Developmental traumatology: a contributory mechanism for alcohol and substance use disorders

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Abstract

Early childhood traumatic experiences, such as childhood maltreatment, are associated with an enhanced risk of adolescent and adult alcohol and substance use disorders (defined as DSM-IV alcohol or substance abuse or dependence). Maltreated children and adolescents manifest dysregulation of major biological stress response systems including adverse influences on brain development. Dysregulation of biological stress response systems may lead to an enhanced vulnerability for psychopathology, particularly posttraumatic stress disorder (PTSD) and depression. These negative affect disorders may put a child at increased risk for adolescent or young adult onset alcohol or substance use disorders. Thus, studies in developmental traumatology may prove to be critical in the effort to attempt to link the neurobiology of maltreatment-related PTSD with the neurobiology of alcohol and substance use disorders and in developing early strategies for the prevention of adolescent and adult alcohol and substance use disorders. © 2001 Elsevier Science Ltd. All rights reserved.

Keywords: Child maltreatment; PTSD; Catecholamines; Cortisol; Alcohol and substance abuse/dependence

1. Introduction

Early childhood traumatic experiences are associated with an enhanced risk of adolescent and adult alcohol and substance use disorders (defined as DSM-IV alcohol...
or substance abuse or dependence), and for child and adult psychiatric illness (Bremner et al., 1993; Davidson and Smith, 1990; Johnson et al., 1999; Widom et al., 1995; Widom, 1999). Maltreatment of children, defined as neglect, physical abuse, sexual abuse, and emotional abuse (which includes witnessing domestic violence), is increasing in the United States (US Department of Health and Human Services, 1999). Maltreatment in childhood may be the most common cause of interpersonal traumas and posttraumatic stress disorder (PTSD) in children and adolescents today (De Bellis, 1997). Nationally, over 3 million children were referred for investigation to Child Protective Services (CPS) in 1996 (US Department of Health and Human Services, 1998). Only 1% of these cases were identified as ‘false reports’ (US Department of Health and Human Services, 1998). There has been a disproportionate increase in the incidence of maltreatment among young children (under 12 years of age) (Sedlak and Broadhurst, 1996). Child neglect is the most prevalent form of child maltreatment, followed in order by physical abuse, sexual abuse, and emotional abuse (National Research Council, 1993). Furthermore, for the child victim, maltreatment is a chronic and stressful life condition particularly because various forms of child abuse and neglect tend to co-exist (McGee et al., 1995; Widom, 1989).

Childhood trauma has psychopathological and developmental consequences (Pynoos et al., 1995) including adverse emotional (Cicchetti and Lynch, 1995; National Research Council, 1993) behavioral (Shields et al., 1994) and cognitive consequences (Cahill et al., 1999; Perez and Widom, 1994). Developmental traumatology is the systematic investigation of the psychiatric and psychobiological impact of overwhelming and chronic interpersonal stress on the developing child. It is a relatively new area of study that synthesizes knowledge from an array of scientific fields including developmental psychopathology, developmental neuroscience, and stress and trauma research.

In this review, it is hypothesized that permanent changes occur in the major biological stress response systems of children secondary to experiencing traumatic stress. These changes may, in turn, lead to an enhanced vulnerability to psychopathology and later-onset adolescent and adult alcohol and substance use disorders. Most investigators have examined the relationship between childhood trauma and its effects on two of the body’s major stress systems, the hypothalamic–pituitary–adrenal (HPA) axis, the locus ceruleus (LC)–norepinephrine (NE)/sympathetic nervous system (SNS) (i.e. ‘the catecholamine system’) and their influence on brain maturation (De Bellis et al., 1999a,b; De Bellis and Putnam, 1994). Although this article will focus mainly on these two biological stress systems, there are multiple, densely interconnected neurobiological systems that are likely impacted by childhood maltreatment which may also contribute to later-onset alcohol and substance use disorders (De Bellis and Putnam, 1994). In this article, evidence will be reviewed supporting the idea that dysregulation of biological stress response systems and associated PTSD symptoms contribute to adolescent and adult alcohol and substance use disorders.
2. Pediatric maltreatment-related PTSD

The diagnosis of PTSD is made after a person experiences a traumatic event(s) and reacts with fear or disorganized behavior, followed by complaints of three categorical symptoms for at least one month: (1) intrusive re-experiencing of the trauma(s), (2) persistent avoidance of stimuli associated with the trauma(s) or numbing of responsiveness, and (3) persistent symptoms of increased physiological arousal (American Psychiatric Association, 2000). The diagnostic picture of PTSD in children is similar to that of adults (for review see Pynoos and Eth, 1985) with the exception of children less than 4 years old where objective criteria based on observable behaviors are warranted (Scheeringa et al., 1995). Results from a recent meta-analysis suggest that children are more likely to be diagnosed with PTSD, once traumatized, than their adult counterparts (Fletcher, 1996).

A review of the PTSD literature to date reveal that the experience of severe trauma of interpersonal origins (i.e. child abuse or neglect, sexual assault, warfare) heighten the risk for PTSD and its associated impairments in the majority of victims (for review see De Bellis, 1997). Thus, the experience of severe and overwhelming trauma may override any genetic, constitutional, social, or psychological resilience factors. Consequently, PTSD lifetime prevalence rates for trauma of interpersonal origins in all age and gender groups are high and range from approximately 30% to 50% (for review see De Bellis, 2001).

Childhood maltreatment, particularly sexual abuse, is associated with high incidence rates of acute and chronic PTSD. Childhood victims of sexual or physical abuse and neglect were found to be at increased risk for developing a lifetime history of PTSD when assessed prospectively in young adulthood (Widom, 1999). In clinically referred samples, the reported incidence rates of PTSD resulting from sexual abuse range from 42% to 90% (McLeer et al., 1994) and from witnessing domestic violence from 50% to 100% from domestic homicide (Pynoos and Nader, 1989). In non-clinically referred samples, a 39% incidence rate of PTSD was reported in abused and neglected children (Famularo et al., 1993). Recently, a 36.3% incidence rate of PTSD in non-clinically referred sexually abused children was reported (McLeer et al., 1998). To date, there are no studies that directly examine PTSD in neglected children. Although child neglect is not abuse, neglect may be perceived by the child as traumatic. An unsupervised non-abused young child is more likely to witness interpersonal traumas such as domestic and community violence or experience traumatic accidents. It is estimated that one-third to one-half of neglected children witness domestic violence (for review see Sassetti, 1993; Lyon, 1999). At least half of these children will experience PTSD symptoms.

Furthermore, a review of the longitudinal course of PTSD suggested that PTSD symptoms are common within the first 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). However, chronic PTSD symptoms as well as experiencing PTSD during childhood can be associated with pervasive psychopathology. Thus, PTSD can be conceptualized as a dimensional process rather than a categorical yes-or-no outcome. Pervasive complete and partial PTSD responses are usually seen in
many forms of trauma including victims of childhood maltreatment (Armsworth and Holaday, 1993; Famularo et al., 1994; Hillary and Schare, 1993; Mannarino et al., 1994; Wolfe et al., 1994; Wolfe and Charney, 1991).

Psychiatric symptoms of mood and anxiety disorders, particularly symptoms of PTSD are thought to be caused by dysregulation of biological stress response systems. Hence, changes in biological stress response systems associated with childhood trauma can render one vulnerable to primary psychiatric disorders and thus lead to ‘self-medicating’ with alcohol and various illicit substances. Additionally, dysregulated biological stress response systems can be associated with maturation failures in the frontal and prefrontal cortex, which may lead to failures in self-regulation and a greater incidence of impulsive behaviors (i.e. alcohol and substance abuse). The following review of the psychobiology of childhood trauma will further elucidate these ideas. See Fig. 1.

3. The psychobiology of trauma

Multiple neurotransmitter systems and neuroendocrine axes are activated during acute stress (reviewed by Charney et al., 1993). Traumatic stress may have negative effects on the development of these systems (reviewed by De Bellis and Putnam, 1994). There is little research on the neurobiological effects of trauma in developing children. Studies of the neurobiological effects of overwhelming stress in animal models, of the psychobiology of adult PTSD, and traumatized children with mood and anxiety disorders provide comparative models. Most investigators have focused on two of the body’s major stress systems, the catecholamine system and the HPA axis.

Fig. 1. The psychobiology of childhood trauma.
Animal studies examining catecholamine function show that traumatic stress activates the locus coeruleus, the major catecholamine (specifically NE) containing nucleus in the brain, and the SNS, leading to the biologic changes of the ‘fight-or-flight’ reaction. Direct and indirect effects of this activation include increases in catecholamine turnover in the brain, the SNS, and adrenal medulla which lead to increases in heart rate, blood pressure, metabolic rate, alertness, and in the circulating catecholamines (epinephrine (EPI), NE, and dopamine (DA)) (for review see Aston-Jones et al., 1994). During severe stress, the locus coeruleus stimulates the HPA axis via indirect connections through the brain’s limbic system and hypothalamic corticotropin-releasing hormone (CRH) is released. CRH activates the HPA axis and stimulates adrenocorticotropic (ACTH) and cortisol release, and centrally causes behavioral activation and intense arousal (for review see Chrousos and Gold, 1992). This activation results in animal behaviors consistent with anxiety and hypervigilance, which are the core symptoms of PTSD.

In adult PTSD, it is hypothesized that the catecholamine system and HPA axis responses to stress become maladaptive, causing long-term negative consequences (reviewed by Charney et al., 1993). Results from adult combat-related PTSD studies suggest that there is increased sensitivity of the catecholamine system that is most clearly evident under experimental conditions of stress or challenge (for review see Southwick et al., 1998). These findings include increased heart rate, systolic blood pressure, skin conductance, and other SNS responses to adrenergic or traumatic reminder challenge compared to healthy combat or non-combat controls. Although most baseline studies of single or multiple time point plasma catecholamines found no significant differences between adult PTSD and controls, elevated levels of catecholamines were found in 24-h urinary excretions in three of five studies (for review see Southwick et al., 1995). Single time point measures of catecholamines and cortisol may not provide an accurate measure of baseline functioning. These neurotransmitters have circadian influences and the stress of a single-stick venipuncture may result in elevations of cortisol and catecholamine concentration alone, obscuring any baseline differences. Thus, in adult PTSD, elevated 24-h urinary excretion of catecholamines provides evidence of an increase in baseline functioning of the catecholamine system.

Unlike the increased sensitivity to stress of the catecholamine system seen in adult PTSD, results from baseline and challenge studies of the HPA axis appear to show that this system functions in a more complicated manner (for review see Southwick et al., 1995). Thus, the discrepant findings may be a reflection of a compensatory adaptation of the HPA axis to persistently elevated levels of central CRH secondary to the traumatic experience. In adult combat-related PTSD, elevated levels of central CRH were found (Baker et al., 1999; Bremner et al., 1997). Infusion studies of metyrapone, which blocks the conversion of 11-deoxycortisol to cortisol and allows for the direct measure of pituitary release of ACTH, suggested that there is down-regulation of anterior pituitary CRH receptors presumably secondary to elevated central CRH and enhanced negative feedback inhibition of the pituitary for cortisol (Yehuda et al., 1996). Low 24-h urinary free cortisol (UFC) levels were found in one study of male and female adults with PTSD who survived the Holocaust during
childhood compared to survivors without PTSD (Yehuda et al., 1995). In two other studies, 24-h UFC concentrations were higher in male combat veterans with PTSD compared to combat veterans without PTSD (Pittman and Orr, 1990) and in women with PTSD secondary to childhood sexual abuse compared to women abused as children without PTSD and healthy non-abused controls (Lemieux and Coe, 1995). Trauma-induced ‘priming’ or a hypersensitivity of the HPA axis to later stress (Chrousos and Gold, 1992) may explain these later results.

4. The psychobiology of pediatric maltreatment-related PTSD

Since there is little research on the psychobiology of pediatric PTSD, this topic is examined more broadly. The few published studies of traumatized children who share symptoms in common with PTSD, namely symptoms of mood and anxiety disorders, will be reviewed. For example, Table 1 lists the results of studies on catecholamine and HPA function in maltreated children.

Findings of elevated baseline 24-h urinary catecholamine concentrations suggest an increase in baseline functioning of the catecholamine system in sexually abused girls, 58% of whom have histories of severely depressed mood with suicidal behavior (but only one of whom had PTSD), compared with demographically matched non-abused controls (De Bellis et al., 1994b). Levels of 24-h urinary NE were elevated in male children who suffer from severe clinical depression and have a history of parental neglect (Queiroz et al., 1991). Perry (1994) found decreased platelet adrenergic receptors and increased heart rate following orthostatic challenge in physically and sexually abused children with PTSD, suggesting an enhancement of SNS tone in childhood PTSD. An increase in baseline functioning of the catecholamine system in childhood PTSD is also provided by two separate, open-label treatment trials of the medications: clonidine (a central alpha_2-adrenergic partial agonist) and propranolol (a beta-adrenergic antagonist), both of which dampen catecholamine transmission. Clonidine treatment was associated with general clinical improvement and decreases in the arousal cluster of PTSD symptoms and basal heart rate (Perry, 1994). Propranolol treatment was associated with decreases in aggressive behaviors and insomnia (Famularo et al., 1988). Furthermore, medication-naïve, maltreated children with PTSD excreted significantly greater amounts of urinary NE and dopamine than non-abused healthy and non-traumatized anxious controls (De Bellis et al., 1999a). These biological stress measures correlated positively with duration of the PTSD trauma and symptoms of intrusive thoughts, avoidance, and hyperarousal. Consequently, the limited data published to date suggest that maltreated children suffer from depression and anxiety (particularly PTSD symptoms), but may or may not have a diagnosis of PTSD. These maltreated children evidence an enhancement of baseline SNS tone and catecholamine activity.

Studies of the HPA axis and childhood trauma also suggest that the HPA functions in a complex manner. As suggested from studies of adult PTSD, the findings seen in traumatized children also may be a reflection of a compensatory adaptation of the HPA axis to persistently elevated levels of central CRH secondary to the traumatic
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experience. Thus, seemingly discrepant results can be explained when addressed as a reflection of a chronic compensatory adaptation of the HPA axis to persistently elevated levels of central CRH long after trauma exposure. Hypersecretion of cortisol, presumably secondary to elevated central CRH, is seen during the initial trauma period. The enhanced negative feedback inhibition of the pituitary for cortisol leading to lower baseline cortisol levels may be seen as a long term consequence of trauma. This compensatory adaptation may be the result of a down-regulation of CRH receptors in the pituitary secondary to higher concentrations of central CRH. This process will result in lower ACTH plasma levels. This stress-induced change in the HPA axis may lead to ‘priming’ or a hypersensitivity of the HPA axis to later stress (Chrousos and Gold, 1992).

In studies of children with PTSD or those who are undergoing current adversity, evidence of elevations of cortisol or ACTH are seen. Augmented mean morning serial plasma cortisol levels were found in sexually abused girls recruited within six months of disclosure compared with non-abused sociodemographically matched controls. This suggests morning hypersecretion of cortisol in sexually abused girls (Putnam et al., 1991). Maltreated young children with major depression failed to show the expected diurnal decrease in cortisol secretion from morning to afternoon (Hart et al., 1996; Kaufman, 1991). Medication naïve maltreated children with PTSD excreted significantly greater 24-h UFC levels than non-abused healthy controls (De Bellis et al., 1999a). Increased ACTH response to human CRH, but normal cortisol secretion, was reported in maltreated prepubertal depressed children undergoing current psychosocial adversity compared to depressed children with prior histories of maltreatment, depressed non-abused children, and healthy children (Kaufman et al., 1997). These results are similar to a recent study which showed that women with a history of child abuse evidenced hypersensitivity of the HPA axis while undergoing a social stressor (Heim et al., 2000) and may the a result of ‘priming’.

However, in studies of children with past trauma, evidence of elevated central CRH and chronic compensatory adaptation of the HPA axis is seen. Attenuated plasma ACTH responses to ovine CRH in sexually abused girls studied several years after abuse disclosure was reported (De Bellis et al., 1994a). These subjects were currently living in a stable home environment and studied in a quiet relaxed setting. The abused subjects had histories of severely depressed mood with suicidal behavior, but only one had a diagnosis of PTSD. The abused girls exhibited reduced evening basal, ovine CRH-stimulated, and time integrated total plasma ACTH concentrations compared with controls. Plasma total and free cortisol responses to ovine CRH stimulation did not differ between the two groups. Thus, sexually abused girls manifest a dysregulatory disorder of the HPA axis, associated with hyporesponsiveness of the pituitary to exogenous CRH and normal overall cortisol secretion to CRH challenge. CRH hypersecretion may have led to an adaptive down-regulation of CRH receptors in the anterior pituitary. Armenian adolescents who lived close to the epicenter of the, 1988 earthquake and experienced a significant direct threat to life had greater PTSD and co-morbid depressive symptoms, lower baseline mean salivary cortisol levels, and greater afternoon suppression of cortisol by dexamethasone, five years after exposure, compared to Armenian adolescents who lived 20 miles from the
epicenter (Goenjian et al., 1996). These results are similar to the HPA axis findings of low 24-h UFC levels in baseline studies of combat-related adult PTSD (Yehuda et al., 1991a,b).

These studies suggest that there are persistent stress-induced changes in the HPA axis during childhood trauma. Chronic hyperactivity of central CRH are consistent with these changes, which, in turn, may lead to symptoms of PTSD as well as depression (Chrousos and Gold, 1992; Owens and Nemeroff, 1991).

5. A developmental traumatology contributory mechanism for alcohol and substance use disorders

Elevations of catecholamines and central CRH during development may enhance one’s risk for an alcohol and/or substance use disorder. Traumatic experiences may interact with inherent individual differences (i.e. genetic vulnerabilities) in the ability of an individual’s biological stress response systems to either maintain homeostasis in the face of chronic and severe stress or to permanently change in response to stressors. Dysregulation of these biological stress response systems can lead to negative affect symptoms, which in turn may lead to an increased risk for ‘self-medicating’ with alcohol and substances in later life.

Thus, maltreatment experiences in childhood and adolescence may increase the probability of alcoholism and substance abuse through attempts to use alcohol and other drugs to reduce symptoms of PTSD and its common co-morbid symptoms of depression (Labouvie, 1986; Newcombe and Harlowe, 1986), and to dampen the effects of dysregulated biological stress response systems (Higley et al., 1991). On the other hand, adolescent onset alcohol and substance abuse and dependence may cause further dysregulation of biological stress response systems by increasing plasma cortisol levels (Groote Veldman and Meinders, 1996; Rivier, 1996). This process may be neurotoxic to the hippocampus, a brain structure involved in facilitating attention, concentration and short-term memory (De Bellis et al., 2000a).

Furthermore, in the developing brain, elevated levels of catecholamines and cortisol may lead to adverse brain development through the mechanisms of accelerated loss (or metabolism) of neurons (Sapolsky, 2000; Simantov et al., 1996; Smythies, 1997), delays in myelination (Dunlop et al., 1997), abnormalities in developmentally appropriate pruning (Lauder, 1988; Todd, 1992) and/or by inhibiting neurogenesis (Gould et al. 1997a,b, 1998; Tanapat et al., 1999). Consequently, a stress-related adverse brain development mechanism may also contribute to maturation failures in the frontal and prefrontal cortex. Recently, lower N-acetylaspartate/creatine ratios, which were suggestive of neuronal loss in the anterior cingulate region of the medial prefrontal cortex were described in maltreated children and adolescents with PTSD compared to sociodemographically and IQ matched controls (De Bellis et al., 2000b). Hence these stress-induced mechanisms may lead to failures in executive function and self-regulation and a greater incidence of impulsive behaviors (i.e. alcohol and substance abuse).
6. The association of child maltreatment with adolescent and adult alcohol and substance use disorders

A review of the literature suggests that childhood maltreatment and the diagnosis of PTSD are associated with an enhanced risk of adolescent and adult alcohol and substance use disorders. Physical and sexual abuse are associated with adolescent PTSD, and often occur prior to alcohol and substance abuse in adolescents (Clark et al., 1997a,b; Dembo et al. 1988, 1992; Deykin and Buka, 1997). The results of a survey of public school students in grades 6, 9, and 12, found that physical and sexual abuse were associated with an increased likelihood of alcohol, marijuana, and other substance use (Harrison et al., 1997). This association was particularly strong for sexual abuse (Hussey and Singer, 1993; Singer et al., 1989). In community adult samples, sexual abuse histories have been found to be associated with increased alcohol consumption (Widom et al., 1995; Wilsnack et al., 1997). Childhood maltreatment was found to moderate the relationship between parental alcoholism and young adulthood alcoholism in offspring (Sher et al., 1997).

A recent study involving an HMO sample found that adults with a history of adverse childhood experiences had an increased incidence of alcohol and substance use disorders (Felitti et al., 1998). Furthermore, after controlling for family background and parental psychopathology in female twin pairs discordant for sexual abuse, the twin exposed to sexual abuse was found to have a substantially increased risk for developing alcohol and other drug dependence (Kendler et al., 2000). These results lead the authors to state that childhood sexual abuse was causally related to an increased risk for adult psychiatric and alcohol and substance use disorders.

7. Conclusions

Early childhood traumatic experiences, such as childhood maltreatment, are associated with an enhanced risk of adolescent and adult alcohol and substance use disorders. The adverse influences of maltreatment on major biological stress response systems and brain development may be contributory to this enhanced risk. Thus, alterations in biological stress systems may enhance vulnerability for psychopathology and put a child at increased risk for adolescent or young adult onset alcohol or substance use disorders.

These studies are based on cross sectional analyses of limited data. Thus causation can not be proven. In view of these limitations, this review also did not attempt to link the neurobiology of maltreatment-related PTSD with the neurobiology of alcohol and substance use disorders. Future studies in developmental traumatology that are aimed at understanding this association may have important implications for the prevention and treatment of adult alcohol and substance use disorders. Elucidating the biological sequelae and mechanisms of symptom production in PTSD and associated co-morbid psychiatric disorders will help the field begin to understand some of the environmental-related mechanisms involved in the development of adolescent or young adult onset alcohol or substance use disorders. Accordingly, prospective
longitudinal studies in developmental traumatology designed to attenuate the psycho-biological dysregulation associated with childhood maltreatment, may prove to be critical in the effort to develop early alcohol and substance abuse prevention and intervention programs.

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