INTRODUCTION

Childhood exposure to trauma is a common phenomenon, with 25% of young people experiencing a traumatic event such as physical abuse; sexual abuse; witnessing violence, war, and terrorism; natural disasters; illness; or injury by the time they reach age 16 years. Internal (eg, genetics and individual traits) and environmental (eg, home, school, community) factors interact to determine the outcome that trauma will have on a child. Many young people demonstrate resilience, the ability to adapt and cope despite adversity, and continue to develop normally. Some will be susceptible to develop mood and/or anxiety disorders. Yet others are at risk to develop posttraumatic stress disorder (PTSD), which can

KEYWORDS

- Pediatric posttraumatic stress disorder
- Childhood trauma
- Childhood development
- Neurobiology
- Interventions

KEY POINTS

- It has been proposed that posttraumatic stress disorder (PTSD) should be conceptualized as a dimensional and continuous, rather than a categorical, clinical entity in youth.
- As a result of young children’s limitations in their verbal capacity, they may use other means to express themselves such as being fussy or having temper tantrums, types of behavior often overlooked as symptoms of PTSD.
- Chronic periods of stress may impair the hypothalamic–pituitary–adrenal (HPA) axis resulting in dysregulation of cortisol secretion, which has been suggested as a marker for PTSD; though neuroendocrine studies have yielded mixed results regarding the relationship of cortisol and pediatric PTSD, the majority of studies report high levels of cortisol to be indicative of PTSD.
- Psychotherapeutic interventions have the potential to modulate negative effects of PTSD by providing new experiences that repair brain function and promote the growth of neural connections.

The authors have nothing to disclose.
Department of Psychiatry and Behavioral Sciences, Division of Child and Adolescent Psychiatry, Stanford School of Medicine, Stanford University, 401 Quarry Road, Stanford, CA 94305, USA
* Corresponding author.
E-mail address: vcarrion@stanford.edu

http://dx.doi.org/10.1016/j.chc.2012.05.004
childpsych.theclinics.com
1056-4993/12/$ – see front matter © 2012 Elsevier Inc. All rights reserved.
have a negative impact on biological, cognitive, emotional, behavioral, and social domains of the child. Although trauma exposure is associated with the development of different conditions, this article focuses specifically on PTSD because it is the disorder (or injury) with the most widely studied outcome across populations and trauma types.

CLASSIFICATION OF PTSD

It has long been established that PTSD occurs in adults; however, it was not until the publication of the DSM-III-R that it was recognized that young people experience similar symptoms. The diagnosis of PTSD is based on six major criteria, two of which define the trauma (criterion A). The DSM-IV-TR provides the following description:

1. The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.
2. The person’s response involved intense fear, helplessness, or horror.

In children, the latter may be expressed as disorganized or agitated behavior. Symptoms of PTSD are divided into three clusters:

1. **Reexperiencing of the traumatic event** (criterion B), which includes flashbacks, nightmares, exaggerated startle response, and intrusive recollections.
2. **Avoidance of trauma** with regard to relevant stimuli and numbing of general responsiveness (criterion C), which includes feeling detached or estranged from others and deriving markedly less pleasure from activities that previously were enjoyed.
3. **Hyperarousal** (criterion D), which includes irritability, hypervigilance, and difficulties in sleep and concentration.

Symptoms must persist for at least 1 month (criterion E) and cause significant distress or impairment in functioning (criterion F).

Proposed changes to the PTSD diagnosis for DSM-V take into account developmental variations in symptom manifestation, including a separate subtype for children younger than age 6 years. Additional changes include clarification of what constitutes a traumatic event, elimination of criterion A2, division of cluster C avoidance and numbing into two separate criteria, and elimination of the acute versus chronic designation. In addition, cluster D (numbing) has new symptoms emphasizing the role of self or other blaming and persistently negative emotional states and cluster E (hyperarousal) has an added symptom of reckless or self-destructive behavior. Table 1 shows the current DSM-IV criteria and highlights the proposed DSM-V changes.

| Table 1 | Shows the current DSM-IV criteria and highlights the proposed DSM-V changes. |

There are instances in which PTSD symptoms may differ in young people. For example, children may engage in repetitive play and experience frightening dreams without specific content. Many children develop fears associated with certain aspects of a trauma that may develop into phobias. Increased irritability and anger may lead to aggressive behavior in such cases. Children with PTSD often experience guilt over what they should or could have done in a particular situation. A number of cognitive problems have also been observed in traumatized youth. Children with PTSD frequently report difficulties in concentration, especially with regard to schoolwork. They may develop memory problems, both in learning new material and in remembering previously acquired skills.
Table 1
Proposed changes in the diagnostic criteria of PTSD in the DSM-V

<table>
<thead>
<tr>
<th>DSM-IV Criteria</th>
<th>DSM-V Proposed Changes</th>
</tr>
</thead>
</table>
| **A.** The person has been exposed to a traumatic event in which both of the following were present:  
1. The person experienced, witnessed, or was confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others.  
2. The person’s response involved intense fear, helplessness, or horror. Note: In children, this may be expressed instead by disorganized or agitated behavior. | **A.** The person was exposed to one or more of the following event(s): death or threatened death, actual or threatened serious injury, or actual or threatened sexual violation, in one or more of the following ways:  
1. Experiencing the event(s) him/herself  
2. Witnessing, in person, the event(s) as they occurred to others  
3. Learning that the event(s) occurred to a close relative or close friend; in such cases, the actual or threatened death must have been violent or accidental  
4. Experiencing repeated or extreme exposure to aversive details of the event(s) (eg, first responders collecting body parts; police officers repeatedly exposed to details of child abuse); this does not apply to exposure through electronic media, television, movies, or pictures, unless this exposure is work related.  
Criterion A2 eliminated. |
| **B.** The traumatic event is persistently reexperienced in one (or more) of the following ways:  
1. Recurrent and intrusive distressing recollections of the event, including images, thoughts, or perceptions. Note: In young children, repetitive play may occur in which themes or aspects of the trauma are expressed.  
2. Recurrent distressing dreams of the event. Note: In children, there may be frightening dreams without recognizable content.  
3. Acting or feeling as if the traumatic event were recurring (includes a sense of reliving the experience, illusions, hallucinations, and dissociative flashback episodes, including those that occur on awakening or when intoxicated). Note: In young children, trauma-specific reenactment may occur.  
4. Intense psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event.  
5. Physiological reactivity on exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event. | **B.** Intrusion symptoms that are associated with the traumatic event(s) (that began after the traumatic event(s)), as evidenced by one or more of the following:  
1. Spontaneous or cued recurrent, involuntary, and intrusive distressing memories of the traumatic event(s). Note: In children, repetitive play may occur in which themes or aspects of the traumatic event(s) are expressed.  
2. Recurrent distressing dreams in which the content and/or affect of the dream is related to the event(s). Note: In children, there may be frightening dreams without recognizable content.  
3. Dissociative reactions (eg, flashbacks) in which the individual feels or acts as if the traumatic event(s) were recurring (Such reactions may occur on a continuum, with the most extreme expression being a complete loss of awareness of present surroundings.) Note: In children, trauma-specific reenactment may occur in play.  
4. Intense or prolonged psychological distress at exposure to internal or external cues that symbolize or resemble an aspect of the traumatic event(s).  
5. Marked physiological reactions to reminders of the traumatic event(s). |

(continued on next page)
<table>
<thead>
<tr>
<th>DSM-IV Criteria</th>
<th>DSM-V Proposed Changes</th>
</tr>
</thead>
<tbody>
<tr>
<td>C. Persistent avoidance of stimuli associated with the trauma and numbing of general responsiveness (not present before the trauma), as indicated by three (or more) of the following:</td>
<td>Avoidance and numbing as two separate clusters.</td>
</tr>
<tr>
<td>1. Efforts to avoid thoughts, feelings, or conversations associated with the trauma.</td>
<td>C. Persistent avoidance of stimuli associated with the traumatic event(s) (that began after the traumatic event(s)), as evidenced by efforts to avoid one or more of the following:</td>
</tr>
<tr>
<td>2. Efforts to avoid activities, places, or people that arouse recollections of the trauma.</td>
<td>1. Avoids internal reminders (thoughts, feelings, or physical sensations) that arouse recollections of the traumatic event(s).</td>
</tr>
<tr>
<td>3. Inability to recall an important aspect of the trauma.</td>
<td>2. Avoids external reminders (people, places, conversations, activities, objects, situations) that arouse recollections of the traumatic event(s).</td>
</tr>
<tr>
<td>4. Markedly diminished interest or participation in significant activities</td>
<td>D. Negative alterations in cognitions and mood that are associated with the traumatic event(s) (that began or worsened after the traumatic event(s)), as evidenced by three or more of the following: Note: In children, as evidenced by two or more of the following:</td>
</tr>
<tr>
<td>5. Feeling of detachment or estrangement from others.</td>
<td>1. Inability to remember an important aspect of the traumatic event(s) (typically dissociative amnesia; not due to head injury, alcohol, or drugs).</td>
</tr>
<tr>
<td>6. Restricted range of affect (eg, unable to have loving feelings).</td>
<td>2. <strong>Persistent and exaggerated negative expectations about one’s self, others, or the world</strong> (eg, “I am bad,” “no one can be trusted,” “I’ve lost my soul forever,” “my whole nervous system is permanently ruined,” “the world is completely dangerous”).</td>
</tr>
<tr>
<td>7. Sense of a foreshortened future (eg, does not expect to have a career, marriage, children, or a normal life span).</td>
<td>3. <strong>Persistent and exaggerated negative expectations about the cause or consequences of the traumatic event(s).</strong></td>
</tr>
</tbody>
</table>

| D. Persistent symptoms of increased arousal (not present before the trauma), as indicated by two (or more) of the following: | E. Alterations in arousal and reactivity that are associated with the traumatic event(s) (that began or worsened after the traumatic event(s)), as evidenced by three or more of the following: Note: In children, as evidenced by two or more of the following: |
| 1. Difficulty falling or staying asleep | 1. Irritable or aggressive behavior |
| 2. Irritability or outbursts of anger | 2. **Reckless or self-destructive behavior** |
| 3. Difficulty concentrating | 3. Hypervigilance |
| 4. Hypervigilance | 4. Exaggerated startle response |
| 5. Exaggerated startle response. | 5. Problems with concentration |

The distinction between single-event and multiple-event trauma (Table 2) has been referred to by various designations such as acute versus chronic trauma, type I versus type II trauma, and simple versus complex trauma.\textsuperscript{12,13}

**Type I Trauma**

Type I trauma has been defined as a single-event trauma that usually meets the DSM criteria for PTSD and is characterized by the classic symptom clusters of repetition, avoidance, and increased arousal.\textsuperscript{12} In addition, young people with single-event trauma may experience symptoms of trauma including specific fears; regressive behavior; loss and grief reactions; cognitive–perceptual distortions; changed attitudes about the self, others, or the future; and reexperiencing of perceptual, affective, ideational, or somatic components of the trauma. This type of trauma generally occurs along with a background of normal development. Although impairments are expected in such areas as academic performance, maintenance of peer and family relationships and, at times, in daily living activities, individuals are expected to make a full recovery in functioning.\textsuperscript{14}

**Type II Trauma**

Type II trauma refers to multiple-event trauma and often presents with the classic symptoms of PTSD. Repeated exposure can also lead to the development of more severe symptoms such as massive denial, psychic numbing, self-anesthesia, and personality problems.\textsuperscript{12} In this type of trauma, the deviation from the normal trajectory of development occurs earlier and the chronic nature inhibits the trajectory from returning to its normal course. Herman (1992) coined the term “complex trauma” to describe the chronic effects of type II trauma and maintains that the much more diverse consequences of this type of trauma require a more comprehensive diagnosis than the PTSD normally observed in single-incident trauma.\textsuperscript{13} In line with this, a new provisional diagnosis of Developmental Trauma Disorder has been proposed to better capture the experiences of youth with complex trauma such as community violence that is characterized by multiple events and/or prolonged exposure.\textsuperscript{15} The premise of this proposed diagnosis is that multiple trauma exposures have consistent and predictable outcomes that impact on many domains of functioning. Specifically, impairment occurs in seven domains: attachment, biology, affect regulation, dissociation, behavioral regulation, cognition, and self-concept.\textsuperscript{16} The diagnosis centers on triggered dysregulation in response to traumatic reminders, stimulus generalization, and behavioral attempts to avoid the reexperiencing of traumatic effects. Thus it is suggested that treatment should target three key areas: establishing safety and competence, dealing with traumatic reenactments, and integrating body and mind.\textsuperscript{15}

**Table 2**

<table>
<thead>
<tr>
<th>Single-Event Trauma</th>
<th>Multiple-Event Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Acute, one-time event.</td>
<td>Chronic and/or many events.</td>
</tr>
<tr>
<td>Classic symptoms of PTSD.</td>
<td>Symptoms are more intrinsic (eg, numbing, denial, personality changes).</td>
</tr>
<tr>
<td>Normal development is resumed.</td>
<td>Normal development is inhibited.</td>
</tr>
</tbody>
</table>

SINGLE-EVENT VERSUS MULTIPLE-EVENT TRAUMA

The distinction between single-event and multiple-event trauma (Table 2) has been referred to by various designations such as acute versus chronic trauma, type I versus type II trauma, and simple versus complex trauma.\textsuperscript{12,13}
Studies Comparing Single-Event and Multiple-Event Trauma

Several studies have compared single-event and multiple-event traumas in youth. In a sample of sexually abused children, common type I trauma symptoms included reexperiencing events such as nightmares, avoidant behaviors (ie, fear of certain places and situations, withdrawal), and increased hyperarousal (ie, difficulties with sleep and concentration, irritability). Furthermore, it was found that those with type II trauma experienced more anxiety and depression, and had deficits in coping strategies related to daily and extreme stresses. They tended to have an enduring maladaptive attributional style (ie, learned helplessness); often experienced dissociative states (ie, massive denial and numbing); and frequently had excessive, poorly regulated responses to anger-provoking stimuli. In a sample of South African adolescents, those exposed to multiple-event traumas experienced more PTSD symptoms and depression than those exposed to single-event traumas. Hagenaars and colleagues found that individuals with multiple-event trauma experienced more dissociation and shame compared to those with single-event trauma.

Limitations of the Current Classification of PTSD

There has been much debate regarding whether the current diagnostic criteria, which rely on behavioral descriptions, adequately capture the presentation of PTSD in youth. It has been proposed that PTSD should be conceptualized as a dimensional and continuous, rather than a categorical, clinical entity in youth. Developmental variations may affect children’s expression of PTSD symptoms and there has been much criticism that the current DSM criteria do not account for the developmental nature of the disorder. For example, young children are limited in their verbal capacity and thus they may use other means to express themselves such as being fussy or having temper tantrums. These types of behavior are often overlooked as symptoms of PTSD. Furthermore, relying on the current categorical model for diagnosis of PTSD may result in neglect of individuals who fall short of meeting the full criteria but may still be experiencing significant impairment. Several studies have found that subthreshold PTSD is similar to full-criteria PTSD in terms of its adverse effects. This suggests that the diagnosis of PTSD in youth might be more accurate based on the intensity of symptoms related to functional impairment rather than number of symptoms met.

Another major limitation of behavioral descriptions is the lack of recognition that fear networks underlying the mechanism of PTSD may perpetuate other forms of anxiety. Young people with PTSD are at greater risk of the future development of anxiety disorders such as obsessive–compulsive disorder, generalized anxiety disorder, and phobias. Thus, the diagnostic criteria ought to consider that PTSD may precede the development of other anxiety disorders and that there is potential for symptom overlap. Comorbidity rates for mood and anxiety disorders are as high as 80% in posttraumatic populations, which suggests that these comorbidities may need to be included in the classification of PTSD.

Assessment of PTSD

Numerous instruments exist for screening and diagnosis of childhood PTSD, as well as evaluation of trauma exposure and associated symptoms. Although a full review of all available measures is beyond the scope of this discussion, some of the most widely used instruments are described.
Clinician-Administered PTSD Scale for Children and Adolescents

The Clinician-Administered PTSD Scale for Children and Adolescents (CAPS-CA) is considered the gold standard in childhood PTSD assessment. The CAPS-CA is used to assess PTSD and associated symptoms in youth ages 8 to 18. It consists of 36 questions based on a specific event that the child identifies as most distressing. The CAPS-CA evaluates current and lifetime diagnosis; frequency and intensity of symptoms; and functioning in social, developmental, and academic domains.

UCLA PTSD Reaction Index for DSM-IV

The UCLA PTSD Reaction Index for DSM-IV (PTSD-RI) is a self-report measure with child (ages 7–12), adolescent (ages 13–18), and parent versions. The PTSD-RI contains 48 items that assess exposure to 26 types of traumatic events and evaluates DSM-IV PTSD criteria for the event that the child identifies as the most distressing. Youth rate symptoms on a 5-point Likert scale (0 = “none of the time” to 4 = “most of the time”) and the parent version has an option of responding “don’t know” to account for symptoms that the parent may not have observed. It also evaluates associated symptoms of guilt and fear of recurrence of the event.

Child PTSD Interview

This is a 95-item semistructured interview that assesses DSM-IV PTSD criteria and associated symptoms. Questions are written at a third-grade level, though the measure has been used with younger children. Each symptom item is rated as either being present or absent. There is also a parent form that assesses the same dimensions as the child form.

Children’s PTSD Inventory

The Children’s PTSD Inventory (CPTSDI) is a clinician-administered measure for children ages 6 to 18 based on the DSM-IV criteria for PTSD. The child is first screened for exposure to various traumatic events by being asked if he or she ever experienced it or felt upset for not being able to stop it from happening. If an event meets the screening criteria, then symptoms are assessed in reference to the event. In addition to a PTSD total score, the CPTSDI also yields scores on five subscales: Situational Reactivity, Reexperiencing, Avoidance and Numbing, Increased Arousal, and Significant Impairment.

Child PTSD Symptom Scale

The Child PTSD Symptom Scale (CPSS) is a 26-item self-report that assesses PTSD and symptom severity in youth ages 8 to 18. Items are rated on a 4-point Likert scale (0 = “not at all” to 3 = “5 or more times a week”). Functional impairment is also assessed (0 = “absent” and 1 = “present”) but is not based on DSM-IV criteria. The measure provides total scores for symptom severity and severity of impairment, as well as scores for PTSD symptom clusters.

Trauma Symptom Checklist for Children

The Trauma Symptom Checklist for Children (TSCC) is a brief self-report for children ages 8 to 16 that screens for trauma exposure and posttraumatic stress but is not intended to be diagnostic. It consists of two validity scales (over- and underreporting) and six clinical scales (anxiety, depression, posttraumatic stress, sexual concerns, dissociation, and anger). Children are presented with thoughts, feelings,
and behaviors related to traumatic events and are asked to mark how often they happened on a 4-point Likert scale (0 = “never” to 3 = “almost all the time”).

**Childhood Trauma Questionnaire**

The Childhood Trauma Questionnaire (CTQ) is a 28-item, self-report that screens for a history of abuse and neglect in children over the age of 12. It assesses exposure to five types of trauma: emotional, physical, and sexual abuse, and emotional and physical neglect. In addition, it contains a three-item minimization/denial scale for detecting under-reporting.

**THE ROLE OF NEUROBIOLOGY IN UNDERSTANDING PTSD**

**The Role of Cortisol**

The endocrine system, crucial to growth and development, is influenced by the hypothalamic–pituitary–adrenal (HPA) axis that secretes the hormone cortisol during times of stress to mobilize an individual into action. Chronic periods of stress, however, may impair the HPA axis, resulting in dysregulation of cortisol secretion, which has been suggested as a marker for PTSD. Neuroendocrine studies have yielded mixed results regarding the relationship of cortisol and pediatric PTSD. The majority of studies report high levels of cortisol to be indicative of PTSD. On the other hand, lower cortisol levels have been associated with PTSD in youth exposed to an earthquake, sexually abused girls, and youth bereaved by the September 11, 2001 terrorist attacks. Attempts have been made to clarify these inconsistencies. In a sample of youth exposed to interpersonal violence, a higher level of salivary cortisol was positively associated with PTSD among individuals with recent traumas (previous year), but in individuals with distal traumas (more than a year prior to assessment) the association was the opposite; the more PTSD symptoms the lower the levels of cortisol. These findings highlight the importance of “time since trauma” when evaluating cortisol levels in PTSD. In conclusion, it appears that what were thought inconsistencies in the neuroendocrine literature might not be so, as the latest evidence suggests that cortisol levels in traumatized youth may be dependent upon trauma type and duration.

It has also been suggested that significant variations exist in patterns of cortisol regulation among traumatized youth. For example, a study examining different trauma types found that high morning and afternoon cortisol levels were typical of youth with both physical and sexual abuse. However, those with only physical abuse had lower levels of cortisol in the morning, with a smaller decrease in levels from morning to afternoon. In another study of youth exposed to interpersonal trauma, individuals with posttraumatic symptoms had sharper morning declines and higher evening cortisol levels than nontraumatized youth.

It therefore appears that traumatized young people may display greater fluctuations in cortisol levels throughout the day. In conclusion, it appears that what were thought inconsistencies in the neuroendocrine literature might not be so, as the latest evidence suggests that cortisol levels in traumatized youth may be dependent upon trauma type and duration.

**The Role of Neuroimaging**

Neuroimaging (structural magnetic resonance imaging [MRI], functional MRI [fMRI], and magnetic resonance spectroscopy [MRS]) studies indicate that abnormalities in brain structure and function are also linked to the pathophysiology of PTSD. Three
brain regions that are particularly vulnerable to the effects of childhood trauma are the amygdala, prefrontal cortex, and hippocampus (Fig. 1).

The amygdala
The amygdala, which is part of the limbic system, is responsible for the processing of emotions and facilitates consolidation of emotional memories.54 It plays an important role in assessing threatening stimuli.55 Potentially dangerous stimuli are first processed in the thalamus and either directly or indirectly reach the amygdala where an emotional response will be developed with or without cognitive input. Pediatric neuroimaging studies suggest that there are no amygdala volume differences between traumatized youth with PTSD and healthy controls.56–58 Although one study found a 5.1% reduction in amygdala volume of maltreated youth compared to healthy controls, when the results were corrected for total brain gray matter, no significant differences remained.59 Numerous functional imaging studies, utilizing threatening words or faces, have identified hyper-responsiveness of the amygdala in traumatized adults.60 A similar result was found in one study of trauma0tized youth. In youth exposed to interpersonal trauma, individuals with posttraumatic stress symptoms had increased amygdala activation in response to threatening facial expressions when compared with healthy controls.61 In this study, 23 medication-naive youth with PTSD symptoms and 23 age- and gender-matched healthy controls underwent fMRI while viewing fearful, angry, sad, happy, and neutral expressions. The PTSD group had greater activation in the early part of the epoch, including greater early phase amygdala activation to angry faces compared to the controls.
The prefrontal cortex

The prefrontal cortex (PFC) is the anterior part of the frontal lobes involved in executive function. Executive function involves operations related to attention regulation, memory processing, and response inhibition.62

The majority of the pediatric PTSD structural neuroimaging studies indicate abnormalities of the PFC. Maltreated youth failed to show the typical frontal lobe asymmetry (right > left) seen in controls, mainly due to larger left frontal lobe volume.59 A follow-up study on a similar sample found reduced white matter volume in the PFC although this was no longer significant after adjustment for intracranial volume.58 In a study examining the relationship between cortisol and the PFC in children exposed to interpersonal trauma compared to healthy controls, youth with PTSD symptoms had decreased left ventral and left inferior prefrontal gray volumes.63 In addition, elevated pre-bedtime cortisol levels were associated with reduced left ventral PFC gray volume. The results suggest a potential link between cortisol dysregulation and PFC volume. One study found no PFC differences between traumatized youth and healthy controls.56

Within the PFC lies the medial prefrontal cortex (mPFC) which, through its connection with the amygdala, is involved in emotional regulation and the processing of fear.54 Impairments in this region have also been implicated in pediatric PTSD. De Bellis and colleagues used single-voxel proton MRS (proton MRS) to measure the relative concentration of N-acetylaspartate and creatine, markers of neural integrity, in the anterior cingulate, a specific area within the mPFC.64 The lower concentration of N-acetylaspartate compared to creatine suggests abnormal neuronal metabolism. In an fMRI task assessing sustained attention and response inhibition, maltreated adolescents with PTSD showed relatively decreased activation of the middle frontal cortex, but increased activation in the mPFC when compared to healthy controls.65 These findings suggest that traumatized youth with PTSD are not engaging core areas of the PFC to the same extent as healthy individuals.

The hippocampus

The hippocampus is a structure in the limbic system responsible for consolidation of memory.66 Although adult trauma studies have consistently found reduced hippocampal volumes, findings from a meta-analytic study indicate that hippocampal volumes fail to differ in maltreated children relative to controls based on both cross-sectional child studies and one longitudinal study.67 More recent longitudinal studies, however, have found differences in the hippocampus of traumatized youths. Carrion and colleagues found that severity of PTSD and cortisol levels, independently, predicted reductions in hippocampal volume over a 12- to 18-month interval.66 During a memory retrieval task, youth with PTSD showed reduced activation of the right hippocampus compared to nontraumatized controls.68 Severity of avoidance and emotional numbing symptoms correlated with reduced left hippocampal activation during retrieval in the PTSD group. The inconsistencies found in hippocampal volume between traumatized adults and traumatized youth may be due to developmental processes. A study of rats exposed to early stress and sacrificed at different ages showed that differences in the hippocampus emerged only after young adulthood.69 Thus, trauma-induced glucocorticoid exposure may damage the hippocampus, but it is possible that decreased hippocampal volume occurs only in later development after chronic trauma exposure.

Neurobiological Studies Supplement Behavioral Studies

Although the behavioral descriptions of the DSM criteria are useful in providing a common language for PTSD, the aforementioned neurobiological studies may be used to supplement these descriptions to further enhance our understanding of the clinical profile of
PTSD. By facilitating the identification of biological risk factors and markers associated with childhood PTSD symptoms, these studies may aid in early detection of individuals susceptible to the disorder. In addition, such studies may help to clarify individual symptom presentations and identify markers associated with resiliency. The core areas targeted in current interventions match the impairments found in these studies. Thus, outcomes from these studies may inform treatment development and implementation. Furthermore, biological markers may assist in tailoring individualized treatments.

**BRAIN PLASTICITY**

Throughout childhood, brain development occurs during specific stages, termed critical periods, in which a specific brain structure is receptive to input from environmental stimuli and forms rapidly. Experience shapes the extensive formation and organization of neural connections. Failure to form connections results in selective elimination or “pruning” of unused neurons. As discussed, childhood trauma has been linked with alterations in the neurobiological systems involved in brain development and function. Impairments in these systems have been associated with PTSD; however, the brain has the ability to generate new neurons and repair connections in response to novel experiences, thus reversing the negative effects of trauma. The brain is at its most malleable during childhood, so early intervention can be critical. It has been suggested that the use of evidence-based psychotherapy to treat trauma symptoms may improve brain function by promoting cortical neurogenesis.

Adult studies have proven the utility of psychotherapy in improving brain function for a variety of disorders. For example, in a sample of adults with PTSD secondary to a motor vehicle accident, cognitive behavioral therapy (CBT) compared to a waitlist control group was effective in reducing right hemisphere activation, which is associated with avoidance symptoms. Another study found that before treatment, depressed individuals had reduced activation of the dorsolateral PFC, parietal cortex, and striatum but that group CBT helped restore activation of these regions to the same level as healthy controls. Similar studies devoted to youth are now emerging that examine this same interface. For example, a family-based intervention consisting of 22 weekly group sessions and 10 biweekly home visits was successful in altering cortisol levels in preschoolers at risk for conduct problems, compared to healthy controls.

**PSYCHOTHERAPEUTIC INTERVENTIONS**

**Single-Event Trauma**

The most widely used treatment for single-event trauma has been CBT. Many variations of trauma-specific CBT interventions exist. However, all share components summarized by the acronym PRACTICE:

- Psychoeducation
- Relaxation and coping skills
- Affective expression and modulation
- Cognitive processing
- Trauma narrative
- In vivo exposure to trauma reminders
- Conjoint child–parent sessions
- Enhancing safety and future development

The majority of these treatments have been implemented as school-based group interventions.
The Multi-Modal Trauma Treatment Protocol (MMTT), an intervention utilizing developmentally sensitive methods, has been successfully implemented in both school and community mental health settings.\textsuperscript{81,82}

The Cognitive Behavioral Intervention for Trauma in Schools (CBITS) is a 10-session treatment that has been shown to improve psychosocial functions in youth exposed to violence.\textsuperscript{83}

Several studies of earthquake survivors, victims of the Bosnian war, and victims of community violence, respectively, have found that trauma/grief focused therapy resulted in significant reduction of PTSD symptoms.\textsuperscript{84–86}

Eye movement desensitization and reprocessing (EMDR) has also been used for the treatment of single-trauma treatment. EMDR is believed to work by helping to reprocess traumatic memories through forming associations with positive information stored in other memory networks.\textsuperscript{87} A randomized, controlled trial of EMDR in youth exposed to a natural disaster resulted in a decrease in trauma related memories and their associated symptoms.\textsuperscript{87} Other modalities that have been employed in single-trauma intervention include play therapy and mind–body skills.\textsuperscript{88,89}

### Table 3

<table>
<thead>
<tr>
<th>Single Trauma</th>
<th>Multiple Trauma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Trauma-focused cognitive behavioral therapy (TF-CBT)</td>
<td>TF-CBT Website: TF-CBT.musc.edu</td>
</tr>
<tr>
<td>Website: TF-CBT.musc.edu</td>
<td></td>
</tr>
<tr>
<td>Multi-Modal Trauma Treatment Protocol (MMTT)</td>
<td>Trauma systems therapy (TST) Website: <a href="http://www.aboutourkids.org">www.aboutourkids.org</a></td>
</tr>
<tr>
<td>Website: <a href="http://www.ccfh.nc.org">www.ccfh.nc.org</a></td>
<td></td>
</tr>
<tr>
<td>Cognitive Behavioral Intervention for Trauma in Schools (CBITS)</td>
<td>CBITS Website: <a href="http://www.cbitsprogram.org">www.cbitsprogram.org</a></td>
</tr>
<tr>
<td>Website: <a href="http://www.cbitsprogram.org">www.cbitsprogram.org</a></td>
<td></td>
</tr>
<tr>
<td>Trauma/grief focused therapy</td>
<td>Child–parent psychotherapy (CPP) Website: <a href="http://www.childtrauma.ucsf.edu">www.childtrauma.ucsf.edu</a></td>
</tr>
<tr>
<td>Website: <a href="http://www.nctsnet.org">www.nctsnet.org</a></td>
<td></td>
</tr>
<tr>
<td>Eye movement desensitization and reprocessing (EMDR)</td>
<td>Parent–child interaction therapy (PCIT) Website: <a href="http://www.pcit.org">www.pcit.org</a></td>
</tr>
<tr>
<td>Play therapy</td>
<td>Intergenerational Trauma Treatment Model (ITTM) Website: theittm.com</td>
</tr>
<tr>
<td>Mind–body skills</td>
<td>Cue-centered treatment (CCT) Website: <a href="http://www.elsrp.stanford.edu">www.elsrp.stanford.edu</a></td>
</tr>
</tbody>
</table>

Multiple-Event Trauma

Just as with single-trauma treatment, CBT-based approaches are the most common for multiple-trauma treatment (Table 3).

- **Trauma-focused cognitive behavioral therapy (TF-CBT)** combines both individual and parent-child sessions, and consists of the PRACTICE components described previously.\textsuperscript{80} TF-CBT has proven efficacious in numerous randomized controlled trials for reduction of PTSD, depression, and other emotional and behavioral difficulties for both single-event and multiple-event
Trauma-focused CBT has also been proven to be superior to child-centered therapy in reducing PTSD symptoms, especially hyperarousal and avoidance in youth exposed to intimate partner violence.93

- **Trauma systems therapy (TST)** is an individual treatment addressing trauma-related symptoms and the environmental factors perpetuating them.94 TST has demonstrated improvements in PTSD symptoms, environmental stability, and functioning.94

Other treatments for multiple-trauma include parent–child therapies based on psychodynamic theory:

- **Child–parent psychotherapy (CPP)** is a dyadic treatment in which play and other expressive methods are used to repair attachment and regulate traumatic stress.95
- **Parent–child interaction therapy (PCIT)** has also been found to improve social, emotional, and behavioral functioning through play therapy and live coaching aimed at improving attachment.96
- **The Intergenerational Trauma Treatment Model (ITTM)**, an intervention aimed at monitoring dysfunctional family patterns and altering them, has resulted in improvements in social functioning in traumatized children.97

A manual-based hybrid treatment protocol, **The Stanford Cue-Centered Therapy (CCT)**, combines elements of CBT, psychodynamic, expressive, and family therapies and augments these with education in classical conditioning and trauma-related reminders (cues), focusing on how these are linked to current behaviors, emotions, thoughts, and physiological reactions.98 CCT emphasizes the importance of collaboration between the therapist, child, and caregiver to increase a sense of efficacy and empowerment through knowledge. CCT is divided into four parts:

1. Psychoeducation and coping strategies
2. Incorporating traumas into life narratives involving expression of emotions, filling of memory gaps, identification of cognitive distortions, and integration of the traumas into the greater context of the child’s life
3. Gradual exposure to cues while replacing maladaptive behaviors with adaptive ones
4. Consolidation of learned skills.

The AACAP 2010 practice parameters consider trauma-focused therapies to be the first line of treatment for youth with PTSD.99 The parameters state that these therapies ought to directly address the traumatic experience, include caregivers in the therapy process as agents of change, and focus not only on symptom improvement but also on enhancing functioning, resiliency, and developmental trajectories. The parameters state that, while studies on the efficacy of PTSD interventions are limited, CBT treatments and especially TF-CBT are the most widely researched and accepted. In addition, psychodynamic and family therapies are also suitable.100 Psychotherapy is considered the first choice of treatment for childhood PTSD, however psychotropic medications such as selective serotonin reuptake inhibitors (SSRIs) may be warranted in situations of severe symptoms, comorbidity, or when psychotherapy is not effective. A review of all psychotropic medication that may be effective in treating childhood PTSD is beyond the scope of this article; however, Wilkinson and Carrion provide such a review.100
IMPLICATIONS FOR RESEARCH AND CLINICAL PRACTICE

There is a growing consensus that the current diagnostic model for PTSD is neither sensitive enough nor sufficient for traumatized youth. Research from multiple disciplines suggests a distinction between the manifestations of PTSD in children compared to adults. This research also hints at the crucial role for the accumulation of stressors throughout life in shaping PTSD. The duration of this process of stress accumulation, referred to as “allostatic load,” may be more critical than chronological age of the individual for physiologic effects and symptom development. For example, an adult with no previous trauma history may present with a characteristically “young” response (ie, dissociation, high levels of diurnal cortisol, and no markers of stress on brain structures) whereas a child with chronic trauma may demonstrate more typical DSM symptoms, low levels of diurnal cortisol, and insidious effects of cortisol in key brain regions. The posttraumatic period is a dynamic process, exacerbated by the presence of trauma mimetic cues and worsening when inadequate or no intervention is available. Vietnam combat veterans who developed PTSD have significantly more history of child maltreatment compared to veterans with no PTSD. While PTSD was not manifested after child maltreatment, this previous history may have started a process that facilitated the development of the disorder after subsequent trauma.

Advancements in the field of neurobiology have aided assessment and treatment by identifying neurobiological risk factors and biomarkers for PTSD. Cortisol abnormalities have been associated with PTSD and appear to be affected by the type and duration of trauma. Impairments in certain brain regions, especially the amygdala, PFC, and hippocampus are also prominent in traumatized youth. Psychotherapeutic interventions have the potential to modulate these negative effects by providing new experiences that repair brain function and promote the growth of neural connections. Current evidence-based treatments employ such methods as emotional and behavioral regulation, cognitive processing, coping strategies, and exposure to traumatic reminders to target the areas identified from neurobiological studies. Future treatment outcome studies should integrate neuroscience with psychotherapy. Such studies will help identify which components of treatment are the most crucial for specific populations. These studies will also inform treatment outcome.

REFERENCES


100. Wilkinson J, Carrion VG. When and how to use psychopharmacology for pediatric PTSD. Curr Psychopharmacol, in press.