatment and related family and psychosocial stressors and their effects on the development and regulation of major biological stress response systems and their influence on childhood brain development and function.

In developmental traumatology research, abuse and neglect are seen as a most extreme form of dysfunctional family and interpersonal functioning on a continuous spectrum of adverse life circumstances and dysfunctional interpersonal and family relationships. These adverse life circumstances include socioeconomic disadvantage, parental mental illness (including alcohol and substance abuse), community violence, and a lack of adequate social support and experience-expected environmental stimulation. The influence of maltreatment, as well as these other factors on biological stress systems regulation and brain maturation, are complicated and very difficult to disentangle. An important mission for the field of developmental traumatology research is to unravel the complex interaction between an individual's genetic constitution, unique psychosocial environment, and proposed critical periods of vulnerability for and resilience to maltreatment experiences, and how such factors may influence changes in biological stress systems, adverse brain development, and known serious consequences associated with child maltreatment. Developmental traumatology is the study of these complex interactions.

The Basic Assumptions of Developmental Traumatology Research

This review focuses on the psychobiological development of maltreated children. A mental health model, the diagnosis of posttraumatic stress disorder (PTSD), is applied to ideas arising from developmental psychopathology and developmental neuroscience. It is hypothesized that the potential psychobiological sequelae of child maltreatment may be regarded as *an environmentally induced complex developmental disorder*. Based on a synthesis of the relevant literature, a number of assumptions are made in this review.

The first assumption is that while there are an infinite number of stressors that can cause a subjective sense of overwhelming stress and distress in a child, there are finite ways that the brain and the body (i.e., biological stress systems) can respond to those stressors. Therefore, our discussion of the consequences of childhood maltreatment will purposely and nonspecifically include a broad definition of maltreatment (abuse and neglect), unless the current data support different outcomes for the various types of maltreatment. In accord with this idea, it is noted that child abuse and neglect are chronic conditions and that various forms of abuse and neglect tend to coexist (Cicchetti & Barnett, 1991). Therefore, in this review the terms maltreatment and chronic trauma are used interchangeably.

The second assumption, and the most important one in using the PTSD model in child maltreatment, involves the nature of the stressor, which is a dysfunctional and traumatized interpersonal relationship. The trauma is not only the act of maltreatment itself (e.g., physical abuse or sexual abuse) but also the relationship the victim has with the perpetrator of the trauma. Therefore, clinical identification of traumatic triggers may involve identification of subtleties that can sometimes be difficult to clinically assess. Along these lines, interpersonal stressors, such as child maltreatment, are more likely to be chronic and more severe than noninterpersonal traumas. An interpersonal stressor likely involves the maltreated child losing faith and trust in a parent or an authority figure. Thus, for the maltreated child the ability to form relationships and attachments is intact (e.g., the hardwiring is present) but traumatized (e.g., the software is programmed to distrust and fear relationships). Consequently, the maltreated child will be more difficult to treat in psychotherapy and have a harder time forming healthy social relationships, because the establishment

of a therapeutic alliance involves a process of desensitizing the maltreated individual to distrust and will take more time to progress.

The third assumption is that maltreatment in childhood may be more detrimental than trauma experienced in adulthood secondary to interactions between trauma and psychological and neurodevelopment. Hence, maltreatment in childhood may cause delays in or deficits of multisystem developmental achievements in behavioral, cognitive, and emotional regulation. The fourth assumption is that biological stress system responses will be based on several principles, including the nature of the stressor, the frequency and chronicity of the stressor, the individual differences (i.e., genetic vulnerabilities) in biological stress systems regulation and in their response to the stressor, and the ability of biological stress systems to either maintain homeostasis in the face of chronic and severe stress or permanently change in response to the stressor.

The fifth assumption is that PTSD symptoms are common responses to severe stressors. The sixth assumption is these changes in biological stress systems cause psychiatric symptoms, particularly symptoms of PTSD. Therefore, lack of PTSD symptoms after experiencing a severe stressor will be associated with little psychopathology. However, experiencing PTSD after a severe stressor in childhood will lead to an increase risk of suffering from chronic PTSD, other psychopathology (i.e., internalizing or emotional and externalizing or behavioral disorders), and other cognitive and psychosocial consequences. The seventh assumption is that when trauma occurs during development, chronic PTSD symptoms can be seen as the trajectory to more severe comorbidity and compromised cognitive and psychosocial functioning and this pathway increases the intergenerational transmission of abuse and neglect. (See Figure 1.)

Throughout this review, we will present and review data to support these ideas and descriptions of the state of research. The similarities and differences between children, adolescents, and adults, and the methodological difficulties and controversies inherent in this field of study, will be emphasized. Suggestions for future research opportunities, treatment, and policy will be offered.

Is the PTSD Model Appropriate for Studies of Maltreated Children?

The diagnosis of PTSD is made after a person experiences one or more intense, overwhelming, traumatic event and reacts with fear or disorganized behavior and with complaints of three clusters of categorical symptoms for at least 1 month (a) intrusive reexperiencing of the trauma(s), (b) persistent avoidance of stimuli associated with the trauma, and (c) persistent symptoms of increased physiological arousal (American Psychiatric Association, 1994). The diagnostic picture of PTSD in children and adolescents is similar to adults (for review, see De Bellis, 1997; Pynoos & Eth, 1985), with the exception of children less than 4 years old, where more objective criteria based on observable behaviors are warranted (Scheeringa, Zeanah, Drell, & Larrieu, 1995).

A review of the longitudinal course of PTSD suggested that PTSD symptoms are common within the 1st month of a trauma and that these symptoms may be a normal response to severe stress, as these symptoms usually fade within 3 months (Blank, 1993). PTSD may be better conceptualized as a dimensional process rather than a categorical all-or-none outcome, as complete and partial PTSD responses are usually seen in many forms of trauma including victims of childhood maltreatment (Armsworth & Holaday, 1993; Famularo, Fenton, & Kinscherff, 1994; Hillary & Schare, 1993; Mannarino, Cohen, & Berman, 1994; Wolfe, Sas, & Wekerle, 1994; Wolfe & Charney, 1991). Chronic PTSD symptoms may provide the mechanisms for the pervasive psychopathology seen in maltreated children. No complaints of PTSD symptoms after experiencing a severe stressor (i.e., lack of sleep disturbances, intrusive symptoms, or concentration impairments) may be associated with little psychopathology. Along these lines, children and adolescents with PTSD secondary to maltreatment may be one of the most seriously affected groups of maltreated children. These ideas

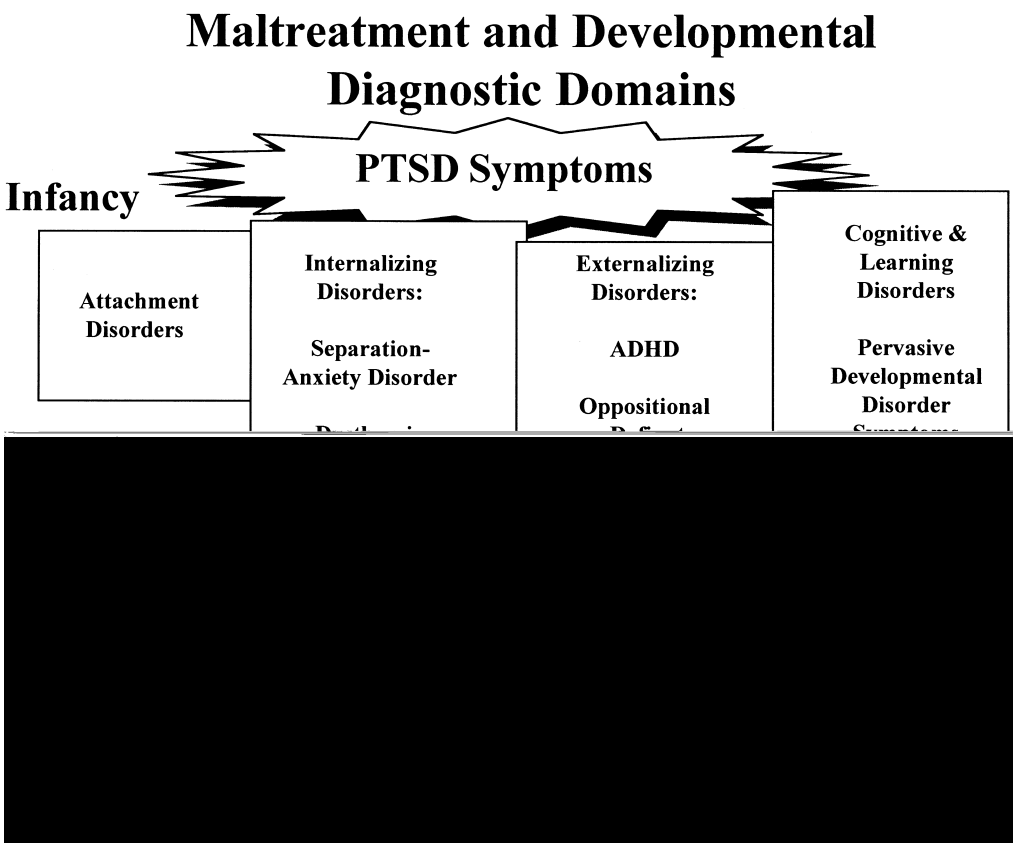

Figure 1. A developmental traumatology model for the intergenerational transmission of maltreatment. In this model the intergenerational transmission of child abuse and neglect is seen as transmitted through parental mental illness, and it is understood to be a result of (a) the impact of childhood traumatic stress on later parental biopsychosocial development, (b) consequent adverse parental brain development, (c) consequent parental mental illness, which may lead to (d) adverse parenting skills. This model is best thought of as multidetermined (i.e., genetic and environment interactions leading to outcomes).

represent an understudied area and perhaps a fruitful area for further research. However, because of the lack of "dimensional" PTSD data, the ideas presented in this review are based on the results of clinical studies, which defined PTSD as a categorical all-or-none outcome.

In clinically referred samples, the reported incidence rates of PTSD resulting from sexual abuse range from 42 to 90% (Dubner & Motta, 1999; Lipschitz, Winegar, Hartnick, Foote, & Southwick, 1999; McLeer, Callaghan, Henry, & Wallen, 1994), from witnessing domestic violence from 50 to 100% (for domestic homicide; Pynoos & Nader, 1989), and from physical abuse to as high as 50% (Dubner & Motta, 1999; Green, 1985). Only a few studies have focused on assessing PTSD in nonclinically referred maltreated children. Famularo et al. (1994) reported a 39% incidence rate of PTSD in a nonclinically referred maltreated sample interviewed within 8 weeks of abuse or neglect disclosure. About a third of the PTSD subjects reexamined from the original sample continued to meet PTSD criteria at 2-year follow-up (Famularo, Fenton, Augustyn, & Zuckerman, 1996). Recently, (McLeer, Dixon, Henry, Ruggiero, Esovitz, Niedda, & Scholle, 1998) reported prevalence rates of PTSD of 36.3% in nonclinically referred sexually abused children 60 days immediately following sexual

(^{1}H) spectrum mainly comprises of *N*-acetylaspartate (NAA) and is considered to be a marker of neural integrity. Decreased NAA concentrations are associated with increased metabolism and loss of neurons (for review, see Prichard, 1996). We recently completed a preliminary investigation suggesting that maltreated children and adolescents with PTSD have lower NAA-to-creatine ratios that are suggestive of neuronal loss in the anterior cingulate region of the medial prefrontal cortex compared to sociodemographically matched controls (De Bellis, Keshavan, Spencer, & Hall, 2000). These results were not specific to gender. These findings may reflect global neuronal loss in childhood PTSD, a possibility supported by our previous findings discussed above. Neuronal loss in the anterior cingulate of pediatric PTSD patients agree with the recent adult neuroimaging studies, which provide evidence for anterior cingulate dysfunction in adult PTSD. Thus, dysfunction of the anterior cingulate cortex, which is involved in the extinction of conditioned fear responses, may be implicated in the pathophysiology of both adult and child PTSD.

PTSD is an anxiety disorder and anxiety disorders have been associated with dysregulation of growth hormone secretion (Pine, Cohen, & Brook, 1996). Stress-related abnormalities in growth hormone may affect brain and body maturation (for review, see De Bellis & Putnam, 1994). We undertook a pilot study comparing measures of brain regions in nontraumatized, nonmaltreated children and adolescents with DSM-IV generalized anxiety disorder with sociodemographically matched controls. Right and total amygdala volumes were significantly larger in subjects with generalized anxiety disorder compared to controls (De Bellis, Casey, Dahl, Birmaher, Williamson, Thomas, Axelson, Frustaci, Boring, Hall, & Ryan, 2000). Intracranial, cerebral, cerebral gray and white matter, temporal lobe, hippocampal and basal ganglia volumes, and measures of the midsagittal area of the corpus callosum did not differ between groups. The amygdala and its related circuits are important in fear-related behaviors (Davis, 1997; LeDoux, 1998). These results are consistent with the idea that alterations in the amygdaloid structure and function, but not of global brain measures, may be associated with pediatric generalized anxiety disorder. Increased amygdala volume may index some genetic trait such as increased sensitivity to threat cues, which could create a vulnerability for pediatric generalized anxiety disorder. Unlike children with pure anxiety disorders, maltreated children and adolescents with PTSD showed global adverse brain development and no anatomical changes in limbic (hippocampal or amygdala) structures (De Bellis, 1999). These results provide indirect evidence that PTSD in maltreated children may be regarded as a *complex environmentally induced developmental disorder. This provides further evidence for the need to study brain maturation in traumatized children and carefully characterize trauma histories when studying the psychobiology of psychiatric disorders that share symptoms in common with PTSD, throughout the life span. The safety and availability of MR methods make this an exciting time to disentangle these challenges.*

Our results of lack of hippocampal findings are in contrast to the adult PTSD literature. *We did not find the predicted decrease in hippocampal volume.* Subcortical gray matter structures that include the limbic system (septal area, hippocampus, amygdala) actually show an increase in volume during adolescence (Jernigan & Sowell, 1997). This increase may "mask" any effects of traumatic stress in maltreated children with PTSD. *Since our subjects had less comorbid histories of alcohol and substance abuse* (*four of 43 subjects*), our maltreated PTSD subjects differed in the degree of psychiatric comorbidity from the adult PTSD studies. High rates of comorbid lifetime alcohol dependence were seen in adult MRI studies of PTSD secondary to combat exposure (Bremner et al., 1995; Gurvits et al., 1996), adult PTSD secondary to child abuse (Bremner et al., 1997), and female adult survivors of childhood sexual abuse (Stein et al., 1997). Although these investigators attempted to control for lifetime alcohol consumption, these studies may not have controlled for adolescent onset alcohol abuse. Results from animal studies show that the hippocampus is susceptible to the effects of chronic

alcohol administration (Lescaudron, Jaffard, & Verna, 1989). The additional negative impact of excessive alcohol consumption on the regulation of NMDA receptors (Breese, Freedman, & Leonard, 1995) in persons with trauma history may lead to more profound excitotoxic neuronal damage in alcoholic adults comorbid for PTSD.

Furthermore, maltreated children are at increased risk for adolescent alcohol and substance use disorders (Clark, Lesnick, & Hegedus, 1997; Dembo, Williams, Wothke, Schmeidler, & Brown, 1992; Deykin & Buka, 1997). In community adult samples, sexual abuse histories have been found to be associated with increased alcohol consumption and related problems (Widom, Ireland, & Glynn, 1995; Wilsnack, Vogeltanz, Klassen, & Harris, 1997). Childhood abuse has been found to moderate the relationship between parental alcohol abuse or dependence and young adulthood offspring alcohol abuse or dependence (Sher, Gershuny, Peterson, & Raskin, 1997). Dysregulation of biological stress systems and self-medication of chronic PTSD symptoms may constitute the mechanisms for the association between maltreatment and alcohol and substance abuse and dependence disorders. Maltreatment experiences in childhood and adolescence may increase the likelihood of alcoholism and substance abuse through attempts to use alcohol and other drugs to reduce symptoms of PTSD and its common comorbid symptoms of depression (Labouvie, 1986; Newcombe & Harlowe, 1986) and to dampen the effects of dysregulated biological stress systems (Higley, Hasert, Suomi, & Linnoila, 1991). On the other hand, adolescent onset alcohol and substance abuse and dependence may cause further dysregulation of biological stress systems (Groote Veldman & Meinders, 1996; Rivier, 1996). (See Figure 1.)

Adolescent onset alcohol abuse and dependence may be neurotoxic to the hippocampus. MRI measures of hippocampal volumes were significantly smaller in 12 subjects with adolescent onset alcohol abuse or dependence compared with 24 sociodemographically matched controls (De Bellis, Clark, et al., 2000). These findings were not explained by comorbid PTSD in the adolescent onset alcohol group. Global brain regions did not differ between groups. Adolescent alcohol abuse and dependence may be major confounds when studying adults who were maltreated as children. *These issues need to be addressed in future research designs involving maltreatment studies.*

Cognitive Functioning in Maltreated Children and Adolescents

Adult studies reported cognitive changes in individuals with PTSD (Wolfe & Charney, 1991), particularly concentration, learning, and memory problems (Boulanger, 1985; Sutker, Winstead, Galina, & Allain, 1990, 1991). One study suggested that premorbid lower IQ may increase the risk for combat-related PTSD (Macklin, Metzger, Litz, McNally, Lasko, Orr, & Pitman, 1998), but these subjects were not screened for child abuse history. Child abuse history is a risk factor for later onset PTSD in combat veterans (Bremner, Southwick, et al., 1993).

PTSD symptoms associated with child maltreatment experiences may have broad developmental ramifications. While most studies report temporal stability of intelligence in various pediatric populations including handicapped children (Atkinson, Bowman, Dickens, Blackwell, Vasarhelyi, Szep, Dunleavy, MacIntyre, & Bury, 1990; Elliot & Boeve, 1987), a literature review suggests that intellectual ability, as reflected by IQ score, may be a consequence of child maltreatment. A variety of intellectual and academic impairments, with resultant poor school performance (National Research Council, 1993; Trickett, McBride–Chang, & Putnam, 1994), have been consistently reported in abused children not evaluated for PTSD (Augoustinos, 1987; Azar, Barnes, & Twentyman, 1988; Kolko, 1992). Carrey, Butter, Persinger, and Bialik (1995) noted negative correlation between Verbal IQ score and severity of abuse. Perez and Widom (1994) reported lower IQ and reading ability in a large sample of adult survivors of child maltreatment who were followed in a long-term, well-controlled prospective study of early onset abuse or neglect (before age 11 years). Investigations re-

ported changes in IQ in high-risk samples that are related to the quantity of parent–child interaction and home environment and to the degree of maternal depression (Money, Annecillo, & Kelly, 1983; Pianta, Egeland, & Erickson, 1989). In one case control study (Money et al., 1983), low and persistent impairment of IQ were associated with abuse disclosure, while IQ elevations were significantly correlated with duration of "rescue" (in years) from an abusive upbringing. There is a positive relationship in adults between IQ and brain size (Andreasen, Flaum, Swayze, O'Leary, Alliger, Cohen, Ehrhardt, & Yuh, 1993). In our MRI study, the known positive correlations between IQ scales and intracranial volume were seen for Verbal ($r = .24$; $p = .01$), Performance ($r = .25$; $p < .01$), and Full Scale ($r = .29$; $p < .003$) IQ. However, Verbal ($r = -.36$; $p < .0001$), Performance ($r = -.42$; $p < .0001$), and Full Scale ($r = -.43$; $p < .0001$) IQ showed negative correlations with abuse duration (in years) that led to PTSD (De Bellis, Keshavan, et al., 1999). These findings lead us to hypothesize that these smaller intracranial volumes may be associated with permanent neuronal loss leading to lower IQ. To date, cognitive function, as indexed by performance on standardized neuropsychological instruments, has not been evaluated in maltreated children with and without PTSD. *Given that maltreated children tend to do poorly in school and our current lack of understanding of cognitive development in maltreated children, this is another extremely important area for future research.*

Is There a Psychobiology of Hope?

In this review, data show that the effects of traumatic stress on the developing brain may be severe and persistent and may lead to adverse brain development. However, neurobiological development is regulated by the complex interactions of genes and experiences. Quality of childcare is associated with a buffering of HPA axis to stress (Gunnar, 1998). This buffering should lead to fewer psychosocial impairments. Neuronal loss may not be permanent. There is a capacity for primate neurogenesis in the hippocampus and frontal cortex (Gould, Reeves, Graziano, & Gross, 1999; Gould, Tanapat, McEwen, Flugge, & Fuchs, 1998). Environmental stress and adrenal steroids inhibit this neurogenesis (Gould, McEwen, et al., 1997; Gould, Tanapat, & Cameron, 1997; Tanapat et al., 1998). When rescued from extremely neglectful and abusive environments, some profoundly maltreated children are capable of accelerated rates of catch-up growth, including remission of severe psychopathology and normalization of cognitive function (Koluchova, 1972, 1976; Money et al., 1983). *Brain maturation studies in maltreated children will help the field begin to understand these processes in humans. Yet, despite the clinical and recent neurobiological data showing that there is hope for maltreated individuals, clinical intervention research for maltreated children and their families is markedly underfunded.*

A Psychobiological Model for the Intergenerational Cycle of Maltreatment

Some of the factors that contribute to the difficulty in understanding the etiology of child maltreatment are (a) the relatively low prevalence of child abuse and neglect, (b) the extreme socially deviant nature of these parenting behaviors, (c) the presence of other confounding factors (i.e., poverty and family violence), and (d) the changing political and historical definitions of child abuse and neglect (for review see National Research Council, 1993).

There is an unequivocal relation between parental poverty and child maltreatment (Garbarino, 1982; Pelton, 1981; Russell & Trainor, 1984). While poverty may expose parents to more stressors and risk factors for child abuse and neglect, it is clear that most poor parents do not maltreat their children (Besharov & Laumann, 1997). A ecological–transactional model suggests that maltreatment exists when multiple risk factors outweigh protective, compensatory, and buffering factors (Cicchetti & Lynch, 1993; Cicchetti & Rizley, 1981). Approximately one quarter to one third of maltreated children grow up to repeat the cycle of abuse and neglect (Kaufman & Zigler, 1987; Widom, 1989). How-

ever, the majority of parents involved in a child maltreatment disclosure had a personal history of abuse or neglect (Kaufman & Zigler, 1987).

It is proposed that child abuse and neglect and the associated factors of substance abuse and family violence may be the result of "untreated" psychopathological (signs and symptoms of PTSD) and developmental consequences of growing up with traumatic stress. It is further proposed that these negative developmental consequences are mediated by PTSD symptoms and its trajectory to serious and comorbid adult mental disorders (see Figure 1). Recent studies have found increased rates of major depression, PTSD substance abuse–dependence, and antisocial behaviors in parents of maltreated children recruited from child protective service samples (Famularo, Kinscherff, & Fenton, 1992; Kaplan, Pelkovit, Saltzinger, & Ganeles, 1983; Taylor, Norman, Murphy, Jellinek, Quinn, Poitrast, & Groshko, 1991) and from epidemiological samples (Bland & Orn, 1986; Dinwiddie & Bucholz, 1993; Egami, Ford, Greenfield, & Crum, 1996). Furthermore, maltreatment in families involves acts of commission (the abuse or neglect) and omission (not protecting the child from abuse and neglect). Certain symptoms of untreated mood disorders (hopelessness, low self-worth) make these individuals more likely to commit these acts of omission, which put their child at risk for abuse and neglect. Other symptoms of untreated mood disorders (poor concentration, anhedonia) make learning effective parenting skills and enjoying one's child a difficult task. Mood, posttraumatic stress, substance abuse, and personality disorders are treatable illnesses. Persons with these disorders require long-term clinical attention to alleviate their suffering and to become "good enough" parents. However, one must note that some serious mental disorders are less likely to be amendable to conventional community outpatient or inpatient clinical interventions (i.e., schizophrenia, severe substance abuse–dependence, antisocial personality disorder, and violent and criminal behaviors) and that even adequately treated individuals with these disorders may never be able to adequately care for a child. *Given that maltreating parents are likely to have serious but treatable comorbid mental disorders and our current lack of understanding of their treatment needs and mental health resources for these individuals, this is another extremely important area for future prevention and intervention research.*

Can a Psychobiological Model for the Intergenerational Cycle of Maltreatment Inform the Nation About Maltreatment Policy?

This model described in Figure 1 suggests several mechanisms for therapeutic intervention to break the intergenerational cycle of abuse and neglect. However, research on childhood maltreatment is often viewed as addressing social rather than scientific public health problems. Given how common PTSD is in maltreated children, it is sad that there is no U.S. national policy for mental health screening of all parents and children involved with child protection services. Furthermore, the assessment and management of parents involved in maltreatment and their maltreated children will, more often than not, require clinical skills in interviewing persons with PTSD symptoms. Consequently, licensed mental health professionals from multidisciplinary fields have much to offer our nation's child protective services. Public Law 104-235, the Child Abuse Prevention and Treatment Act Amendment of 1996, established federal guidelines to execute and coordinate various functions and activities aimed at the prevention and treatment of child abuse and neglect. Public Law 105-89, the Adoption and Safe Families Act of 1997, clarified federal guidelines for the establishment of reasonable efforts to maintain a maltreated child in his and her home and for a child's reunification with his and her family of origin. This law also established safety requirements for foster care and adoptive placements and guidelines for the termination of parental rights. However, these laws do not comprehensively address the complexities of the mental health issues involved in child maltreatment. Since an essential way to secure secondary prevention of maltreatment and to break the intergenera-

tional cycles of abuse and neglect in already existing maltreating families is to identify and treat PTSD and related comorbid psychiatric diagnoses of caregivers involved in maltreatment and their maltreated children, express language concerning the mental health needs of maltreating families would greatly strengthen the existing laws and should help decrease future incidences of child maltreatment. *Regarding child abuse and neglect from a public health perspective rather than a social problem would open up opportunities for treatment research which would rationally inform these important life decisions and is an essential way to break the intergeneration cycle of abuse and neglect.*

Summary

Child maltreatment has a traumatic impact on biological development and is a negative life altering experience for children. Data were reviewed that support that the psychobiological sequelae of child maltreatment may be regarded as *an environmentally induced complex developmental disorder*. In this review, a developmental traumatology model of child maltreatment and the risk for the intergenerational cycle of abuse and neglect using a mental health or posttraumatic stress model was described. Data to support this view and descriptions of the state of the psychobiology of maltreatment research, emphasizing the similarities and differences between children, adolescents, and adults, were reviewed. Many suggestions for important future research opportunities as well as public policy ideas were offered. These questions should be of interest to basic scientists, clinical and policy researchers, and therapists who have long struggled with this difficult area. Although trauma in childhood may have profound and long-lasting impacts on development, it is always helpful to note that individual children strive toward growth and that there is a psychobiology of hope.

References

Alexander, G. E., & Goldman, P. S. (1978). Functional development of the dorsolateral prefrontal cortex: An analysis utilizing reversible cryogenic depression. *Brain Research, 143,* 233–249.

American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.

Andreasen, N. C., Flaum, M., Swayze, V., II, O'Leary, D. S., Alliger, R., Cohen, G., Ehrhardt, J., & Yuh, W. T. C. (1993). Intelligence and brain structures in normal individuals. *American Journal of Psychiatry, 150,* 130–134.

Armanini, M. P., Hutchins, C., Stein, B. A., & Sapolsky, R. M. (1990). Glucocorticoid endangerment of hippocampal neurons is NMDA-receptor dependent. *Brain Research, 532,* 7–12.

Armsworth, M. W., & Holaday, M. (1993). The effects of psychological trauma on children and adolescents. *Journal of Counseling and Development, 72,* 49–56.

Arnsten, A. F. T. (1998). The biology of being frazzled. *Science,* 1711–1712.

Arnsten, A. F. T., & Goldman–Rakic, P. S. (1998). Noise stress impairs cortical function: Evidence for a hyperdopaminergic mechanism. *Archives of General Psychiatry, 55,* 362–368.

Atkinson, L., Bowman, T. G., Dickens, S., Blackwell, J., Vasarhelyi, J., Szep, P., Dunleavy, B., MacIntyre, R., & Bury, A. (1990). Stability of Wechsler Adult Intelligence Scale—Revised factor scores across time. *Psychological Assessment, 2,* 447–450.

Augoustinos, M. (1987). Developmental effects of child abuse: A number of recent findings. *Child Abuse & Neglect, 11,* 15–27.

Azar, S. T., Barnes, K. T., & Twentyman, C. T. (1988). Developmental outcomes in abused children: Consequences of parental abuse or a more general breakdown in caregiver behavior? *Behavior Therapist, 11,* 27–32.

Baker, D. G., West, S. A., Nicholson, W. E., Ekhator, N. N., Kasckow, J. W., Hill, K. K., Bruce, A. B., Orth, D. N., & Geracioti, T. D., Jr. (1999). Serial CSF corticotropin-releasing hormone levels and adrenocortical activity in combat veterans with posttraumatic stress disorder. *American Journal of Psychiatry, 156,* 585–588.

Benkelfat, C. (1993). Serotonergic mechanisms in psychiatric disorders: New research tools, new ideas. *International Clinical Psychopharmacology, 8*(Suppl. 2), 53–56.

Besharov, D. J., & Laumann, L. A. (1997). Don't call it child abuse if it's really poverty. *Journal of Children and Poverty, 3,* 5–36.

Black, I. B. (1998). Genes, brain, and mind: The evolution of cognition. *Neuron, 20,* 1073–1080.

Bland, R., & Orn, H. (1986). Family violence and psychiatric disorder. *Canadian Journal of Psychiatry, 31,* 129–137.

Blank, A. S. (1993). The longitudinal course of posttraumatic stress disorder. In J. R. T. Davidson & E. B. Foa (Eds.), *Posttraumatic stress disorder: DSM-IV and beyond* (pp. 3–22). Washington, DC: American Psychiatric Press.

Boulanger, G. (Ed.). (1985). *Post-traumatic stress disorder: An old problem with a new name*. Washington, DC: American Psychiatric Press.

Brady, K., Pearlstein, T., Asnis, G. M., Baker, D., Rothbaum, B., Sikes, C. R., & Farfel, G. M. (2000). Efficacy and safety of sertraline treatment of posttraumatic stress disorder: A randomized controlled trial. *Journal of the American Medical Association, 283,* 1837–1844.

Breese, C. R., Freedman, R., & Leonard, S. S. (1995). Glutamate receptor subtype expression in human postmortem brain tissue from schizophrenics and alcohol abusers. *Brain Research, 674,* 82–90.

Bremner, J. D., Davis, M., Southwick, S. M., Krystal, J. H., & Charney, D. S. (Eds.). (1993). *Neurobiology of posttraumatic stress disorder*. Washington, DC: American Psychiatric Press.

Bremner, J. D., Licinio, J., Darnell, A., Krystal, J. H., Owens, M. J., Southwick, S. M., Nemeroff, C. B., & Charney, D. S. (1997). Elevated CSF corticotropin-releasing factor concentrations in posttraumatic stress disorder. *American Journal of Psychiatry, 154,* 624–629.

Bremner, J. D., Narayan, M., Staib, L., Southwick, S. M., McGlashan, T., & Charney, D. S. (1999). Neural correlates of memories of childhood sexual abuse in women with and without posttraumatic stress disorder. *American Journal of Psychiatry, 156,* 1787–1795.

Bremner, J. D., Randall, P., Scott, T. M., Bronen, R. A., Southwick, S. M., Seibyl, J. P., Delaney, R. C., McCarthy, G., Charney, D. S., & Innis, R. B. (1995). MRI-based measurement of hippocampal volume in patients with combat-related posttraumatic stress disorder. *American Journal of Psychiatry, 152,* 973–981.

Bremner, J. D., Southwick, S. M., Johnson, D. R., Yehuda, R., & Charney, D. S. (1993). Childhood physical abuse and combat-related posttraumatic stress disorder in Vietnam veterans. *American Journal of Psychiatry, 150,* 235–239.

Bremner, J. D., Staib, L., Kaloupek, D., Southwick, S. M., Soufer, R., & Charney, D. S. (1999). Neural correlates of exposure to traumatic pictures and sound in Vietnam combat veterans with and without posttraumatic stress disorder: A positron emission tomography study. *Biological Psychiatry, 45,* 806–816.

Breslau, N., Davis, G. C., Peterson, E., & Schultz, L. R. (2000). A second look at comorbidity in victims of trauma: The posttraumatic stress disorder-major depression connection. *Biological Psychiatry, 48,* 902–909.

Caldji, C., Tannenbaum, B., Sharma, S., Francis, D., Plotsky, P. M., & Meaney, M. J. (1998). Maternal care during infancy regulates the development of neural systems mediating the expression of fearfulness in the rat. *Proceedings of the National Academy of Sciences of the United States of America, 95,* 5335–5340.

Carrey, N. J., Butter, H. J., Persinger, M. A., & Bialik, R. J. (1995). Physiological and cognitive correlates of child abuse. *Journal of the American Academy of Child & Adolescent Psychiatry, 34,* 1067–1075.

Center for Substance Abuse Treatment. (1997). *Substance abuse treatment and domestic violence* (Vol. 25). Washington, DC: Government Printing Office.

Chapin, H. D. (1917). Systematized boarding out vs. institutional care for infants and young children. *New York Medical Journal, 22,* 1009–1011.

Charney, D. S., Deutch, A. Y., Krystal, J. H., Southwich, S. M., & Davis, M. (1993). Psychobiological mechanisms of posttraumatic stress disorder. *Archives of General Psychiatry, 50,* 294–305.

Chrousos, G. P., & Gold, P. W. (1992). The concepts of stress and stress system disorders: Overview of physical and behavioral homeostasis. *Journal of the American Medical Association, 267,* 1244–1252.

Cicchetti, D., & Barnett, D. (1991). Toward the development of a scientific nosology of child maltreatment. In D. Cicchetti & W. Grove (Eds.), *Thinking clearly about psychology: Essays in honor of Paul E. Meehl* (Vol. 2, pp. 346–377). Minneapolis, MN: University of Minnesota Press.

Cicchetti, D., & Lynch, M. (1993). Toward an ecological/transactional model of community violence and child maltreatment: Consequences for children's development. *Psychiatry, 56,* 96–118.

Cicchetti, D., & Lynch, M. (1995). Failures in the expectable environment and their impact on individual development: The case of child maltreatment. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental Psychopathology* (Vol. 2, pp. 32–71). New York: Wiley.

Cicchetti, D., & Rizley, R. (1981). Developmental perspectives on the etiology, intergenerational transmission and sequelae of child maltreatment. *New Directions for Child Development, 11,* 31–55.

Clark, D. B., Lesnick, L., & Hegedus, A. (1997). Trauma and other stressors in adolescent alcohol dependence and abuse. *Journal of the American Academy of Child & Adolescent Psychiatry, 36,* 1744–1751.

Davidson, J. R. T., & Fairbank, J. A. (1993). The epidemiology of posttraumatic stress disorder. In J. R. T. Davidson & E. B. Foa (Eds.), *Posttraumatic stress disorder: DSM-IV and beyond* (pp. 147–169). Washington, DC: American Psychiatric Press.

Davidson, S., & Smith, R. (1990). Traumatic experiences in psychiatric outpatients. *Journal of Traumatic Stress Studies, 3,* 459–475.

Davis, M. (1997). Neurobiology of fear responses: The role of the amygdala. *Journal of Neuropsychiatry & Clinical Neurosciences, 9,* 382–402.

De Bellis, M. D. (1997). Posttraumatic stress disorder and acute stress disorder. In R. T. Ammerman & M. Hersen (Eds.), *Handbook of prevention and treatment with children and adolescents* (pp. 455–494). New York: Wiley.

De Bellis, M. D., Baum, A., Birmaher, B., Keshavan, M., Eccard, C. H., Boring, A. M., Jenkins, F. J., & Ryan, N. D. (1999). A. E. Bennett Research Award. Developmental traumatology part I: Biological stress systems. *Biological Psychiatry, 45,* 1259–1270.

De Bellis, M. D., Casey, B. J., Dahl, R., Birmaher, B., Williamson, D., Thomas, K. M., Axelson, D. A., Frustaci, K., Boring, A. M., Hall, J., & Ryan, N. (2000). A pilot study of amygdala volumes in pediatric generalized anxiety disorder. *Biological Psychiatry, 48,* 51–57.

De Bellis, M. D., Chrousos, G. P., Dorn, L. D., Burke, L., Helmers, K., Kling, M. A., Trickett, P. K., & Putnam, F. W. (1994). Hypothalamic–pituitary–adrenal axis dysregulation in sexually abused girls. *Journal of Clinical Endocrinology and Metabolism, 78,* 249–255.

De Bellis, M. D., Clark, D. B., Beers, S. R., Soloff, P., Boring, A. M., Hall, J., Kersh, A., & Keshavan, M. S. (2000). Hippocampal volume in adolescent onset alcohol use disorders. *American Journal of Psychiatry, 157,* 737–744.

De Bellis, M. D., Keshavan, M., Clark, D. B., Casey, B. J., Giedd, J., Boring, A. M., Frustaci, K., & Ryan, N. D. (1999). A. E. Bennett Research Award. Developmental traumatology part II: Brain development. *Biological Psychiatry, 45,* 1271–1284.

De Bellis, M. D., Keshavan, M. S., Spencer, S., & Hall, J. (2000). *N*-acetylaspartate concentration in the anterior cingulate in maltreated children and adolescents with PTSD. *American Journal of Psychiatry, 157,* 1175–1177.

De Bellis, M. D., Lefter, L., Trickett, P. K., & Putnam, F. W. (1994). Urinary catecholamine excretion in sexually abused girls. *Journal of the American Academy of Child & Adolescent Psychiatry, 33,* 320–327.

De Bellis, M. D., & Putnam, F. W. (1994). The psychobiology of childhood maltreatment. *In Child and Adolescent Psychiatric Clinics of North America, 3,* 663–677.

De Kloet, R., Vreugdenhil, E., Oitzl, M. S., & Joes, A. (1998). Brain corticosteroid receptor balance in health and disease. *Endocrine Reviews, 19,* 269–301.

Dembo, R., Williams, L., Wothke, W., Schmeidler, J., & Brown, C. H. (1992). The role of family factors, physical abuse and sexual victimization experiences in high-risk youths' alcohol and other drug use and delinquency: A longitudinal model. *Violence and Victims, 7,* 245–266.

Deykin, E. Y., & Buka, S. L. (1997). Prevalence and risk factors for posttraumatic stress disorder among chemically dependent adolescents. *American Journal of Psychiatry, 154,* 752–757.

Dinwiddie, S., & Bucholz, K. (1993). Psychiatric diagnosis of self reported child abusers. *Child Abuse & Neglect, 17,* 465–476.

Dubner, A. E., & Motta, R. W. (1999). Sexually and physically abused foster care children and posttraumatic stress disorder. *Journal of Consulting & Clinical Psychology, 67,* 367–373.

Dunlop, S. A., Archer, M. A., Quinlivan, J. A., Beazley, L. D., & Newnham, J. P. (1997). Repeated prenatal corticosteroids delay myelination in the ovine central nervous system. *Journal of Maternal–Fetal Medicine, 6,* 309–313.

Edwards, E., Harkins, K., Wright, G., & Menn, F. (1990). Effects of bilateral adrenalectomy on the induction of learned helplessness. *Behavioral Neuropsychopharmacology, 3,* 109–114.

Egami, Y., Ford, D., Greenfield, S., & Crum, R. (1996). Psychiatric profile and sociodemographic characteristics of adults who report physically abusing or neglecting their children. *American Journal of Psychiatry, 153,* 921–925.

Elliot, S. N., & Boeve, K. (1987). Stability of WISC-R IQs: An investigation of ethnic differences over time. *Educational and Psychological Measurement, 47,* 461–465.

Famularo, R., Fenton, T., Augustyn, M., & Zuckerman, B. (1996). Persistence of pediatric post traumatic stress disorder after 2 years. *Child Abuse & Neglect, 20,* 1245–1248.

Famularo, R., Fenton, T., & Kinscherff. (1994). Maternal and child posttraumatic stress disorder in cases of maltreatment. *Child Abuse & Neglect, 18,* 27–36.

Famularo, R., Kinscherff, R., & Fenton, T. (1992). Psychiatric diagnoses of abusive mothers. *Journal of Nervous and Mental Disease, 180,* 658–661.

Famularo, R., Kinsherff, R., & Fenton, T. (1988). Propranolol treatment for childhood posttraumatic stress disorder, acute type. *American Journal of the Diseases of Children, 142,* 1244–1247.

Felitti, V. J., Anda, R. F., Nordenberg, D., Williamson, D. F., Spitz, A. M., Edwards, V., Koss, M. P., & Marks, J. S. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes if death in adults. *American Journal of Preventive Medicine, 14,* 245–258.

Fletcher, K. E. (1996). Childhood posttraumatic stress disorder. In E. J. Mash & R. A. Barkley (Eds.), *Child psychopathology* (pp. 242–276). New York: Guilford Press.

Francis, D. D., & Meaney, M. J. (1999). Maternal care and the development of stress responses. *Current Opinion in Neurobiology, 9,* 128–134.

Fuster, J. M. (1980). *The prefontal cortex: Anatomy, physiology, and neuropsychology of the frontal lobe*. New York: Raven Press.

Garbarino, J. (1982). *Children and families in the social environment*. New York: Aldine.

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Castellanos, X., Liu, H., Zijdenbos, A., Paus, T., Evans, A. C., & Rapoport, J. L. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience, 2,* 861–863.

Giedd, J. N., Blumenthal, J., Jeffries, N. O., Rajapakse, J. C., Vaituzis, A. C., Liu, H., Tobin, M., Nelson, J., & Castellanos, X. (1999). Development of the human corpus callosum during childhood and adolescence: A longitudinal MRI study. *Progress in NeuroPsychopharmacology and Biological Psychiatry, 23,* 571–588.

Giedd, J. N., Castellanos, F. X., Casey, B. J., Kozuch, B. A., King, A. C., Hamburger, S. D., & Rapoport, J. L. (1994). Quantitative morphology of the corpus callosum in attention deficit hyperactivity disorder. *American Journal of Psychiatry, 151,* 665–669.

Goenjian, A. K., Yehuda, R., Pynoos, R. S., Steinberg, A. M., Tashjian, M., Yang, R. K., Najarian, L. M., & Fairbanks, L. A. (1996). Basal cortisol, dexamethasone suppression of cortisol, and MHPG in adolescents after the 1988 earthquake in Armenia. *American Journal of Psychiatry, 153,* 929–934.

Goldman, P. S., & Rosvold, H. E. (1970). Localization of function within the dorsolateral prefrontal cortex of the rhesus monkey. *Experimental Neurology, 27,* 291–304.

Goldman–Rakic, P. S. (1994). Working memory dysfunction in schizophrenia. *Journal of Neuropsychiatry and Clinical Neurosciences, 6,* 348–357.

Goldstein, G., Van Kammen, W., Shelly, C., Miller, D. J., & Van Kammen, D. P. (1987). Survivors of imprisonment in the Pacific theater during World War II. *American Journal of Psychiatry, 144,* 1210–1213.

Goodwin, F. K., & Jamison, K. R. (1990). Manic–depressive illness (pp. 416–447). New York: Oxford University Press.

Gould, E., McEwen, B. S., Tanapat, P., Galea, L. A., & Fuchs, E. (1997). Neurogenesis in the dentate gyrus of the adult tree shrew is regulated by psychosocial stress and NMDA receptor activation. *Journal of Neuroscience, 17,* 2492–2498.

Gould, E., Reeves, A. J., Graziano, M. S. A., & Gross,

C. G. (1999). Neurogenesis in the neocortex of adult primates. *Science, 286,* 548–552.

Gould, E., Tanapat, P., & Cameron, H. A. (1997). Adrenal steroids suppress granule cell death in the developing dentate gyrus through an NMDA receptor-dependent mechanism. *Brain Research and Developmental Brain Research, 103,* 91–93.

Gould, E., Tanapat, P., McEwen, B. S., Flugge, G., & Fuchs, E. (1998). Proliferation of granule cell precursors in the dentate gyrus of adult monkeys is diminished by stress. *Proceedings of the National Academy of Sciences of the United States of America, 95,* 3168–3171.

Green, A. (Ed.). (1985). *Children traumatized by physical abuse*. Washington, DC: American Psychiatric Press.

Groote Veldman, R., & Meinders, A. E. (1996). On the mechanism of alcohol-induced pseudo-Cushing's syndrome. *Endocrine Review, 17,* 262–268.

Gunnar, M. R. (1998). Quality of early care and buffering of neuroendocrine stress reactions: Potential effects on the developing human brain. *Preventive Medicine, 27,* 208–211.

Gurvits, T. V., Shenton, M. E., Hokama, H., Ohta, H., Lasko, N. B., Gilbertson, M. W., Orr, S. P., Kikinis, R., Jolesz, F. A., McCarley, R. W., & Pitman, R. K. (1996). Magnetic resonance imaging study of hippocampal volume in chronic, combat-related posttraumatic stress disorder. *Biological Psychiatry, 40,* 1091–1099.

Hamner, M. B., Lorberbaum, J. P., & George, M. S. (1999). Potential role of the anterior cingulate cortex in PTSD: Review and hypothesis. *Depression & Anxiety, 9,* 1–14.

Hart, J., Gunnar, M., & Cicchetti, D. (1996). Altered neuroendocrine activity in maltreated children related to symptoms of depression. *Development and Psychopathology, 8,* 201–214.

Higley, J. D., Hasert, M. F., Suomi, S. J., & Linnoila, M. (1991). A nonhuman primate model of alcohol abuse: Effects of early experience, personality and stress on alcohol consumption. *Proceedings of the National Academy of Sciences, 88,* 7261–7265.

Hillary, B. E., & Schare, M. L. (1993). Sexually and physically abused adolescents: An empirical search for PTSD. *Journal of Clinical Psychology, 49,* 161–165.

Hofer, M. A. (1996). On the nature and consequences of early loss. *Psychosomatic Medicine, 58,* 570–581.

Jernigan, T. L., & Sowell, E. R. (1997). Magnetic resonance imaging studies of the developing brain. In M. S. Keshavan & R. M. Murray (Eds.), *Neurodevelopment & adult psychopathology* (pp. 63–70). Cambridge: Cambridge University Press.

Johnson, J. G., Cohen, P., Brown, J., Smailes, E. M., & Berstein, D. P. (1999). Childhood maltreatment increases risk for personality disorders during early adulthood. *Archives of General Psychiatry, 56,* 600–606.

Kaplan, S., Pelkovit, D., Saltzinger, S., & Ganeles, D. (1983). Psychopathology of parents of abused and neglected children and adolescents. *Journal of the American Academy of Child & Adolescent Psychiatry, 22,* 238–244.

Kaufman, J. (1991). Depressive disorders in maltreated children. *Journal of the American Academy of Child & Adolescent Psychiatry, 30,* 257–265.

Kaufman, J., Birmaher, B., Perel, J., Dahl, R. E., Moreci, P., Nelson, B., Wells, W., & Ryan, N. (1997). The corticotropin-releasing hormone challenge in depressed abused, depressed nonabused, and normal control children. *Biological Psychiatry, 42,* 669–679.

Kaufman, J., & Zigler, E. (1987). Do abused children become abusive parents? *American Journal of Orthopsychiatry, 57,* 186–192.

Kinzie, J. D., Sack, W., Angell, R., & Clarke, G. (1989). A three year follow-up of Cambodian young people traumatized as children. *Journal of the American Academy of Child and Adolescent Psychiatry, 28,* 501–504.

Kluznik, J. C., Speed, N., Van Valkenburg, C., & Magraw, R. (1986). 40-year follow-up of US prisoners of war. *American Journal of Psychiatry, 143,* 1443–1446.

Kolko, D. (1992). Characteristics of child victims of physical violence: Research findings and clinical implications. *Journal of Interpersonal Violence, 7,* 244–276.

Koluchova, J. (1972). Severe deprivation in twins: A case study. *Journal of Child Psychology and Psychiatry, 13,* 107–114.

Koluchova, J. (1976). The further development of twins after severe and prolonged deprivation: A second report. *Journal of Child Psychology and Psychiatry, 17,* 181–188.

Kuhn, C. M., Pauk, J., & Schanberg, S. M. (1990). Endocrine response to mother–infant separation in developing rats. *Developmental Psychobiology, 23,* 395–410.

Labouvie, E. (1986). Alcohol and marijuana use in relation to adolescent stress. *The International Journal of the Addictions, 21,* 333–345.

Lauder, J. M. (1988). Neurotransmitters as morphogens. *Progress in Brain Research, 73,* 365–388.

LeDoux, J. (1998). Fear and the brain: Where have we been, and where are we going? *Biological Psychiatry, 44,* 1229–1238.

Lemieux, A. M., & Coe, C. L. (1995). Abuse-related posttraumatic stress disorder: Evidence for chronic neuroendocrine activation in women. *Psychosomatic Medicine, 57,* 105–115.

Lescaudron, L., Jaffard, R., & Verna, A. (1989). Modification in the number and morphology of dendritic spines resulting from chronic ethanol consumption and withdrawal: A Golgi study in the mouse anterior and posterior hippocampus. *Experimental Neurology, 106,* 156–163.

Lipschitz, D. S., Winegar, R. K., Hartnick, E., Foote, B., & Southwick, S. M. (1999). Posttraumatic stress disorder in hospitalized adolescents: Psychiatric comorbidity and clinical correlates. *Journal of the American Academy of Child & Adolescent Psychiatry, 38,* 385–392.

Luntz, B. K., & Widom, C. S. (1994). Antisocial personality disorder in abused and neglected children grown up. *American Journal of Psychiatry, 151,* 670–674.

Lyon, T. D. (1999). Are battered women bad mothers? Rethinking the termination of abused women's parental rights for failure to protect. In H. Dubowitz (Ed.), *Neglected children: Research, practice, and policy* (pp. 237–260). Thousand Oaks, CA: Sage.

Maas, J. W., Koslow, S. H., Davis, J., Katz, M., Frazer, A., Bowden, C. L., Berman, N., Gibbons, R., Stokes, P., & Landis, D. H. (1987). Catecholamine metabolism and disposition in healthy and depressed subjects. *Archives of General Psychiatry, 44,* 337–344.

Macklin, M. L., Metzger, L. J., Litz, B. T., McNally, R. J., Lasko, N. B., Orr, S. P., & Pitman, R. K.

(1998). Lower precombat intelligence is a risk factor for posttraumatic stress disorder. *Journal of Consulting & Clinical Psychology, 66,* 232–236.

Mannarino, A. P., Cohen, J. A., & Berman, S. R. (1994). The relationship between preabuse factors and psychological symptomatology in sexually abused girls. *Child Abuse & Neglect, 18,* 63–71.

Mason, J. W., Giller, E. L., Kosten, T. R., Ostroff, R. B., & Podd, L. (1986). Urinary-free cortisol levels in posttraumatic stress disorder patients. *Journal of Nervous and Mental Disease, 174,* 145–159.

McLeer, S. V., Callaghan, M., Henry, D., & Wallen, J. (1994). Psychiatric disorders in sexually abused children. *Journal of the American Academy of Child & Adolescent Psychiatry, 33,* 313–319.

McLeer, S. V., Dixon, J. F., Henry, D., Ruggiero, K., Escovitz, K., Niedda, T., & Scholle, R. (1998). Psychopathology in non-clinically referred sexually abused children. *Journal of the American Academy of Child & Adolescent Psychiatry, 37,* 1326–1333.

Meaney, M. J., Diorio, J., Francis, D., Widdowson, J., LaPlante, P., Caldji, C., Sharma, S., Seckl, J. R., & Plotsky, P. M. (1996). Early environmental regulation of forebrain glucocorticoid receptor gene expression: implications for adrenocortical responses to stress. *Developmental Neuroscience, 18,* 49–72.

Money, J., Annecillo, C., & Kelly, J. F. (1983). Abuse–dwarfism syndrome: After rescue, statural and intellectual catchup growth correlate. *Journal of Clinical Child Psychology, 12,* 279–283.

National Research Council. (1993). *Understanding child abuse and neglect*. Washington, DC: National Academy Press.

Newcombe, M. D., & Harlowe, L. L. (1986). Life events and substance use among adolescents: Mediating effects of perceived loss of control and meaninglessness in life. *Journal of Personality and Social Psychology, 51,* 564–577.

Pelton, L. (1981). *The social context of child abuse and neglect*. New York: Human Sciences Press.

Perez, C., & Widom, C. S. (1994). Childhood victimization and long-term intellectual and academic outcomes. *Child Abuse & Neglect, 18,* 617–633.

Perry, B. D. (Ed.). (1994). *Neurobiological sequelae of childhood trauma: PTSD in children*. Washington, DC: American Psychiatric Press.

Pianta, R., Egeland, B., & Erickson, M. F. (1989). Results of the mother–child interaction research project. In D. Cicchetti & V. Carlson (Eds.), *Child maltreatment: Theory and research on the causes and consequences of child abuse and neglect* (pp. 203–253). Cambridge, MA: Harvard University Press.

Pine, D. S., Cohen, P., & Brook, J. (1996). Emotional problems during youth as predictors of stature during early adulthood: Results from a prospective epidemiologic study. *Pediatrics, 97,* 856–863.

Pittman, P. K., & Orr, S. P. (1990). Twenty-four hour cortisol and catecholamine excretion in combat-related posttraumatic stress disorder. *Biological Psychiatry, 27,* 245–247.

Posner, M. I., & Petersen, S. E. (1990). The attention system of the human brain. *Annual Review of Neuroscience, 13,* 25–42.

Prichard, J. W. (1996). MRS of the brain-prospects for clinical application. In I. R. Young & H. C. Charles (Eds.), *MR spectroscopy: Clinical applications and techniques* (pp. 1–25). London: Livery House.

Putnam, F. W. (1997). *Dissociation in children and adolescents: A developmental perspective*. New York: Guilford Press.

Putnam, F. W., Trickett, P. K., Helmers, K., Dorn, L., & Everett, B. (1991). Cortisol abnormalities in sexually abused girls. Paper presented at the 144th Annual Meeting, Washington, DC.

Pynoos, R. S., & Eth, S. (1985). Witnessing acts of personal violence. In S. Eth & R. S. Pynoos (Eds.), *Posttraumatic stress in children* (pp. 17–43). Washington, DC: American Psychiatric Press.

Pynoos, R. S., Frederick, C., Nader, K., Arroyo, W., Steinberg, A., Eth, S., Nunez, F., & Fairbanks, L. (1987). Life threat and post-traumatic stress in school-aged children. *Archives of General Psychiatry, 44,* 1057–1063.

Pynoos, R. S., & Nader, K. (1989). Children's memory and proximity to violence. *Journal of the American Academy of Child & Adolescent Psychiatry, 28,* 236–241.

Pynoos, R. S., & Nader, K. (1990). Mental health disturbances in children exposed to disaster: Prevention intervention strategies. In S. Goldston, J. Yager, C. Heinicke, & R. S. Pynoos (Eds.), *Preventing mental health disturbances in childhood* (pp. 211–233). Washington, DC: American Psychiatric Press.

Pynoos, R. S., & Nader, K. (1993). Issues in the treatment of posttraumatic stress disorder in children and adolescents. In J. Wilson & B. Raphael (Eds.), *International handbook of traumatic stress syndromes* (pp. 535–549). Washington, DC: American Psychiatric Press.

Pynoos, R. S., Steinberg, A. M., & Wraith, R. (1995). A developmental model of childhood traumatic stress. In D. Cicchetti & D. J. Cohen (Eds.), *Developmental psychopathology* (Vol. 2, pp. 72–95). New York: Wiley.

Queiroz, E. A., Lombardi, A. B., Santos Furtado, C. R. H., Peixoto, C. C. D., Soares, T. A., Fabre, Z. L., Basques, J. C., Fernandes, M. L. M., & Lippi, J. R. S. (1991). Biochemical correlate of depression in children. *Arquivos de Neuro-Psiquiatra, 49,* 418–425.

Ramaekers, G., & Njiokiktjien, C. (1991). The child's corpus callosum. *Pediatric behavioral neurology* (Vol. 3). Amsterdam: Suyi Publications.

Rivier, C. (1996). Alcohol stimulates ACTH secretion in the rat: Mechanisms of action and interactions with other stimuli. *Alcoholism: Clinical and Experimental Research, 20,* 240–254.

Rosenberg, D. R., & Keshavan, M. S. (1998). A. E. Bennett Research Award. Toward a neurodevelopmental model of obsessive–compulsive disorder. *Biological Psychiatry, 43,* 623–640.

Rothbaum, B. O., & Foa, E. B. (Eds.). (1993). *Subtypes of posttraumatic stress disorder and duration of symptoms*. Washington, DC: American Psychiatric Press.

Russell, A. B., & Trainor, C. M. (1984). *Trends in child abuse and neglect: A national perspective*. Denver, CO: American Association for Protecting Children, American Humane Association.

Sapolsky, R. M., Uno, H., Rebert, C. S., & Finch, C. E. (1990). Hippocampal damage associated with prolonged glucocorticoid exposure in primates. *Journal of Neuroscience, 10,* 2897–2902.

Sassetti, M. R. (1993). Domestic violence. *Primary Care, 20,* 289–305.

Scheeringa, M. S., Zeanah, C. H., Drell, M. J., & Larrieu, J. A. (1995). Two approaches to the diagnosis of post-

traumatic stress disorder in infancy and early childhood. *Journal of the American Academy of Child & Adolescent Psychiatry, 34,* 191–200.

Sher, K. J., Gershuny, B. S., Peterson, L., & Raskin, G. (1997). The role of childhood stressors in the intergenerational transmission of alcohol use disorders. *Journal of the Study of Alcohol, 58,* 414–427.

Shin, L. M., McNally, R. J., Kosslyn, S. M., Thompson, W. L., Rauch, S. L., Alpert, N. M., Metzger, L. J., Lasko, N. B., Orr, S. P., & Pitman, R. K. (1999). Regional cerebral blood flow during script-imagery in childhood sexual abuse-related PTSD: A PET investigation. *American Journal of Psychiatry, 156,* 575–584.

Siever, L., & Trestman, R. L. (1993). The serotonin system and aggressive personality disorder. *International Clinical Psychopharmacology, 8*(Suppl. 2), 33–40.

Simantov, R., Blinder, E., Ratovitski, T., Tauber, M., Gabbay, M., & Porat, S. (1996). Dopamine induced apoptosis in human neuronal cells: Inhibition by nucleic acids antisense to the dopamine transporter. *Neuroscience, 74,* 39–50.

Smythies, J. R. (1997). Oxidative reactions and schizophrenia: A review–discussion. *Schizophrenia Research, 24,* 357–364.

Southwick, S. M., Krystal, J. H., Johnson, D. R., & Charney, D. S. (1992). Neurobiology of posttraumatic stress disorder. In A. Tasman & M. B. Riba (Eds.), *Review of psychiatry* (pp. 347–367). Washington, DC: American Psychiatric Press.

Southwick, S. M., Yehuda, R., & Morgan, C. A. (1995). Clinical studies of neurotransmitter alterations in post-traumatic stress disorder. In M. J. Friedman, D. S. Charney, & A. Y. Deutch (Eds.), *Neurobiological and clinical consequences of stress: From normal adaptation to posttraumatic stress disorder* (pp. 335–349). Philadelphia: Lippincott–Raven.

Southwick, S. S., Yehuda, R., & Wang, S. (1998). Neuroendocrine alterations in posttraumatic stress disorder. *Psychiatric Annals, 28,* 436–442.

Starkman, M. N., Gebarksi, S. S., Berent, S., & Schteingart, D. E. (1992). Hippocampal formation volume, memory dysfunction, and cortisol levels in patients with Cushing's syndrome. *Biological Psychiatry, 32,* 756–765.

Stein, M. B., Koverola, C., Hanna, C., Torchia, M. G., & McClarty, B. (1997). Hippocampal volume in women victimized by childhood sexual abuse. *Psychological Medicine, 27,* 1–9.

Steiner, H., Garcia, I. G., & Matthews, Z. (1997). Posttraumatic stress disorder in incarcerated juvenile delinquents. *Journal of the American Academy of Child & Adolescent Psychiatry, 36,* 357–365.

Sulser, F. (1987). Serotonin–norepinephrine receptor interactions in the brain: Implications for the pharmacology and pathophysiology of affective disorders. *Journal of Clinical Psychiatry, 48*(Suppl. 3), 12–18.

Sutker, P. B., Winstead, D. K., Galina, Z. H., & Allain, A. N. (1990). Trauma-induced weight loss and cognitive deficits among former POW survivors of the Korean Conflict. *Journal of Personality Assessment, 54,* 170–180.

Sutker, P. B., Winstead, D. K., Galina, Z. H., & Allain, A. N. (1991). Cognitive deficits and psychopathology among former prisoners of war and combat veterans of the Korean conflict. *American Journal of Psychiatry, 148,* 67–72.

Tanapat, P., Galea, L. A., & Gould, E. (1998). Stress inhibits the proliferation of granule cell precursors in the developing dentate gyrus. *Journal of Developmental Neuroscience, 16,* 235–239.

Taylor, C., Norman, N., Murphy, M., Jellinek, M., Quinn, D., Poitrast, F. G., & Groshko, M. (1991). Diagnosed intellectual and emotional impairment among parents who seriously mistreat their children: Prevalence, type, and outcome in a court sample. *Child Abuse & Neglect, 15,* 389–401.

Thompson, P. M., Giedd, J. N., Woods, R. P., MacDonald, D., Evans, A. C., & Toga, A. W. (2000). Growth patterns in the developing brain detected by using continuum mechanical tensor maps. *Nature, 404,* 190–193.

Todd, R. D. (1992). Neural development is regulated by classical neuro-transmitters: Dopamine D2 receptor stimulation enhances neurite outgrowth. *Biological Psychiatry, 31,* 794–807.

Trickett, P. K., & McBride–Chang, C. (1995). The developmental impact of different forms of child abuse and neglect. *Developmental Review, 15,* 311–337.

Trickett, P. K., McBride–Chang, C., & Putnam, F. W. (1994). The classroom performance and behavior of sexually abused girls. *Development and Psychopathology, 6,* 183–194.

True, W. R., Rice, J., Eisen, S. A., Heath, A. C., Goldberg, J., Lyons, M. J., & Nowak, J. (1993). A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. *Archives of General Psychiatry, 50,* 257–264.

Uno, H., Tarara, R., Else, J., Suleman, M. A., & Sapolsky, R. M. (1989). Hippocampal damage associated with prolonged and fatal stress in primates. *Journal of Neuroscience, 9,* 1705–1711.

van der Kolk, B. A., Dreyfuss, D., Michaels, M., Shera, D., Berkowitz, R., Fisler, R., & Saxe, G. (1994). Fluoxetine in posttraumatic stress disorder. *Journal of Clinical Psychiatry, 55,* 517–522.

van der Kolk, B. A., Greenberg, M. S., Orr, S. P., & Pitman, R. K. (1989). Endogenous opioids, stress induced analgesia, and posttraumatic stress disorder. *Psychopharmacology Bulletin, 25,* 417–421.

van der Kolk, B. A., Perry, J. C., & Herman, J. L. (1991). Childhood origins of self-destructive behavior. *American Journal of Psychiatry, 148,* 1665–1671.

Walker, E. A., Unutzer, J., Rutter, C., Gelfand, A., Saunders, K., VonKorff, M., Koss, M. P., & Katon, W. (1999). Costs of health care use by women HMO members with a history of childhood abuse and neglect. *Archives of General Psychiatry, 56,* 609–613.

Widom, C. S. (1989). The cycle of violence. *Science, 244,* 160–166.

Widom, C. S. (1999). Posttraumatic stress disorder in abused and neglected children grown up. *American Journal of Psychiatry, 156,* 1223–1229.

Widom, C. S., Ireland, T., & Glynn, P. J. (1995). Alcohol abuse in abused and neglected children followed-up: Are they at increased risk? *Journal of Studies on Alcohol, 56,* 207–217.

Wilsnack, S. C., Vogeltanz, N. D., Klassen, A. D., & Harris, T. R. (1997). Childhood sexual abuse and women's substance abuse: National survey findings. *Journal of the Study of Alcohol, 58,* 264–271.

Wolfe, D. A., Sas, L., & Wekerle, C. (1994). Factors associated with the development of posttraumatic stress disorder among victims of sexual abuse. *Child Abuse & Neglect, 18,* 37–50.

Wolfe, J., & Charney, D. S. (1991). Use of neuropsycho-

logical assessment in posttraumatic stress disorder. *Psychological Assessment: A Journal of Consulting and Clinical Psychology, 3,* 573–580.

Yehuda, R., Kahana, B., Binder–Brynes, K., Southwick, S., Mason, J. W., & Giller, E. L. (1995). Low urinary cortisol excretion in Holocaust survivors with posttraumatic stress disorder. *American Journal of Psychiatry, 152,* 982–986.

Yehuda, R., Levengood, R. A., Schmeidler, J., Wilson, S., Guo, L. S., & Gerber, D. (1996). Increased pituitary activation following metyrapone administration in post-traumatic stress disorder. *Psychoneuroendocrinology, 21,* 1–16.

Yehuda, R., Southwick, S., Giller, E. L., Ma, X., & Mason, J. W. (1991). Low urinary cortisol excretion in PTSD. *Journal of Nervous and Mental Disease, 178,* 366–369.

Yehuda, R., Southwick, S., Giller, E. L., Ma, X., & Mason, J. W. (1992). Urinary catecholamine excretion and severity of PTSD symptoms in Vietnam combat veterans. *Journal of Nervous and Mental Disease, 180,* 321–325.

Yeo, H. M., & Yeo, W. W. (1993). Repeated deliberate self-harm: A link with childhood sexual abuse? *Archives of Emergency Medicine, 10,* 161–166.

Zubieta, J. K., Chinitz, J. A., Lombardi, U., Fig, L. M., Cameron, O. G., & Liberzon, I. (1999). Medial frontal cortex involvement in PTSD symptoms: A SPECT study. *Journal of Psychiatric Research, 33,* 259–264.