

A

ABUSE, CHILD PHYSICAL

A 1962 article by Kempe and his colleagues (Kempe, Silverman, Steele, Droegemueller, & Silver, 1962) called attention to the clinical condition of child physical abuse in its title “The Battered Child Syndrome” and propelled the problem of child abuse into national attention. In 1974, the first U.S. federal statute (Pub. L. 93-247) was passed outlining the responsibilities of the states to develop standards for defining abuse, to establish mandatory reporting of suspicions of maltreatment, and to identify state agencies responsible for investigating abuse allegations (Child Abuse Prevention and Treatment Act of 1974 [CAPTA]). Most recent statistics from the Administration on Children, Youth, and Families indicate that an estimated 3.6 million children in the United States were investigated and 899,000 determined to be victims of abuse or neglect in 2005 (U.S. Department of Health and Human Services, Administration on Children, Youth, and Families, 2007). Physical child abuse accounted for 16.6% of these cases and almost a quarter (24.1%) of fatalities associated with child maltreatment in 2005.

Definition of Physical Child Abuse

Physical abuse of children is generally defined as an act or acts of commission by a parent, guardian, or caretaker resulting in actual or potential harm or injury. The Federal Child Abuse Prevention and Treatment Act (CAPTA; 42 USCA §5106g), as amended by the Keeping Children and Families Safe Act of 2003, provides a foundation for states by identifying a minimum set of acts or behaviors that define child abuse. CAPTA does not provide definitions for specific types of child abuse. Legal definitions

of child physical abuse vary by state, but generally include physical injury (ranging from minor bruises to severe fractures or death) as a result of punching, beating, kicking, biting, shaking, throwing, stabbing, choking, hitting (with a hand, stick, strap, or other object), burning, or otherwise harming a child. Injurious consequences can range from red marks that persist for a matter of hours to bruises, other soft tissue injuries, or fractures. A “child” under this definition generally means a person who is under the age of 18 or who is not an emancipated minor. For research purposes, operational definitions of physical abuse vary, reflecting local statutes, regulations, or policies and/or methodological or theoretical positions of researchers; there is no gold standard against which definitions of physical abuse can be judged.

Short- and Long-Term Consequences

Immediate consequences may involve physical injuries that can have lasting effects on the subsequent development of the child. For example, some forms of physical abuse (e.g., battering) may lead to developmental retardation that, in turn, may affect school performance and behaviors. A child does not need to be struck on the head to sustain brain injuries, since infants may be shaken so forcefully that they suffer intracranial and intraocular bleeding with no signs of external trauma. Furthermore, the emotional and developmental scars that physically abused children receive may persist into adolescence and adulthood.

Physical abuse may affect multiple domains of functioning. Neurological and medical consequences range from minor physical injuries to severe brain damage and even death. Studies with physically abused children have documented

significant neuropsychological handicaps, including growth retardation, central nervous system damage, mental retardation, learning and speech disorders, and poor school performance. Deficiencies in reading ability and academic performance have been documented in physically abused children followed up into adolescence and young adulthood. Physically abused children also manifest behavioral and social problems, including reports of being physically assaultive toward peers and aggressive in school settings at young ages and at risk for conduct disorder, school problems, delinquency, crime, and violence in adolescence and young adulthood. Psychologically and emotionally, physical abuse takes a toll on the development of children. Physically abused children are at increased risk for posttraumatic stress disorder (PTSD; Widom, 1999) and major depressive disorder (Widom, DuMont, & Czaja, 2007) as well as self-destructive behaviors (suicide attempts and self-mutilation) and revictimization.

Consequences of Physical Abuse

A variety of theories have been offered to explain consequences associated with childhood physical abuse, although most have focused on the externalizing or aggressive and violent behavioral consequences. From a social learning perspective, physical aggression between family members provides a likely model for the learning of aggressive behavior as well as for the appropriateness of such behavior within the family (Bandura, 1973). Children learn behavior, at least in part, by imitating someone else's behavior, and this modeling of behavior is particularly potent when the model observed is someone of high status (such as a parent).

Bowlby's (1951) attachment theory (*see: Attachment*) has also influenced explanations of the developmental outcomes of abused children. The assumption is that infants develop an "internal working model" of the world that functions as a framework for further interaction with the interpersonal environment and involves expectations about the way the world functions. Abusive parenting is thought to lead to the development of an insecure-avoidant child,

likely to interpret neutral or even friendly behavior as hostile, and to show inappropriate aggressive behavior.

Other writers have speculated that physical abuse may alter a child's self-concept, attitudes, or attributional styles, which, in turn, may influence his or her response to later situations. Experiences of childhood physical abuse may lead to physiological changes in the child that, in turn, relate to the development of antisocial and aggressive behaviors. For example, as a result of being beaten continually, or as a result of the severe stress associated with intermittent physical abuse, a child might become "desensitized" to future painful or anxiety-provoking experiences. Such desensitization might result in a diminished physiological response to the needs of others and manifest traits such as callousness, lack of empathy, and lack of remorse or guilt. Relatedly, physical abuse may cause stress that, if occurring during critical periods in development, may alter normal brain chemistry leading to aggressive or withdrawn behaviors. Increasingly scholars are conducting research with nonhuman infants (rats or monkeys) using laboratory analogs to assess the effects of physical abuse on development. It is also possible that violent behavior is a genetic predisposition that is passed on from generation to generation (DiLalla & Gottesman, 1991).

Physically abused children may adopt maladaptive styles of coping. For example, characteristics such as a lack of realistic long-term goals, being conniving or manipulative, pathological lying, or glibness or superficial charm might begin as a means of coping with an abusive home environment. They may also withdraw or disengage from activities and relationships as a means of coping with anxiety, shame, or grief. Adaptations or coping styles that may be functional at one point in development (e.g., running away, avoiding an abusive parent, fighting to protect oneself or one's friends or family, using alcohol or drugs, or desensitizing oneself against feelings), may later compromise the person's ability to draw on and respond to the environment in an adaptive and flexible way.

Critical Questions

Important questions remain that challenge investigators and clinicians in the field:

What are the mechanisms whereby physical child abuse leads to short and long-term consequences?

What might account for the fact that not all physically abused children manifest negative consequences and, according to some studies, appear rather resilient?

To what extent does physical child abuse reflect a traumatic experience?

Or to what extent does physical abuse represent the extreme end of a continuum of physical discipline?

How do subcultural differences in normative standards of physical child abuse affect consequences for children?

Given that much research and clinical practice is based on a person's (client's) report of his or her childhood experiences, to what extent does the person's cognitive appraisal of the child's experience or experience with the events influence outcomes?

To what extent does the long-term impact of childhood physical abuse depend on characteristics of the community or practices of the community and justice and social service systems in which the child lived at the time of the abuse?

All of these questions require answers and those answers will inform interventions with parents to prevent child abuse from occurring and direct the treatment of child victims.

REFERENCES

- Bandura, A. (1973). *Aggression: A social learning analysis*. Englewood Cliffs, NJ: Prentice-Hall.
- Bowlby, J. (1951). *Maternal care and mental health*. Geneva, Switzerland: World Health Organization.
- Child Abuse Prevention and Treatment Act of 1974, Pub. L. No. 93-247, § 88, Stat 4, codified as amended by Keeping Children and Families Safe Act of 2003, Pub. L. No. 108-36, § 1(a), 117 Stat 800 (2003).
- DiLalla, L. F., & Gottesman, I. I. (1991). Biological and genetic contributors to violence: Widom's untold tale. *Psychological Bulletin*, 109, 125–129.
- Kempe, C. H., Silverman, F. N., Steele, B. F., Droegemueller, W., & Silver, H. K. (1962). The battered-child syndrome. *Journal of the American Medical Association*, 181, 17–24.
- U.S. Department of Health and Human Services, Administration on Children, Youth, and Families. (2007). *Child maltreatment 2005*. Washington, DC: U.S. Government Printing Office.
- Widom, C. S. (1999). Posttraumatic stress disorder in abused and neglected children grown up. *American Journal of Psychiatry*, 156, 1223–1229.
- Widom, C. S., DuMont, K. A., & Czaja, S. J. (2007). A prospective investigation of major depression disorder and comorbidity in abused and neglected children grown up. *Archives of General Psychiatry*, 64, 49–56.
- Belsky, J. (1993). Etiology of child maltreatment: A developmental-ecological analysis. *Psychological Bulletin*, 114, 415–434.
- Widom, C. S. (2000). Understanding the consequences of child abuse and neglect. In R. M. Reece (Ed.), *Treatment of child abuse* (pp. 339–361). Baltimore: Johns Hopkins University Press.

RECOMMENDED READINGS

- Belsky, J. (1993). Etiology of child maltreatment: A developmental-ecological analysis. *Psychological Bulletin*, 114, 415–434.
- Widom, C. S. (2000). Understanding the consequences of child abuse and neglect. In R. M. Reece (Ed.), *Treatment of child abuse* (pp. 339–361). Baltimore: Johns Hopkins University Press.

CATHY SPATZ WIDOM

City University of New York

See also: Abuse, Child Sexual; Child Maltreatment

ABUSE, CHILD SEXUAL

Child sexual abuse (CSA) is a particularly repugnant and pernicious form of child maltreatment and can result in multiple types of psychological and social harm, including psychological trauma. Once considered to be rare, the scope of the problem is now understood to be vast, with global estimates of 150 million girls and 73 million boys under the age of 18 being forced into sexual intercourse or other forms of sexual exploitation (World Health Organization, 2006). Varying degrees of awareness and acknowledgment of CSA and its

ramifications in terms of psychological trauma, both within and between nations, have made the assessment of its international public health importance and the response to its clinically pertinent consequences particularly difficult to mobilize and coordinate.

Clinical and Legal Frameworks

Although the task of defining CSA has sometimes proven elusive and controversial, there is reasonable consensus among clinicians and researchers that CSA can be thought of as having two distinct components that may potentially overlap. Those are “(a) forced or coerced sexual behavior imposed on a child, and (b) sexual activity between a child and a much older person, whether or not obvious coercion is involved” (Browne & Finkelhor, 1986, p. 66). A commonly used standard in the United States for defining “much older” is an age difference of 5 or more years, implying that the perpetrator may be an adult or an older child. Types of sexual activity involved can range from exposure and display (e.g., child pornography, lewd exposure by the perpetrator) to various forms of sexualized bodily contact, which might include genital or anal penetration.

Clinicians have found it useful to analyze the phenomenon of sexual abuse within a framework that differentiates the degrees to which sexual actions are abusive. Such a framework identifies differences between the offender to the victim, in terms of three factors: (1) a power differential (i.e., the extent to which the offender controls/or has more power than the victim); (2) a knowledge differential (i.e., the extent to which the offender has a more sophisticated understanding of the act or is developmentally more advanced than the victim); and (3) a gratification differential (i.e., the extent to which the primary purpose is sexual gratification of the offender versus mutual gratification of both persons). The severity of sexual abuse increases as the extent to which the potentially abusive sexual acts involve the offender having greater power, knowledge, and gratification than the victim (Faller, 1993).

In addition to clinical frameworks, an equally relevant but distinguishable and informative perspective is the legal framework for defining CSA that has been developed in some countries. While the age of consent for sexual involvement may vary with and between countries, sexual relations with a child below the age of consent are illegal in the United States and in most of the Western industrialized nations. Most, if not all, nations and cultures set limits on sexual contact with children, though these may not always take the form of clearly defined legal statutes. In the United States, legal principles for determining the legality of sexual activity with children can be found in both civil and criminal law, namely the legal statutes that define the conditions required for child protection or welfare, and the legal statutes that prohibit criminal behavior, respectively. Violation of these laws can result in substantial penalties, depending on the age of child, the level of physical force or harm involved in sexually abusive acts, the relationship between victim and offender, and the type of sexual act (Faller, 1993).

CSA and Psychological Trauma

Psychological trauma by definition represents the overwhelming or flooding of an individual's capacity to cope with the emotions, thoughts, and somatic experiences associated with a event(s) that involve either the threat of death or a violation of the person's bodily integrity (American Psychiatric Association, 2000), can be particularly debilitating during childhood because this is a developmental period in which psychological and physiological defenses are rapidly developing and relatively immature. Given the dynamic and sensitive nature of child development, a combination of intrinsic (e.g., individual child characteristics) and extrinsic (e.g., different types of stressful events; social support) factors influence the extent to which a particular event or circumstance is experienced by the child as psychologically traumatic. The severity of psychological trauma caused by CSA therefore depends on factors such as the power and knowledge

differentials described above, but also on other factors including (Cicchetti, 2004):

- Whether the CSA involved a single sexual act or series of sexual acts or encounters
- The child's age (with younger children generally more vulnerable than older children; although this is a matter of degree, and sexual acts or encounters that involve a power, knowledge, and gratification differential almost invariably are considered to be psychologically traumatic for children of all ages)
- The presence of an attachment relationship between the child and the perpetrator (most notably in the case of incest by a parent or primary guardian) that therefore compromises the child's ability to develop a secure sense of attachment and trust in caring relationships (*see: Attachment; Betrayal Trauma; Complex Posttraumatic Stress Disorder*)
- The frequency and chronicity (length of time over which they occurred) of the abusive acts, with greater frequency and chronicity usually more traumatic
- The severity of bodily violation

The psychological impact of CSA can include symptoms that are generally associated with posttraumatic stress disorder (PTSD). However, CSA also may lead to a wide range of other behavioral and emotional problems or symptoms that have been described as the result of the "traumagenic dynamics" of CSA. These problems include excessively sexualized behavior, a profound sense of powerlessness and stigma that can cause or exacerbate affective or anxiety disorders, and a sense of betrayal that can compromise the child's ability to develop safe and trusting relationships (Finkelhor, 1990). Some experts contend that CSA is not so much an event as it is a chronic situation, referring to the observation that CSA is often a recurring process subsumed in a familiar relationship with a caregiver or family member or responsible adult (Finkelhor, 1990). Particular to CSA, the notion of *complex*

PTSD (*see: Complex Posttraumatic Stress Disorder*) has been put forth to more fully capture the nature of CSA-associated problems of emotional arousal and regulation, somatization (i.e., stress-related breakdowns in bodily health and functioning), changes in perception of self (such as viewing oneself as permanently damaged), changes in relationship patterns (such as avoidance or excessive seeking of intimacy, and extreme degrees of conflict), and a loss of sustaining beliefs or spiritual faith (Herman, 1997).

Various dimensions of psychological trauma associated with CSA have been conceptualized and highlighted over the past 2 decades, beginning with Finkelhor and Browne's traumagenic dynamics model (1985). This model continues to be one of the widely used frameworks for describing the harmful effects of CSA, and has fueled multiple programs of research and clinical applications (Banyard et al., 2001). The model outlines four core dimensions of trauma experienced by the CSA victim, namely (1) traumatic sexualization, (2) betrayal, (3) stigmatization, and (4) powerlessness. *Traumatic sexualization* associated with CSA has been found to impact a child's sexuality either through hypersexual behaviors (i.e., an extremely early age of onset and excessive involvement in sexual behavior) or through avoidance and negative sexual encounters (Meston, Rellini, & Heiman, 2006). *Betrayal* trauma may have a profound traumatic effect because it signifies a breakdown of trust in caretaking relationships and has been shown to be linked to anger and acting out behaviors, and significant difficulties in relationships (*see: Betrayal Trauma*). *Stigmatization*, also referred in the literature as "damaged goods syndrome" (Jennings, 2003), is manifested in feelings of guilt and beliefs centered on self-blame or the assumption that other persons would blame the victim for the abuse and for the consequences of disclosure (such as for legal charges being brought against a perpetrator of CSA, or shame and embarrassment experienced by the family, or for the child her- or himself or siblings being taken from the family by child protective services agencies).

These beliefs and feelings related to betrayal and stigma may be expressed or coped with through behaviors that are self-destructive or risk-taking, such as self-mutilation, suicidal attempts, substance abuse, and other provocative behaviors that elicit punishment.

The fourth traumagenic dynamic, namely *powerlessness*, is characterized by feelings of vulnerability and helplessness, balanced against aggressive impulses to gain control of the situation. Feelings of acute helplessness, as a result of the belief that one is powerless to stop powerful other persons from inflicting violation and harm, can lead to avoidant and dissociative behaviors, such as phobias, eating disorders, and revictimization. A sense of powerlessness also can lead the CSA survivor to develop a pervasive desire to control others and to prevail in any event or experience that is perceived as a personal threat or challenge, which can lead to identification with the aggressor (i.e., admiring or attempting to model oneself after the perpetrator of abuse or other supposedly powerful persons), and in some cases to engaging in acts that involve the exploitation of others. Although some perpetrators of CSA have themselves been victims of CSA in their childhoods, most CSA survivors do not ever become perpetrators of CSA. They may however struggle emotionally with thoughts and feelings that involve a wish to be able to turn the tables and be the “powerful” person in control in relationships, which can lead to many conflicts and difficulties in important relationships such as marriage or parenting.

Another approach to examining the relationship between psychological trauma and CSA has been to focus on various aspects of the CSA experience. Examples of these aspects of the abuse include the type of abusive act, circumstances surrounding the abuse, the duration of the abusive pattern, the age of the child when the abuse began (onset) and when it ended (offset), characteristics of the perpetrator (e.g., age, relationship to the victim, the number of perpetrators), and characteristics of the victim (e.g., age while abuse was occurring, gender, education level and intellectual

abilities, extent and type of social support during and after the abuse), and to relate these dimensions to various trauma-related outcomes (Manly, Kim, Rogosch, & Cicchetti, 2001). Such models have sought to explain how the characteristics of the abuse, the perpetrator, and the victim together influence the type, magnitude, and persistence or patterns of traumatic stress problems in the time since the abuse began. In general, rather than any one dimension standing out in predicting traumatic impact, research suggests that CSA may best be viewed as a multidimensional construct. Depending on the specific nature of the abuse and the characteristics and relationships of the perpetrator(s) and victim, CSA can have a range of differential effects on the victim’s emotional and behavioral functioning and on developmental outcomes such as the child’s ability to achieve expectable physical, psychological, educational, and social milestones (*see: Adolescence; Child Development*).

Aftereffects of CSA during Childhood

Great strides have been made in our understanding of CSA since the 1970s, when acknowledgment and awareness of the issue in the United States and a few other Western industrialized nations began to fuel research and clinical knowledge in this domain. The first 2 decades largely involved retrospective studies of adults abused as children, culminating in the landmark report of the Adverse Childhood Experiences Study (ACES; Felitti et al., 1998) in which more than 20,000 adults in a U.S. health-care organization were surveyed concerning their stressful and traumatic childhood experiences and their current psychological and medical health. Exposure to adversity in childhood, including CSA, was found to be associated with as much as a 20-fold increase in the risk of serious psychological and medical disorders.

More recent research in the past decade has included prospective longitudinal studies (*see: Research Methodology*) of sexually abused children who were surveyed over the course of

their childhoods into adolescence and adulthood (Putnam, 2003). These and other recent studies have focused on children at different ages and their ecological contexts, thus enabling a developmental lens to be applied to the issue (Murthi & Espelage, 2005). The recent developmental focus has shed light on the initial or short-term effects in the aftermath of CSA, typically defined as within 2 years of the termination of the abuse. These effects can take the form of internalizing or externalizing problems. Internalizing problems include sleep disturbances, eating disorders, severe anxiety and phobias, depression and suicidality, dissociative disorders, guilt, and shame. Externalizing problems include extreme degrees of, or difficulty in managing, anger, hostility, impulsiveness, risk-taking, and distractibility, which may take the form of oppositional defiant disorder, conduct disorder, substance use disorders, or serious problems with the law, social isolation, educational and work failure, and residential instability and homelessness. CSA is not clearly the cause of these problems, but has been shown to contribute to the person's risk of developing these significant difficulties and the severity of the symptoms or problems.

Research has shown that some sequelae (i.e., aftereffects) of CSA are more prevalent at certain ages than others. Of note, internalizing symptoms are particularly stark for preschoolers, which may be explained in part by the concept of imminent justice, whereby very young children may be particularly likely to view CSA as the negative outcome of their own misbehavior (Quas, Goodman, & Jones, 2003). Other internalizing problems commonly associated with CSA among preschoolers have been anxiety, nightmares, and inappropriate sexual behaviors (Kendall-Tackett, Williams, & Finkelhor, 1993). Although young children who are victims of CSA may show problems with anger, aggression, difficulties with attention and impulsivity, these externalizing problems are particularly likely to occur among school-age children, problems of hyperactivity, regressive behaviors, and learning difficulties often are observed in the wake of CSA (Bromberg & Johnson, 2001). Social stigmatization associated

with CSA can result in withdrawal, aggression, and negative self-perceptions. Adolescents who have experienced CSA are at risk for developing PTSD, depression, suicidal or self-injurious behaviors, substance abuse, running away, school problems, and legal problems (Putnam, 2003).

Aftereffects of CSA during Adulthood

For a variety of reasons, including that CSA often goes undetected and undisclosed during childhood, retrospective studies of adults who were abused as children are far more common than prospective studies that begin in childhood. Long-term sequelae of CSA in adulthood include developmental disabilities, depression, alexithymia, PTSD, sexual dysfunction, eating disorders, substance abuse, homelessness, problems in interpersonal relationships, promiscuity, and avoidance of physical intimacy (Kendler et al., 2000; Murthi & Espelage, 2005). In addition, there has been recent interest on the phenomenon of revictimization among adult survivors of CSA (Messman-Moore, Long, & Siegfried, 2000). It has been found that childhood psychological trauma, especially CSA, may make the adult survivor particularly vulnerable for further victimization, setting off traumatic "chain reactions" across the life span (Banyard et al., 2001). Results from empirical studies point to the importance of understanding the interconnectedness between these multiple victimizations in assessing the overall impact of CSA on adult survivors. Thus, when children who have been victimized by sexual abuse encounter other psychological traumas in childhood, adolescence, or adulthood, this revictimization appears to have a "cumulative" adverse effect in terms of making them more likely to experience a wide range of more severe and persistent psychological and medical problems than other persons who experienced no additional psychological trauma beyond CSA or who did not suffer CSA but have experienced other psychological traumas (Ford, Stockton, Kaltman, & Green, 2006) (*see: Retraumatization*).

There is no definitive answer to the question of why CSA victims are at risk for further psychological trauma, but the research does not support the idea that CSA victims “cause” or “seek” additional traumatic experiences. Instead, it seems more likely that the adversities that often (but not always) co-occur with CSA, including family problems, social isolation, and living with limited socioeconomic resources, may lead to the increased likelihood of retraumatization rather than any characteristic of the CSA victim per se or of the experience of being victimized by CSA. It should also be noted that, while CSA is associated with an array of psychopathological consequences, a considerable proportion of sexually abused children demonstrate adaptive outcomes as they mature, albeit with potentially different affective-cognitive configurations or psychological adaptations than nonabused children.

Treatment Considerations

While treatment goals vary in accordance with the client’s clinical presentation and the treatment modality being employed, there are certain therapeutic goals that are consistently acknowledged as salient to the successful treatment of the aftereffects of CSA. In the immediate aftermath of CSA, short-term goals for the sexually abused child include providing safety and containment within the therapeutic relationship, along with helping the child to distinguish between healthy and destructive coping mechanisms (see: **Child Abuse, Cognitive Behavior Therapy**). Another often cited goal is to clearly identify for the victim that the perpetrator is responsible for the sexual abuse, in order to help the child understand that she or he is not to blame and to begin to therapeutically address the “traumagenic” beliefs that may result from CSA (Finkelhor, 1990).

Effectiveness of treatments for children who were victimized by CSA, above and beyond therapist characteristics and competencies, has been shown to hinge on the therapist helping the child to develop a solid grasp of how and why sexual abuse occurred, accompanied by a thorough assessment of the potential mediating

or moderating roles played by individual, environmental, and CSA-event related factors (Hetzel-Riggin, Brausch, & Montgomery, 2007). Additionally, the heterogeneity of internalizing and externalizing problems associated with CSA complicate treatment selection and outcome measurement. Controversies surrounding treatment referrals, such as whether it is appropriate to therapeutically treat apparently asymptomatic children, can be partly attributed to the nature of child sexual abuse, which is an experience rather than a syndrome or a disorder, and partly due to a noticeable dearth of CSA studies involving the systematic identification of what constitute clinically significant symptoms, and the empirical validation of treatment methods. Current best practice is to provide thorough ongoing screening or assessment of the asymptomatic child and family’s functioning, and education for the child and family about the expectable aftereffects of CSA in a manner that provides them with hope for a positive recovery but awareness of signs of problems that might warrant therapeutic treatment (Ford & Cloitre, in press).

During adulthood, CSA victims may struggle with difficulties in intimate relationships. It has been found that sexually abused adult patients in therapy are several times more likely than nonabused patients to refuse sexual activity at one extreme, or to show promiscuity at the other (Linden & Zehner, 2007). An important therapeutic goal is to help the adult CSA survivor reestablish appropriate interpersonal boundaries, including the clarification and adherence to therapeutic boundaries between the client and the therapist. Another central goal in adult therapy for CSA survivors is for the individual to recognize herself or himself as a survivor (rather than only a victim) of abuse, and to overcome negative and potentially self-destructive behaviors. This is critical given empirical data from meta-analytic studies that adults who have been CSA survivors display a threefold increase in attempted suicidal behavior, citing reasons of despair, guilt, and self-blame (Linden & Zehner, 2007). The presence of other potential long-term sequelae of CSA, such as substance abuse, eating

disorders, and revictimization, make it necessary to design treatments and evaluate the efficacy of treatments for adult CSA survivors with careful consideration of the full range of potential problems that may need to be addressed in order to help the individual recover fully.

In addition to formal psychotherapy (and medication therapy for PTSD, depression, and other associated problems; *see: Pharmacotherapy, Child*), there are other options that have been used to combat the enduring negative consequences of CSA. Many adult survivors turn to self-help books, manualized programs, and support or educational groups aimed at cultivating self-validating behaviors and healthy coping skills. With the rise of the Internet, virtual communities comprised of adult survivors of sexual abuse have become increasingly popular as they foster the sharing of individual stories while still retaining the anonymity of the individual members. Spiritual healing is yet another manner in which adult survivors have sought to rebuild shattered trust through the power of faith and communal support. No matter what the modality, relief from the negative repercussions of CSA has typically involved a delicate balance between disclosure and expression of the horrific experience(s) with the maintenance of the emotional distance from those troubling memories that is needed in order to reestablish a sense of personal safety, to rebuild trust through positive relationship experiences, and to engage in self-affirming behaviors (Harvey, 1996).

Prevalence, Culture, and Attitudes

In several communities around the world, culture and attitudes have been found to play a key role in the extent to which CSA is understood, acknowledged, and addressed. In the United States during 2005, 9% of the 899,000 substantiated cases of child maltreatment were cases of child sexual abuse. Many researchers have argued that these statistics are subject to underreporting, given that CSA cases are often well-hidden within the family context due to shame, stigmatization, fear of prosecution,

fear of loss of close relationships, and victims' unfounded but common beliefs implicating themselves as having been at least partially responsible for their own abuse. Global estimates of sexual violence against children point to the perpetrator as typically a member of the child's family circle. Similarly, in the United States, a majority of perpetrators in substantiated CSA cases are parents or other relatives. Nevertheless, CSA also may be perpetrated by other trusted adults (e.g., religious leaders, teachers, coaches, members of the extended family) or by strangers.

Accepted cultural practices may serve to increase the risk of CSA in many countries. To uncover these cultural nuances and to more fully comprehend the extent of violence against children, the United Nations recently commissioned an overarching study that involved the participation of 133 governments, several hundred organizations, and the unprecedented and substantive participation of children around the world expressing their views on violence as experienced by them (Pinheiro, 2006). The study found that the absence of legally established minimum ages for sexual consent and marriage practices can expose children to substantial partner violence, while harmful traditional practices such as female genital mutilation, violent initiation rites, and dowry-related violence in many cultures may affect children disproportionately, due to their dependent and powerless status. These practices, even if not considered sexual abuse within specific cultural contexts, are likely to be psychologically traumatic. Thus, while the definition of CSA must take into account not only a scientific/clinical perspective but also the beliefs and practices of specific cultures, the traumatic impact of sexual harm to children is a universal clinically/scientifically documented phenomenon.

Many communities around the world currently find themselves battling with the spiraling societal consequences of denial or inadequate attention to the problem of CSA. Results from a participatory action research project conducted by a leading child service agency in Northern Tanzania found linkages

between rampant sexual abuse and primary school dropouts, truancy, and migration to the streets (Mkombozi Center for Street Children, 2006). In the latter study, perpetrators of CSA included older students, parents, and local community members. Further, the study pointed to widespread denial of the issue among government officials, school authorities, and parents. Despite considerable school-based efforts in some regions of the world, cultural sensitivity on the part of the child's family can greatly hinder the appropriate design and effectiveness of psychoeducational services. In a study conducted across seven elementary schools in China, nearly half of all parents surveyed expressed concern that CSA preventive education might result in their children knowing "too much about sex" (Chen, Dunne, & Han, 2007). Moreover, many parents themselves were found to lack knowledge about CSA, especially about the psychological consequences of CSA, the possibility of sexual abuse of boys, and about perpetrator characteristics. Hence, there is considerable variability in CSA knowledge worldwide, due to differing contextual and cultural factors that can substantially influence reporting, prevention, and intervention efforts.

Conclusion

The sexual abuse of children is a well-established risk factor that is associated with a host of psychosocial problems, including symptoms associated with psychological trauma and PTSD. While some children are remarkably resilient to this form of exploitation, most are likely to suffer substantial distress and some will develop clinically significant symptoms of psychiatric disorders. The ways in which children adapt to experiencing sexual abuse in childhood may include interruptions and derailing of normal development that negatively affect their sense of personal identity and their relationships. Because there are culturally diverse perspectives on the sexual roles that are or are not permissible for children and concerning what is acceptable with regard to educating children and families about sexuality and

its place in normal social development, there is also disagreement on what constitutes sexual abuse in distinction to local variations in sexual customs and familial practices. Prevention of sexual abuse is the most ideal form of intervention, but because of the privacy and intimacy of human sexuality, inappropriate and illegal sexual relationships can be readily hidden, especially if those involved in the sexual conduct are motivated to prevent disclosure due to shame, guilt, self-reproach, or the threat of prosecution or violence.

Relief from the enduring negative consequences of sexual abuse can take many forms, including personally resilient adaptation, spiritual forms of healing, self-help groups, and psychotherapy. There are a variety of models for therapeutic treatment of children who have been sexually abused, and the effectiveness of these treatments is greatly influenced by the extent to which due consideration is given to the heterogeneity of the secondary problems associated with CSA as well as the potential mediating and moderating roles played by the unique risk and protective factors associated with a particular child.

Many people who were sexually abused in childhood do not seek treatment until sometime in adulthood, and there are also various approaches to providing therapy for this population. The effectiveness of therapy in adulthood for the aftereffects of CSA may be complicated by the presence of other long-term problems (*see: **Complex Posttraumatic Stress Disorder***). It is also noteworthy that a considerable number of adult survivors of CSA demonstrate strongly positive psychological adaptations. Thus, it appears that more research is needed to clarify the heterogeneity of presentations, identification of clinically significant symptoms, the effectiveness of various treatment elements and the differential outcomes associated with CSA. What is clear is that without a move toward more widespread public awareness, acknowledgment, and psychoeducation regarding the issue worldwide, CSA will remain a clandestine issue that negatively impacts millions of children worldwide.

REFERENCES

- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- Banyard, V., Williams, L., & Siegel, J. (2001). The long-term mental health consequences of child sexual abuse: An exploratory study of the impact of multiple traumas in a sample of women. *Journal of Traumatic Stress, 14*, 697–715.
- Bromberg, D., & Johnson, B. (2001). Sexual interest in children, child sexual abuse, and psychological sequelae for children. *Psychology in the Schools, 38*, 343–355.
- Browne, A., & Finkelhor, D. (1986). Impact of child sexual abuse: A review of the research. *Psychological Bulletin, 99*, 66–77.
- Chen, J., Dunne, M. P., & Han, P. (2007). Prevention of child sexual abuse in China: Knowledge, attitudes, and communication practices of parents of elementary school children. *Child Abuse and Neglect, 31*, 747–755.
- Cicchetti, D. (2004). An odyssey of discovery: Lessons learned through three decades of research on child maltreatment. *American Psychologist, 59*, 731–741.
- Faller, K. C. (1993). *Child sexual abuse: Intervention and treatment issues*. McLean, VA: U.S. Department of Health and Human Services, Circle Solutions.
- Felitti, V., Anda, R., Nordenberg, D., Williamson, D., Spitz, A., Edwards, V., et al. (1998). Relationship of childhood abuse and household dysfunction to many of the leading causes of death in adults. *American Journal of Preventive Medicine, 14*, 245–258.
- Finkelhor, D. (1990). Early and long-term effects of child sexual abuse: An update. *Professional Psychology: Research and Practice, 21*, 325–330.
- Finkelhor, D., & Browne, A. (1985). The traumatic impact of child sexual abuse: A conceptualization. *Journal of Orthopsychiatry, 55*, 530–541.
- Ford, J. D., & Cloitre, M. (in press). Psychotherapy for children and adolescents with complex traumatic stress disorders: Overview and provisional practice principles. In C. Courtois & J. D. Ford (Eds.), *Complex traumatic stress disorders: An evidence based clinician's guide* (Chapter 2). New York: Guilford Press.
- Ford, J. D., Stockton, P., Kaltman, S., & Green, B. L. (2006). Disorders of extreme stress (DESNOS) symptoms are associated with interpersonal trauma exposure in a sample of healthy young women. *Journal of Interpersonal Violence, 21*, 1399–1416.
- Harvey, M. (1996). An ecological view of psychological trauma and trauma recovery. *Journal of Traumatic Stress, 9*, 3–23.
- Herman, J. (1997). *Trauma and recovery*. New York: Basic Books.
- Hetzel-Riggin, M., Brausch, A., & Montgomery, B. (2007). A meta-analytic investigation of therapy modality outcomes for sexually abused children and adolescents: An exploratory study. *Child Abuse and Neglect, 31*, 125–141.
- Jennings, L. P. (2003). *Damaged goods: Once molested, then a predator*. Bloomington, IN: Authorhouse.
- Kendall-Tackett, K., Williams, L., & Finkelhor, D. (1993). Impact of sexual abuse on children: A review and synthesis of recent empirical studies. *Psychological Bulletin, 113*, 164–180.
- Kendler, K., Bulik, C., Silberg, J., Hettema, J., Myers, J. P., & Prescott, C. A. (2000). Childhood sexual abuse and adult psychiatric and substance abused disorders in women. *Archives of General Psychiatry, 57*, 953–959.
- Linden, M., & Zehner, A. (2007). The role of childhood sexual abuse (CSA) in adult cognitive behavior therapy. *Behavioral and Cognitive Psychotherapy, 35*, 447–456.
- Manly, J. T., Kim, J. E., Rogosch, F. A., & Cicchetti, D. (2001). Dimensions of child maltreatment and children's adjustment: Contributions of developmental timing and subtype. *Development and Psychopathology, 13*, 759–782.
- Messman-Moore, T., Long, P., & Siegfried, N. (2000). The revictimization of child sexual abuse survivors: An examination of the adjustment of college women with child sexual abuse, adult sexual assault, and adult physical abuse. *Child Maltreatment, 5*, 18–27.
- Meston, C., Rellini, A., & Heiman, J. (2006). Women's history of sexual abuse, their sexuality, and sexual self-schemas. *Journal of Consulting and Clinical Psychology, 74*, 229–236.
- Mkombozi Center for Street Children. (2006). *Culture and attitude play a key role in child sexual abuse*. Retrieved December 7, 2007, from www.mkombozi.org/publications/press_release/2006_08_23_press_release_sexual_abuse.pdf.

- Murthi, M., & Espelage, D. L. (2005). Childhood sexual abuse, social support, and psychological outcomes: A loss framework. *Child Abuse and Neglect*, 29, 1215–1231.
- Pinheiro, P. S. (2006). *United Nations study on violence against children: Report of an independent expert*. New York: United Nations.
- Putnam, F. (2003). Ten year research update review: Child sexual abuse. *Journal of the American Academy of Child and Adolescent Psychiatry*, 42, 269–278.
- Quas, J., Goodman, G., & Jones, D. (2003). Predictors of attributions of self-blame and internalizing behavior problems in sexually abused children. *Journal of Child Psychology and Psychiatry*, 44, 723–736.
- World Health Organization. (2006). *Global estimates of health consequences due to violence against children*. Geneva, Switzerland: Author.

APARNA RAO

Fielding Graduate University

GILBERT REYES

Fielding Graduate University

JULIAN D. FORD

University of Connecticut School of Medicine

See also: Abuse, Child Physical; Child Maltreatment

ACCEPTANCE AND COMMITMENT TREATMENTS

See: Meditation

ACCIDENT TRAUMA

See: Motor Vehicle Collisions

ACUTE STRESS DISORDER

Acute stress disorder (ASD) is a relatively recent diagnosis that was introduced in *DSM-IV* in 1994 (American Psychiatric Association, 1994). This diagnosis was introduced to describe posttraumatic stress reactions that occur in the initial month after a traumatic event (for a review, see Bryant & Harvey, 2000). It was also intended to identify people shortly after the trauma who are

likely to subsequently develop chronic posttraumatic stress disorder (PTSD). The disorder is present when a person has a fearful response to experiencing or witnessing a threatening event, displays at least three dissociative symptoms, one reexperiencing symptom, marked avoidance, marked anxiety or increased arousal, has significant distress or impairment, and lasts for at least 2 days and a maximum of 4 weeks, after which time a diagnosis of PTSD may be considered.

Aside from ASD's shorter symptom duration, the major difference between the ASD and PTSD criteria is the former's emphasis on acute dissociation. Specifically, the ASD diagnosis requires that people display at least three of the following dissociative symptoms: (a) a subjective sense of numbing or detachment, (b) reduced awareness of their surroundings, (c) derealization, (d) depersonalization, or (e) dissociative amnesia. This requirement was introduced because of a theoretical model that proposes that acute dissociation results in fragmented memories and affect being encoded at the time of trauma, and that these responses impede subsequent processing of traumatic memories and adaptation of traumatic stress.

Does Acute Stress Disorder Predict Posttraumatic Stress Disorder?

There is overwhelming evidence that whereas the majority of trauma survivors will be distressed in the initial weeks after trauma exposure, the majority of people will adapt in the following 3 to 6 months. This pattern poses a challenge for the ASD diagnosis because it intends to discern between those trauma survivors who are experiencing a transient stress reaction from those who will develop PTSD.

Since the introduction of the ASD diagnosis, there has been a series of prospective studies that have assessed ASD in adults and children in the initial month after trauma, and subsequently assessed participants for PTSD at increased variable time periods after the trauma. A significant proportion of studies indicate that approximately three-quarters of trauma survivors who display ASD subsequently develop PTSD. Although this pattern

appears to show promising predictive power of the ASD diagnosis, there is a less encouraging pattern in terms of the people who develop PTSD and who do not initially meet ASD criteria. In terms of people who eventually developed PTSD, approximately half of those met criteria for ASD in the initial month.

This convergence across studies suggests that whereas the majority of people who develop ASD are at high risk for developing subsequent PTSD, there are also many other people who will develop PTSD who do not initially meet ASD criteria. It seems that the major reason for people who are high risk for PTSD not meeting ASD criteria is the requirement that dissociative symptoms be displayed. It is possible that there are multiple pathways for developing PTSD, and that the initial course may not involve dissociative responses.

Treatment of Acute Stress Disorder

The psychological treatment of choice for ASD is cognitive behavior therapy (CBT), and typically comprises psychoeducation, anxiety management, cognitive restructuring, imaginal and *in vivo* exposure, and relapse prevention. Psychoeducation provides information about common symptoms following a traumatic event, legitimizes the trauma reactions, and establishes a rationale for treatment. Anxiety management techniques provide individuals with coping skills to assist them to gain a sense of mastery over their fear, to reduce arousal levels, and to assist the individual when engaging in exposure to the traumatic memories. Anxiety management approaches often include breathing retraining, relaxation skills, and positive self-talk. Prolonged imaginal exposure requires the individual to vividly imagine the trauma for prolonged periods—typically occurring for at least 50 minutes—and is usually supplemented by daily homework exercises. Most exposure treatments supplement imaginal exposure with *in vivo* exposure that involves live graded exposure to the feared trauma-related stimuli (e.g., gradually confronting the feared stimuli associated with the trauma, such as returning to a physical

scene similar to where the trauma occurred). Cognitive restructuring, which is based on the premise that maladaptive appraisals underpinning the maintenance of PTSD involves teaching patients to identify and evaluate the evidence for negative automatic thoughts, as well as helping patients to evaluate their beliefs about the trauma, the self, the world, and the future in an evidence-based manner. The duration of CBT for ASD is typically five sessions. There are numerous controlled trials that attest to the efficacy of CBT for treating PTSD, and approximately 80% of people who complete treatment do not develop PTSD.

Future of Acute Stress Disorder

It is likely that ASD will not survive in the publication of *DSM-V*. The diagnosis can be criticized because (a) the primary role of the ASD diagnosis is to predict another diagnosis, (b) distinguishing between two diagnoses that have similar symptoms primarily on the basis of the duration of the symptoms is not justified, and (c) there is insufficient evidence to support its role as a reliable predictor of subsequent PTSD. More accurate prediction of PTSD will come from a broader range of acute reactions, including biological and cognitive responses, rather than a diagnostic category. Despite the limitations of the ASD diagnosis, the introduction of a diagnostic category has stimulated much research and increased our understanding of acute stress reactions. Most importantly, it has raised the possibility of secondary prevention of PTSD by providing early interventions to those who are at high risk for developing PTSD. Through more rigorous prospective study of acute and chronic reactions to trauma, improved formulae can be developed to identify those people who are most likely to need early intervention after trauma.

REFERENCES

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Bryant, R. A., & Harvey, A. G. (2000). *Acute stress disorder: A handbook of theory, assessment, and*

treatment. Washington, DC: American Psychological Association.

Harvey, A. G., & Bryant, R. A. (2002). Acute stress disorder: A synthesis and critique. *Psychological Bulletin*, 128, 886–902.

RICHARD A. BRYANT
University of New South Wales

See also: Anxiety Management Training; Cognitive Behavior Therapy, Adult; Early Intervention; Exposure Therapy, Adult; Exposure Therapy, Child; Posttraumatic Stress Disorder

ADJUSTMENT DISORDERS

An adjustment disorder (AdjD) is a maladaptive reaction to identifiable stressors or to changes in life circumstances and is thus similar in some respects to posttraumatic stress disorder (PTSD), which is also a reaction to stressful life experiences. The symptoms of AdjD emerge within 3 months of the stressor's onset, and should not persist for more than an additional 6 months. Symptoms may include a wide variety of impairments in social or occupational functioning, as well as maladaptive extremes of anxiety and depression, and impulse control problems. If the symptoms would also satisfy the diagnostic criteria for another clinical (Axis I) disorder, then the other diagnosis should supersede that of AdjD and AdjD should not be diagnosed. There are various subtypes of AdjD, including types with depressed mood, disturbance of conduct, mixed disturbance of emotions and conduct, and an unspecified subtype.

Diagnosing AdjD has been controversial because it is loosely defined, has inadequate support for its validity, and has been neglected by academic scholars and researchers, with only little attention in psychiatry textbooks and very few empirical studies (Strain & Diefenbacher, 2008). However, the AdjD diagnosis is retained because of its usefulness as a clinical concept. In the general population, roughly 1 out of 200 people (0.5%) would qualify for a diagnosis of AdjD, whereas 12% to 20% of patients in medical settings receive this diagnosis.

Researchers proposed grouping AdjD into the (new) category of "Reactions to Severe Stress" that also includes acute stress disorder, posttraumatic stress disorder, and prolonged grief disorder (Maercker, Einsle, & Kollner, 2007). Based on this categorization, AdjD is a consequence of a stressful life event that differs from a traumatic event by its extent of threat to life or physical integrity. Individual predisposition or vulnerability seem to play an important role in the risk of occurrence and manifestation although systematic research is still lacking.

Primary treatment goals for AdjD are to relieve symptoms and re-achieve a level of adaptation by a broad range of psychological (i.e., crisis intervention) or psychosocial (i.e., workload reduction or restructuring) interventions. Treatments include individual psychotherapy and short-term medication led by best practice standards due to the lack of formal treatment guidelines.

REFERENCES

- Maercker, A., Einsle, F., & Kollner, V. (2007). Adjustment disorders as stress response syndromes. *Psychopathology*, 40, 135–146.
- Strain, J. J., & Diefenbacher, A. (2008). The adjustment disorders: The conundrums of the diagnoses. *Comprehensive Psychiatry*, 49, 121–130.

ANDREAS MAERCKER
University of Zurich

See also: Acute Stress Disorder; Posttraumatic Stress Disorder

ADOLESCENCE

Adolescence, the age period approximately from 12 to 18 years old, is a time of rapid change and growth biologically, psychologically, and socially for children. Adolescence often is a time of physical and emotional turmoil, yet is also a period in which critical accomplishments that can shape the individual's adult life can either occur or be thwarted, including: personality development, identity consolidation, peer group formation and social role definition, emergence of sexuality in the form of

interest and exploratory activities, and consolidation of knowledge, skills, and goals in education, work, and avocational/recreational life pursuits. If psychological trauma has occurred earlier in an adolescent's life and has left the imprint of problems with traumatic stress reactions (such as posttraumatic stress disorder or PTSD, or symptoms of other anxiety disorders, depression, or dissociative conduct, eating, or substance use disorders), the normal adolescent emotional/relational turmoil is greatly amplified and those crucial developmental attainments may be hindered, interrupted, altered, or blocked. If psychological trauma occurs during adolescence, the youth is likely to experience stress reactions that also may interfere with the complicated psychosocial, educational/vocational, and self-identity development tasks of adolescence. Following a brief overview of the biological and psychosocial changes that occur normally in adolescence, this entry describes how psychological trauma impacts adolescents.

Adolescent Biological and Psychosocial Development

Although brain development occurs most intensively and rapidly prenatally and in early childhood, the central nervous system (CNS) continues to grow and reshape itself throughout childhood, with a second peak of growth and reorganization in late childhood and early adolescence (Anderson, 2003). CNS areas grow and change at different rates (Anderson, 2003), with deeper (e.g., brainstem, hippocampus) more posterior (e.g., occipital cortex) structures maturing earliest, and the outer front-most area, the prefrontal portion of the frontal cortex (PFC), peaking in growth in early adolescence (Giedd et al., 1999; Kanemura, Aihara, Aoki, Araki, & Nakazawa, 2003). From childhood into adolescence, areas of the brain cortex that are responsible for sensory and perceptual processes appear to shrink and become more efficient (Sowell, Thompson, Tessner, & Toga, 2001), while the brain cortex areas activated by rewarding experiences (particularly the middle and lower portions of the PFC, the medial

and orbital PFC; May et al., 2004) appear to grow in size and complexity (Giedd et al., 1999; Kanemura et al., 2003). These brain changes are consistent with the shift from early to later childhood away from impulsiveness and self-protectiveness toward ego control or inhibition control (Eisenberg et al., 1995). Specific areas in the medial PFC that are required for such mature self-control include the anterior portion of the cingulate cortex, which appears to monitor potential problems with positive or negative outcomes (e.g., conflict, errors) and to signal the upper and side areas of the PFC (dorsolateral PFC) to become engaged when a discrepancy between intended and actual outcomes requires conscious evaluation and effortful correction of behavior (Eisenberger, Lieberman, & Williams, 2003, p. 291). Neural pathways from the orbital PFC reduce reactivity by inhibiting neural activation in the locus coeruleus, amygdala, and hippocampus. The dorsolateral PFC seems to exert preemptive control (i.e., resulting in reactive responding; Matsumoto, Suzuki, & Tanaka, 2004), while the orbital PFC appears to give rise to self-awareness of meaningful and adaptively useful connections between emotions, goals, and behavioral options.

In these areas of the brain's cortex and limbic system, another transitional period occurs late in preadolescence and early in adolescence, in which neuronal growth and shaping in these areas of the brain accompanied by an increase in the creation of the protective covering for neural connections (the myelin sheath) is put into place rapidly in brain areas that are involved in higher-order symbolic thought and memory (e.g., hippocampus; Benes, Turtle, Khan, & Farol, 1994). This "paving over" of the formerly rudimentary pathways connecting crucial areas within and across the cortical and limbic centers of the brain is consistent with the fact that adolescence is a developmental period that is associated with rigidity and inflexibility (e.g., moral and intellectual egocentrism and entitlement) as well as with psychosocial chaos and fluctuation (e.g., emotional and spiritual questioning and confusion). As these areas of the brain become progressively more complex and reliably interconnected (e.g., the myelination process),

the adolescent is increasingly able to not only think in more complex and abstract terms with an expanded base of knowledge, but moreover to think before (re)acting.

Adolescence also tends to involve a shift in relational focus away from bonding and affiliation with family and caregivers and toward peer relationships, which require greater independence of thought and action. Yet, as adolescents acquire increased autonomy, consistent ongoing primary (family) relationships continue to be essential (El-Sheikh, 2001) for sustaining sufficient emotional security to permit the youth to venture into the world of events and ideas and develop increasingly autonomous ways of living. In addition, a stable relational base helps the adolescent to cope with the rapid changes in brain development that occur during this transitional developmental period. With this stability, adolescents are more likely to succeed in coping effectively with the turmoil in their lives, which occurs because "behaviors become unmoored from their entrenched habits, [and] a variety of new forms proliferate for a while" (Lewis, 2005, p. 255). When adolescents are able to successfully handle these transitional challenges, a "subset of these [behavioral patterns] stabilizes, providing new habits for the next stage of development" (p. 256).

Impact of Psychological Trauma on Adolescent Development and Functioning

In adolescence, the aftereffects of psychological traumas experienced earlier in childhood may include problems in the very areas of biopsychosocial functioning that are most crucially and rapidly developing during this transitional period between childhood and adulthood. Adolescents who experienced abuse or domestic violence earlier in childhood are at risk for PTSD and problems with regulating their emotions (e.g., internalizing disorders such as major depressive disorder or dysthymic disorder, agoraphobia/panic or social anxiety disorders, phobias, dissociative disorders) and behavior (e.g., sleep disorders; Noll, Trickett, Susman, & Putnam, 2006); externalizing disorders, such

as oppositional defiant or conduct disorder, attentional or impulse control disorders, or substance use disorders, as well as eating and sexual and gender identity disorders (Cook et al., 2005). Impaired regulation of emotions and behavioral impulses in adolescence may take the form of exacerbated forms of these psychiatric disorders as well as traits that, if continued in adulthood, could constitute personality disorders.

Adolescents who experienced physical abuse before age 5 were more likely to be arrested for violent, nonviolent, and status offenses. Those who had been physically abused also less often graduated from high school and more often were fired from a job, were a teen parent, or had been pregnant or impregnated someone while being unmarried (Lansford et al., 2007). Childhood abuse or domestic violence also is associated with problems among adolescents involved in the juvenile justice system, including truancy, teen pregnancy, gang involvement, and suicidality (Ford, Hartman, Hawke, & Chapman, 2008).

Although neuroimaging studies have not been reported with adolescents who are diagnosed with PTSD (except in mixed samples that include children and adolescents), children with psychiatric disorders or a family history of addiction have been found to have greater difficulties in focusing attention as they traverse adolescence, consistent with findings of reduced volumes of the area in the brain's limbic system that is associated with fear and anxiety, the amygdala (primarily in the brain's right hemisphere; Hill & Shen, 2002). Adolescents who experienced psychological trauma in childhood and were depressed showed less evidence of problems with autobiographical memory ("over general memory retrieval") than depressed adolescents with no reported psychological trauma (Kuyken, Howell, & Dalgleish, 2006). PTSD has a stronger relationship to problems with autobiographical memory in adulthood than exposure to psychological trauma per se, but PTSD was not reported as a potential factor in this study. However, the study's findings suggest that psychological trauma may increase depressed adolescents' focus on self-relevant memories

(compared to the reduction in this which occurs among depressed adolescents generally), and therefore treatments that help depressed adolescents restore or develop autobiographical memory capacities may be more readily undertaken if the adolescent has had traumatic past experiences. Whether adolescents who are depressed and have experienced psychological trauma are good candidates for either trauma memory-focused therapies such as trauma-focused cognitive behavior therapy or personal narrative memory reconstruction therapies (e.g., Cloitre et al., 2006), remains to be tested in psychotherapy research with traumatized adolescents.

When psychological trauma occurs during adolescence, the youth is at risk for PTSD and anxiety, mood, and substance use disorders and problems with risky sexual behavior, suicidal thoughts, and aggression that may persist into adulthood or emerge for the first time later in adulthood (Green et al., 2005). Even a single incident of interpersonal psychological trauma (i.e., sexual assault) in adolescence was found to be associated with an increased likelihood of PTSD and risky sexual behavior among college women (Green et al., 2005). Research with adolescents from a wider range of backgrounds, and with boys as well as girls, is needed to document the effects of psychological trauma exposure before and during adolescence on the posttraumatic stress-related problems that adolescents experience as teens and later in their lives as adults. The likelihood that many adolescent trauma survivors underreport their extent of traumatic stress symptoms (e.g., one in six in a study of emergency department-treated adolescents; McCart et al., 2005) must be considered when estimates are made of the prevalence or severity of posttraumatic stress problems among adolescents.

Interventions for Adolescents Who Are Experiencing Posttraumatic Stress Problems

Developmental transitional periods such as adolescence can be an opportune time for therapeutic and prevention interventions precisely because the developing brain and personality

are in such flux at those times that any stabilizing or informative inputs may help the youth to gain a clearer and more positive direction for the future. Key protective factors that increase the likelihood of positive developmental outcomes for traumatized youths (Collishaw et al., 2007; Dumont, Widom, & Czaja, 2007) and adaptive function by traumatized adults (Schnurr, Lunney, & Sengupta, 2004) include primarily a strong, caring, and reliable social support system (e.g., responsive caregivers in childhood; mentoring, access to socioeconomic and educational resources and a cohesive peer group and family system in adolescence and adulthood) and secondarily personal attributes that enhance psychological hardiness. Interventions are being developed to enable adolescents and their families and communities to build these psychosocial resources, and have shown promise in clinical research studies (e.g., Cloitre, Cohen, & Koenen, 2006; DeRosa & Pelcovitz, in press). Group therapy for sexually abused girls also has shown promise in clinical research, with interpersonal/emotion-focused models (psychodrama) potentially reducing depression symptom severity and cognitive behavior therapy models potentially reducing PTSD symptom severity (Avinger & Jones, 2006). Adolescents have been shown to benefit from family therapy when they have problems such as aggressive behavior, depression, and substance use problems (Diamond & Josephson, 2005), but only one pilot study has evaluated the effectiveness of family therapy, with promising results in terms of reducing the distress reported by adolescents recovering from cancer and their parents (Kazak et al., 2004).

REFERENCES

- Andersen, S. (2003). Trajectories of brain development. *Neuroscience and Biobehavioral Reviews*, 27, 3–18.
- Avinger, K., & Jones, R. (2006). Group treatment of sexually abused adolescent girls: A review of outcome studies. *American Journal of Family Therapy*, 35, 315–326.
- Benes, F., Turtle, M., Khan, Y., & Farol, P. (1994). Myelination of a key relay zone in the hippocampal formation occurs in the human brain during

- childhood, adolescence, and adulthood. *Archives of General Psychiatry*, 51, 477–484.
- Cloitre, M., Cohen, L., & Koenen, K. (2006). *Treating survivors of childhood abuse: Psychotherapy for the interrupted life*. New York: Guilford Press.
- Collishaw, S., Pickles, A., Messer, J., Rutter, M., Shearer, C., & Maughan, B. (2007). Resilience to adult psychopathology following childhood maltreatment: Evidence from a community sample. *Child Abuse and Neglect*, 31, 211–229.
- Cook, A., Spinazzola, J., Ford, J. D., Lanktree, C., Blaustein, M., Cloitre, M., et al. (2005). Complex trauma in children and adolescents. *Psychiatric Annals*, 35, 390–398.
- DeRosa, R., & Pelcovitz, D. (in press). Group treatment for chronically traumatized adolescents: Igniting SPARCS of change. In D. Brom, R. Pat-Horenczyk, & J. D. Ford (Eds.), *Treating traumatized children: Risk, resilience, and recovery*. London: Routledge.
- Diamond, G., & Josephson, A. (2005). Family-based treatment research: A 10-year update. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44, 872–887.
- Dumont, K., Widom, C. S., & Czaja, S. (2007). Predictors of resilience in abused and neglected children grown-up. *Child Abuse and Neglect*, 31, 255–274.
- Eisenberg, N., Fabes, R. A., Murphy, B., Maszk, P., Smith, M., & Karbon, M. (1995). The role of emotionality and regulation in children's social functioning. *Child Development*, 66, 1360–1384.
- Eisenberger, N., Lieberman, M., & Williams, K. (2003). Does rejection hurt? *Science*, 302, 290–292.
- El-Sheikh, M. (2001). Parental drinking problems and children's adjustment: Vagal regulation and emotional reactivity as pathways and moderators of risk. *Journal of Abnormal Psychology*, 110, 499–515.
- Ford, J. D., Hartman, J. K., Hawke, J., & Chapman, J. (2008). Traumatic victimization, posttraumatic stress disorder, suicidal ideation, and substance abuse risk among juvenile justice-involved youths. *Journal of Child and Adolescent Trauma*, 1, 75–92.
- Giedd, J., Blumenthal, J., Jeffries, N., Castellanos, F., Liu, H., Zijdenbos, A., et al. (1999). Brain development during childhood and adolescence: A longitudinal MRI study. *Nature Neuroscience*, 2(10) 861–863.
- Green, B., Krupnick, J., Stockton, P., Goodman, L., Corcoran, C., & Petty, R. (2005). Effects of adolescent trauma exposure on risky behavior in college women. *Psychiatry*, 68, 363–378.
- Hill, S. Y., & Shen, S. (2002). Patterns of visual P3b in association with familial risk and childhood diagnosis. *Biological Psychiatry*, 51, 621–631.
- Kanemura, H., Aihara, M., Aoki, S., Araki, T., & Nakazawa, S. (2003). Development of the prefrontal lobe in infants and children. *Brain and Development*, 25, 195–199.
- Kazak, A. E., Alderfer, M. A., Streisand, R., Simms, S., Rourke, M. T., Barakat, L. P., et al. (2004). Treatment of posttraumatic stress symptoms in adolescent survivors of childhood cancer and their families. *Journal of Family Psychology*, 18, 493–504.
- Kuyken, W., Howell, R., & Dalgleish, T. (2006). Overgeneral autobiographical memory in depressed adolescents with, versus without, a reported history of trauma. *Journal of Abnormal Psychology*, 115, 387–396.
- Lansford, J., Miller-Johnson, S., Berlin, L., Dodge, K., Bates, J., & Petit, G. (2007). Early physical abuse and later violent delinquency: A prospective longitudinal study. *Child Maltreatment*, 12, 233–245.
- Lewis, M. D. (2005). Self-organizing individual differences in brain development. *Developmental Review*, 25, 252–277.
- Matsumoto, K., Suzuki, W., & Tanaka, K. (2003). Neuronal correlates of goal-based motor selection in the prefrontal cortex. *Science*, 301, 229–232.
- May, J. C., Delgado, M., Dahl, R., Stenger, A., Ryan, N., Fiez, J., et al. (2004). Event-related magnetic resonance imaging of reward-related brain circuitry in children and adolescents. *Biological Psychiatry*, 55, 359–366.
- McCart, M., Davies, W. H., Harris, R., Wincek, J., Calhoun, A., & Melzer-Lange, M. (2005). Assessment of trauma symptoms among adolescent assault victims. *Journal of Adolescent Health*, 36(1), 70.e7–70.e13.
- Noll, J., Trickett, P., Susman, E., & Putnam, F. (2006). Sleep disturbances and childhood sexual abuse. *Journal of Pediatric Psychology*, 31, 469–480.
- Schnurr, P. P., Lunney, C., & Sengupta, A. (2004). Risk factors for the development versus maintenance of posttraumatic stress disorder. *Journal of Traumatic Stress*, 17, 85–95.

Sowell, E., Thompson, P., Tessner, K., & Toga, A. (2001). Mapping continued brain growth and gray matter density reduction in dorsal frontal cortex. *Journal of Neuroscience*, 21, 8819–8829.

JULIAN D. FORD

University of Connecticut School of Medicine

See also: Amygdala; Biology, Brain Structure, and Function, Child; Child Development; Cognitive Behavior Therapy, Child Abuse; Family Systems; Hippocampus; Memories of Traumatic Experiences

AGGRESSION

Aggression is a heterogeneous and very broad category of behavior with diverse causes and consequences. Aggression often is defined as hostile, destructive, and/or injurious activity that has the potential and/or intention of inflicting damage to an inanimate object or harm to a living being. Terms such as aggressive, violent, conduct disorder, oppositional, psychopathic, under-aroused, delinquent, and antisocial have all been used to describe persons with persistent and frequent aggressive behavior. Overt aggression involves a direct confrontation with the environment including verbal threats of violence, impulsive episodes of property destruction, self-injurious behaviors, and physical assault. Covert aggression includes hidden and furtive behaviors such as lying, shoplifting, vandalism, and fire setting (Frick et al., 1993). Proactive, instrumental, or predatory aggression is motivated by reward. Reactive, affective, defensive, or impulsive aggression is motivated by reaction to threat or frustration in goal-directed behavior. Aggression can be adaptive or maladaptive based on its appropriateness to the context and its consequences for the person and others.

Although trauma does not cause aggression, traumatized individuals are at increased risk for both expressing aggression and/or being exposed to aggression by others. This may occur through several mechanisms. First, aggression is an associated symptom of diverse disorders such as depression, bipolar disorder,

posttraumatic stress disorder (PTSD), psychosis, attention deficit/hyperactivity disorder, and/or anxiety disorders (Connor & McLaughlin, 2006). Traumatized individuals meeting criteria for PTSD frequently meet diagnostic criteria for other psychiatric disorders that may have aggression as an associated symptom. Additionally, many of these associated diagnoses include symptoms of diminished impulse control that may lead traumatized individuals into environmental situations where they may be exposed to aggression by others. Second, the hyperarousal symptoms of PTSD such as problems with anger or irritability may lead to aggressive behavior or cause the individual to be aggressed upon by others. Third, acute, sudden, life-threatening stress activates many alarm systems in the central nervous system that are designed to perceive and evaluate threat, and to organize an adaptive response that will increase the individual's chances of survival and avoidance of injury. If the threat is too intense, too uncontrollable, too inescapable, or too chronic, these neurobiological mechanisms can become disorganized and are no longer adaptive (Adamec, Blundell, & Burton, 2006; Charney, 2003). Diminished inhibition may then result in the release of explosive moods and behaviors. Even when the acute trauma has resolved, increased rage, hostility, irritability, and aggression in the face of memories or reminders of the traumatic event may then occur, particularly if the past trauma involved being aggressed against and the individual now also is experiencing problems with depression (O'Donnell, Cook, Thompson, Riley, & Neria, 2006).

REFERENCES

- Adamec, R. E., Blundell, J., & Burton, P. (2006). Relationship of the predatory attack experience to neural plasticity, pCREB expression and neuroendocrine response. *Neuroscience and Biobehavioral Reviews*, 30(3), 356–375.
- Charney, D. S. (2003). Neuroanatomical circuits modulating fear and anxiety behaviors. *Acta Psychiatrica Scandinavica Supplementum*, 417, 38–50.
- Connor, D. F., & McLaughlin, T. J. (2006). Aggression and diagnosis in psychiatrically referred children.

Child Psychiatry and Human Development, 37(1), 1–14.

Frick, P. J., Lahey, B. B., Loeber, R., Tannenbaum, L., Van Horn, Y., Christ, M. A. G., et al. (1993). Oppositional defiant disorder and conduct disorder: A meta-analytic review of factor analyses and cross-validation in a clinical sample. *Clinical Psychology Review*, 13, 319–340.

O'Donnell, C., Cook, J. M., Thompson, R., Riley, K., & Neria, Y. (2006). Verbal and physical aggression in World War II former prisoners of war: Role of posttraumatic stress disorder and depression. *Journal of Traumatic Stress*, 19(6), 859–866.

DANIEL F. CONNOR

University of Connecticut School of Medicine

See also: Anger; Antisocial Behavior; Domestic Violence

ALCOHOL USE DISORDERS

People with posttraumatic stress disorder (PTSD) often qualify for diagnoses related to substance use, one of which is alcohol use disorder (AUD). In epidemiological research, lifetime PTSD signals an increased risk for lifetime AUD and prior PTSD predicts the onset of AUDs. Studies of patients with substance use disorders (SUD; i.e., *DSM-IV*-defined substance dependence, substance abuse) find current rates of PTSD to be much higher than those found in the general population, and range from 25–55%; patients seeking PTSD treatment have SUD rates ranging from 65–80% (as reviewed in Ouimette & Brown, 2003). Thus, the co-occurrence of SUD and PTSD is commonly found in community and clinical samples, and across a range of treatment settings.

The prognostic implications for patients with both SUD and PTSD are decidedly negative. Longitudinal studies suggest that over time, patients with both SUDs and PTSD consume more alcohol, relapse more quickly, have less social support, are more likely to be unemployed, and have treatment readmission rates that are higher than those for SUD patients without PTSD. SUD patients with comorbid PTSD incur an estimated \$3,000 more annually

in SUD treatment costs than those without PTSD. Substance abuse also has negative implications for patients seeking PTSD treatment because patients who continue to use substances have poorer PTSD outcomes than those who abstain from using (Ouimette & Brown, 2003).

Models

A number of hypotheses regarding associations between PTSD and AUD have been proposed. Among the most popular of these is the self-medication model, which is based on the premise that substance abuse constitutes an effort to cope with the distressing affect associated with a traumatic event in a manner that is analogous to the use of medication to alleviate symptoms of an illness. According to this model, persons with PTSD use mind-altering substances to mute emotional arousal or to lessen the disturbing effects of traumatic memories. Conversely, it also has been proposed that alcohol use contributes to the occurrence of trauma and PTSD (i.e., the High Risk Hypothesis; see Ouimette & Brown, 2003, for chapters reviewing models). For example, heavy drinkers may put themselves at risk for accidents, violence, or other potentially traumatic events. Additionally, substance use may trigger or exacerbate PTSD symptoms (Saladin, Brady, Dansky, & Kilpatrick, 1995).

A social learning perspective (SLT; Maisto, Carey, & Bradizza, 1999) also can be applied to the PTSD-SUD co-occurrence. According to this conceptualization, the desire to regulate learned emotional responses (fear, arousal, depression) to trauma motivates substance use. A component of SLT is *reciprocal determinism*, which describes dynamic associations among environmental and individual variables and behavior over time. Here, trauma exposure and posttraumatic stress symptoms could be both causes and effects of substance use. Cognitions related to the interpretation of trauma-related cues, the individual's ability to manage emotional cue responses, and beliefs about the palliative effects of substances also play a central role.

Changes in brain structure and chemistry may also explain PTSD-SUD associations. As noted by Brady and Sinha (2005), these underlying biological changes (1) may be shared by PTSD and SUD and/or (2) may be the result of prolonged substance use, which then in turn leads to the development of PTSD. Examples of such structural changes in the brain include, but are not limited to, alterations in the way the amygdala (the brain's emotion processing center; see: **Amygdala**) works, alterations in dopamine in the brain's reward pathways, and problems in regulating noradrenergic and neuroendocrine functioning, both of which are associated with the body's reaction to stressful stimuli.

Nonbiological, shared vulnerability mechanisms also have been implicated in PTSD-SUD co-occurrence. Among these are trait-level constructs such as anxiety sensitivity, negative emotionality, and behavioral undercontrol. Each of these personality variables has been suggested to contribute to increased risk for substance use as well as poor adaptation to trauma (i.e., posttraumatic stress), and thus potentially account for the frequent co-occurrence of the two disorders.

Importantly, although many models of PTSD-AUD associations consider which disorder develops first, the complexity of an individual's trauma history and the challenges of determining substance use and problem onset often make temporal delineation difficult. Thus, though an understanding of the primacy of one disorder may provide useful clinical information, establishing such primacy may prove challenging to ascertain in a clinical setting.

Treatments

While it is generally accepted in academic communities that integrated AUD-PTSD treatment is considered best practice and patients would prefer such concurrent therapy, most clinics do not treat both disorders simultaneously. Clinical researchers have developed integrated AUD-PTSD treatments with or without exposure therapy elements. Debate has ensued

about the inclusion of an exposure component, which is a recommended evidence-based practice for PTSD alone. Concern includes the potential for alcohol relapse, although preliminary data counter such concern. Concurrent treatment of PTSD and cocaine dependence combines coping skills training with exposure therapy for PTSD. Retention rates in this protocol were similar to those in treatments without exposure components, suggesting that exposure did not negatively affect patients' participation.

The most researched integrated treatment to date is called *Seeking Safety*, a present-focused coping skills therapy. Some key elements include: a focus on safety, working concurrently on PTSD and AUD, and coping skills work. Several studies support its effectiveness across several treatment populations. One study randomly assigned women to Seeking Safety or Relapse Prevention with a nonrandomized treatment-as-usual comparison group, and outcomes for Seeking Safety were comparable to Relapse Prevention and better than treatment as usual (see Ouimette & Brown, 2003 for chapters reviewing these interventions).

Conclusion

Further explication of the underlying mechanisms—psychological and biological—of PTSD-SUD comorbidity is needed. Identification of patient subgroups, including gender-specific types would be helpful in improving treatment effectiveness. Treatments need to be further evaluated for efficacy and cost-effectiveness relative to standard care and other programs. Given the popularity and practical utility of self-help approaches in the United States, an investigation of whether self-help can provide a helpful adjunct to integrated treatment would be useful, particularly for adult populations. Last, a preventative approach would be helpful, possibly addressing trauma-related symptoms and alcohol abuse before the development of full-fledged disorders, looking toward other settings such as primary care to identify at-risk individuals.

REFERENCES

- Brady, K. T., & Sinha, R. (2005). Co-occurring mental and substance use disorders: The neurobiological effects of chronic stress. *American Journal of Psychiatry*, 162, 1483–1493.
- Maisto, S. A., Carey, K. B., & Bradizza, C. M. (1999). Social learning theory. In K. E. Leonard & H. T. Blane (Eds.), *Psychological theories of drinking and alcoholism* (2nd ed., pp. 106–163). New York: Guilford Press.
- Ouimette, P., & Brown, P. J. (2003). *Trauma and substance abuse: Causes, consequences, and treatment of comorbid disorders*. Washington, DC: American Psychological Association.
- Saladin, M. E., Brady, K. T., Dansky, B. S., & Kilpatrick, D. G. (1995). Understanding comorbidity between PTSD and substance use disorders: Two preliminary investigations. *Addictive Behaviors*, 20, 643–655.

RECOMMENDED READING

- Coffey, S. F., Read, J. P., & Norberg, M. M. (2008). Posttraumatic stress disorder and substance use disorder: Neuroimaging, neuroendocrine, and physiological findings. In S. H. Stewart & P. J. Conrod (Eds.), *Anxiety and substance abuse disorders: The vicious cycle of comorbidity*. New York: Springer.

PAIGE OUIMETTE

State University of New York

JENNIFER P. READ

State University of New York

See also: Substance Use Disorders

ALEXITHYMIA

The term *alexithymia* literally means “no words for feelings” and is characterized by deficits in the ability to identify and label, as well as communicate affective experience. Alexithymia thus is considered an affect regulation disturbance (Taylor, Bagby, & Parker, 1997). Although alexithymic individuals are able to experience emotions, when asked to describe their internal experience they are able only to give vague, unelaborated descriptions of feelings (e.g., “upset”), or may report somatic or cognitive aspects of their experience. Additionally,

alexithymia is an externally oriented thinking style, characterized by communication that is lacking in personal involvement and devoid of references to personal feelings, thoughts, reactions, or attitudes about the event.

Measurement

Although a number of measures have been developed to assess specific aspects of alexithymia, such as emotional awareness, the Toronto Alexithymia Scale (TAS-20; Bagby, Parker, & Taylor, 1994a, 1994b) is widely accepted as the most comprehensive, reliable, and valid across populations and languages. This 20-item, self-report questionnaire asks individuals to rate, on a five-point Likert-type scale, the degree to which they agree with test items. Subscale items assess the three components of alexithymia described previously. Research (Bagby et al., 1994a) also has established cut-off scores on the TAS-20 used for identifying clinical levels of alexithymia. Prevalence rates, using this TAS-20 criterion, have been estimated as ranging between 13% and 24% in community and undergraduate college samples (Mason, Tyson, Jones, & Potts, 2005; Paivio & McCullough, 2003), and between 17% and 59% in clinical samples (Honkalampi et al., 2004; Muller, Buhner, & Ellgring, 2003).

Associated Psychological Disturbances

Alexithymia has important implications for functioning. Emotions provide vital information about self and relationships that guide perceptions and behavior so that limited awareness of feelings leaves the person disoriented (Damasio, 1999). Individuals without the capacity to identify and communicate feelings are more likely to experience negative emotions as confusing and express distress through somatic symptoms or through physical means, including violence to self or others (Paivio & McCullough, 2003; van der Kolk, Perry, & Herman, 1991). Alexithymia also has been associated with difficulty recognizing emotions in others, interpersonal problems, and limited social support (Turner & Paivio, 2002), and

with a number of psychological disturbances (for a review, see Taylor et al., 1997). These include posttraumatic stress disorder (PTSD), depression, anxiety, eating disorders, substance abuse, somatoform disorders, borderline personality, dissociative experiences, and self-injurious behaviors. Finally, alexithymia has been associated with development of weaker therapeutic alliances (Mallinckrodt, King, & Coble, 1998) and thus can interfere with an individual's capacity to benefit from psychotherapy.

Etiology

Developmental and social learning theories currently are the most commonly accepted explanations for alexithymia. These perspectives (e.g., Gottman, 1997) suggest that alexithymia develops when children learn that communicating emotion is inappropriate or ineffective in getting needs met. Gender role socialization, for example, explains the higher prevalence of alexithymia in males who frequently are taught to suppress normal emotional expressivity. As well, social learning effects are intensified in environments in which children learn that expression of feelings is not only meaningless but dangerous. There is considerable evidence supporting an association between alexithymia and a history of childhood maltreatment (e.g., Paivio & McCullough, 2003; Taylor et al., 1997; Turner & Paivio, 2002; Zlotnick, Mattia, & Zimmerman, 2001). Abused children can learn to ignore or avoid emotional experience as a strategy for coping with overwhelming negative affect and instead learn to rely on external cues and events to guide behavior.

Treatment

There are few psychological treatments specifically for alexithymia although a number of treatments for particular disorders (e.g., borderline personality, complex PTSD, eating disorders) address problems with emotion awareness and modulation. These approaches typically focus on increasing emotion awareness and labeling capacities through skills training. Research supports the efficacy

of these approaches (e.g., Becker-Stoll & Gerlinghoff, 2004; Kennedy & Franklin, 2002; Linehan, 1993). Results of a recent study (Ralston, 2006) indicated significant reductions in alexithymia over the course of emotion focused therapy for child abuse trauma (EFTT; Paivio, Chagigiorgis, Hall, Jarry, & Ralston, 2007). EFTT relies on promoting client experiencing (exploration of feelings and meanings) rather than skills training. This suggests that alexithymia may be more related to problems with complex affective meaning rather than emotion word vocabulary, per se, and treatments focusing on affective meaning construction may be effective options.

REFERENCES

- Bagby, M., Parker, J., & Taylor, G. (1994a). The twenty-item Toronto Alexithymia Scale I: Item selection and cross-validation of the factor structure. *Journal of Psychosomatic Research*, 38, 23–32.
- Bagby, M., Parker, J., & Taylor, G. (1994b). The twenty-item Toronto Alexithymia Scale II: Convergent, discriminant, and concurrent validity. *Journal of Psychosomatic Research*, 38, 33–40.
- Becker-Stoll, F., & Gerlinghoff, M. (2004). Impact of a four month day treatment programme on alexithymia in eating disorders. *European Eating Disorders Review*, 12, 159–163.
- Damasio, A. R. (1999). *The feeling of what happens: Body and emotion in the making of consciousness*. New York: Harcourt Brace.
- Gottman, K. M. (1997). *The heart of parenting: Raising an emotionally intelligent child*. New York: Simon & Schuster.
- Honkalampi, K., Koiivumaa-Honkanen, H., Antikainen, R., Haatiinen, K., Hintikka, J., & Viitamaki, H. (2004). Relationships among alexithymia, adverse childhood experiences, sociodemographic variables, and actual mood disorder: A 2-year clinical follow-up study of patients with major depressive disorder. *Psychosomatics*, 45, 197–204.
- Kennedy, M., & Franklin, J. (2002). Skills based treatment for alexithymia: An exploratory case series. *Behavior Change*, 19, 158–171.
- Linehan, M. M. (1993). *Cognitive-behavioral treatment for borderline personality disorder*. New York: Guilford Press.

- Mallinckrodt, B., King, J. L., & Coble, H. M. (1998). Family dysfunction, alexithymia, and client attachment to therapist. *Journal of Counseling Psychology, 45*, 497–504.
- Mason, O., Tyson, M., Jones, C., & Potts, S. (2005). Alexithymia: Its prevalence and correlates in a British undergraduate sample. *Psychology and Psychotherapy: Theory, Research, and Practice, 78*, 113–125.
- Muller, J., Buhner, M., & Ellgring, H. (2003). Is there a reliable factor structure in the 20 item Toronto Alexithymia Scale? A comparison of factor models in clinical and normative adult samples. *Journal of Psychosomatic Research, 55*, 561–568.
- Paivio, S. C., Chagigiorgis, H., Hall, I., Jarry, J., & Ralston M. (2007). *Comparative efficacy of two versions of emotion focused therapy for child abuse trauma: A dismantling study*. Manuscript submitted for publication.
- Paivio, S. C., & McCullough, C. R. (2003). Alexithymia as a mediator between childhood trauma and self-injurious behaviours. *Child Abuse and Neglect, 28*, 339–354.
- Ralston, M. B. (2006). *Imaginal confrontation versus evocative empathy in emotion focused trauma therapy*. Unpublished doctoral dissertation, University of Windsor, Windsor, Ontario, Canada.
- Taylor, G., Bagby, M., & Parker, J. (1997). *Disorders of affect regulation: Alexithymia in medical and psychiatric illness*. Cambridge: Cambridge University Press.
- Turner A., & Paivio, S. (2002, August). *Relations among childhood trauma, alexithymia, social anxiety, and social support*. Poster presented at the annual meeting of the American Psychological Association, Chicago.
- Van der Kolk, B., Perry, C., & Herman, J. (1991). Childhood origins of self-destructive behavior. *American Journal of Psychiatry, 148*, 1665–1670.
- Zlotnick, C., Mattia, J. I., & Zimmerman, M. (2001). The relationship between posttraumatic stress disorder, childhood trauma, and alexithymia in an outpatient sample. *Journal of Traumatic Stress, 14*, 177–188.

ELISABETH KUNZLE
University of Windsor

SANDRA C. PAIVIO
University of Windsor

See also: Dissociation; Emotional Numbing

ALIENATION AND TRAUMA

Alienation is a psychological concept consisting of some positive and mostly negative feelings. Such positive feelings may involve feeling that the individual is unique emotionally, but negative feelings involve feeling that he or she is alienated, isolated, and withdrawn from reality (Al-Sahel & Hanoora, 2001). Brown, Higgins, and Paulsen (2003) defined alienation “as the negative sense of fragmentation, estrangement, and separation.” Moreover, there are many other definitions of alienation as a psychological concept, however, the main common elements between them is that alienation denotes feelings of estrangement, being lost, loneliness or isolation from others, feeling helpless, and feeling as if one is losing relationships with others (Dimen, 2003; Florence & Bernard, 1967; Hanoora, 1998; Reber, 1995; Seeman, 1959).

Little has been written about the relationship between trauma and the deep-seated sense of guilt and shame of trauma's victims, in which the alienation from others is the main expression of these feelings. In addition, Ebert and Dyck (2004) indicated that exposure to extreme interpersonal stress can lead to alienation from others, shame and guilt, and a sense of being permanently damaged. Psychologically, most of the victims of trauma try to appear normal/healthy when they are still emotionally suffering from the traumatic event, but they do not want others to see that suffering. For example, it was found that most combat veterans remain alienated when they appear to be normal because just under the surface are the unintegrated personality fragments that cannot grieve, feel fear, and express anger without something self-destructive happening (Brende & McDonald, 1989).

Studies show that alienation could be recognized when the traumatic event victim avoids people, activities, or places, which arouse recollections of the original trauma (which is a symptom of PTSD; Forbes et al., 2002; Morison, 2002). In addition, Brende and McDonald (1989) found that severe and intractable symptoms of PTSD in Vietnam combat

veterans were related to entrenched guilt and shame. These symptoms include changes in self-identity, destructive, and self-destructive behavior. It was also found that trauma victims indicated an overall feeling of alienation from self and others (Ehlers et al., 1998; Evans & Stinnett, 2006).

REFERENCES

- Al-Sahel, R. A., & Hanoora, M. A. (2001). Level of feeling of trauma and its relationship with personality values, alienation, and psychological disorders among youth. *Journal of Social Sciences: The Academic Publication Council, Kuwait University*, 29, 55–80.
- Brende, J., & McDonald, E. (1989). Posttraumatic spiritual alienation and recovery in Vietnam combat veterans. *Spirituality Today*, 3, 319–340.
- Brown, M. R., Higgins, K., & Paulsen, K. (2003). Adolescent alienation: What is it and what can educators do about it? *Intervention in School and Clinic*, 39, 3–9.
- Dimen, M. (2003). Keep on keepin' on: Alienation and trauma commentary on Ruth Fallenberg's paper. *Studies in Gender and Sexuality*, 4, 93–103.
- Ebert, A., & Dyck, M. (2004). The experience of mental death: The core feature of complex post-traumatic stress disorder. *Clinical Psychology Review*, 24, 617–636.
- Ehlers, A., Clark, D. M., Dunmore, E., Jaycox, L., Meadows, E., & Foa, E. B. (1998). Prediction response to exposure treatment in PTSD: The role of mental defeat and alienation. *Journal of Traumatic Stress*, 11, 457–471.
- Evans, L. G., & Stinnett, J. O. (2006). Structure and prevalence of PTSD symptomology in children who have experienced a severe tornado. *Psychology in the School*, 43, 283–295.
- Florence, R., & Bernard, K. (1967). Alienation and family crisis. *Sociological Quarterly*, 8, 397–405.
- Forbes, D., Creamer, M., Allen, N., McHugh, T., Debenham, P., & Hopwood, M. (2002). MMPI-2 as predictor of change in PTSD symptoms. *Journal of Personality Assessment*, 8, 183–186.
- Hanoora, M. (1998). *Personality and mental health*. Cairo, Egypt: Anglo Press.
- Morison, J. (2002). PTSD in victims of sexual molestation: Its incidence, characteristics, and treatment strategies. *Behaviour Research and Therapy*, 4, 439–457.
- Reber, A. (1995). *The Penguin dictionary of psychology* (2nd ed.). London: Penguin Books.
- Seeman, M. (1959). On the meaning of alienation. *American Sociological Review*, 24, 783–791.

RECOMMENDED READINGS

- Ehlers, A., Clark, D. M., Dunmore, E., Jaycox, L., Meadows, E., & Foa, E. B. (1998). Prediction response to exposure treatment in PTSD: The role of mental defeat and alienation. *Journal of Traumatic Stress*, 11, 457–471.
- Lieb, R., Wittchen, H., & Van Os, J. (2006). Impact of psychological trauma on the development of psychotic symptoms: Relationship with psychosis proneness. *British Journal of Psychiatry*, 188, 527–533.

RASHED A. AL-SAHHEL
Kuwait University

See also: Avoidance; Social Support

AMYGDALA

The *amygdala* is a complex brain structure involved in a variety of normal brain functions and psychiatric conditions. The existence of the amygdala was first recognized in the early nineteenth century. The name, derived from the Greek language, was meant to denote the almond-like shape of this region in the medial temporal lobe. It is traditionally thought to consist of an evolutionarily primitive division associated with the olfactory (i.e., smell) system (the cortico-medial region) and an evolutionarily newer division associated with the neocortex (the basolateral region).

Each nucleus has unique connections. For example, the lateral amygdala is a major site receiving convergent inputs from visual, auditory, somatosensory (including pain) systems. The central nucleus connects with brainstem areas that control the expression of innate behaviors and associated physiological responses. And the medial nucleus of the amygdala is strongly connected with the olfactory system.

In the late 1930s, researchers observed that damage to the temporal lobe resulted in profound changes in fear reactivity, feeding, and

sexual behavior that came to be called the Kluver-Bucy syndrome. Around mid-century, it was determined that damage to the amygdala accounted for these changes in emotional processing. Numerous studies subsequently attempted to understand the role of the amygdala in emotional functions, especially fear. Studies in rodents have mapped the inputs to and outputs of amygdala nuclei that are involved in fear conditioning. In particular, it is widely accepted that when an animal is repeatedly exposed to a painful stimulus (an unconditioned stimulus) and a neutral stimulus (a conditioned stimulus), this leads to change in the neurons (synaptic plasticity) in the lateral amygdala. When the conditioned stimulus then occurs alone later, neural activation flows through these potentiated synapses to the other amygdala targets and ultimately to the central nucleus, outputs of which control conditioned fear responses. Specific cellular and molecular mechanisms within lateral amygdala cells have been shown to underlie these changes in brain activation associated with learned fear.

Although fear is the emotion best understood in terms of brain mechanisms, the amygdala has also been implicated in a variety of other emotional functions. A relatively large body of research has focused on the role of the amygdala in processing rewards and the use of rewards to motivate and reinforce behavior. As with aversive conditioning, the lateral, basal, and central amygdala have been implicated in different aspects of reward learning and motivation. The amygdala has also been implicated in emotional states associated with aggressive, maternal, sexual, and ingestive (eating and drinking) behaviors. Less is known about the detailed circuitry involved in these emotional states than is known about the brain activity involved in fear.

Because the amygdala is altered by and stores information about emotional events, it is said to participate in *emotional memory*. Emotional memory is viewed as an implicit or unconscious form of memory and contrasts with explicit or declarative memory mediated by the hippocampus.

In addition to its role in emotion and unconscious emotional memory, the amygdala is also

involved in the regulation or modulation of a variety of cognitive functions, such as attention, perception, and explicit memory. It is generally thought that these cognitive functions are modulated by the amygdala's processing of the emotional significance of external stimuli. Outputs of the amygdala then lead to the release of hormones and/or neuromodulators in the brain that alter cognitive processing in cortical areas. For example, via amygdala outputs that ultimately affect the hippocampus, explicit memories about emotional situations are enhanced.

Over the past decade, interest in the human amygdala has grown considerably, spurred on by the progress in animal studies and by the development of functional imaging techniques. As in the animal brain, damage to the human amygdala interferes with fear conditioning and functional activity changes in the human amygdala in response to fear conditioning. Further, exposure to emotional faces potently activates the human amygdala. Both conditioned stimuli and emotional faces produce strong amygdala activation when presented unconsciously, emphasizing the importance of the amygdala as an implicit information processor and its role in unconscious memory. Findings regarding the human amygdala are mainly at the level of the whole region rather than nuclei.

Structural and/or functional changes in the amygdala are associated with a wide variety of psychiatric conditions, including anxiety disorders such as PTSD, phobias, and panic disorder, depression, schizophrenia, and autism. This does not mean that the amygdala causes these disorders. It simply means that in people who have these disorders, alterations occur in the amygdala. Because each of these disorders involves fear and anxiety to some extent, the involvement of the amygdala in some of these disorders may be related to increased anxiety in these patients.

RECOMMENDED READINGS

Cardinal, R. N., Parkinson, J. A., Hall, J., & Everitt, B. J. (2002). Emotion and motivation: The role of the amygdala, ventral striatum, and prefrontal cortex. *Neuroscience and Biobehavior Review*, 26, 321–352.

- Charney, D. S. (2003). Neuroanatomical circuits modulating fear and anxiety behaviors. *Acta Psychiatrica Scandinavica* (Suppl.), 38–50.
- LeDoux, J. E. (1996). *The emotional brain*. New York: Simon & Schuster.
- McGaugh, J. L. (2003). *Memory and emotion: The making of lasting memories*. London: Orion.
- Phelps, E. A. (2006). Emotion and cognition: Insights from studies of the human amygdala. *Annual Review of Psychology*, 57, 27–53.
- Shinnick-Gallagher, P., Pitkanen, A., Shekhar, A., & Cahill, L. (Eds.). (2003). *The amygdala in brain function: Basic and clinical approaches*. New York: New York Academy of Sciences.

JOSEPH LEDOUX
New York University

See also: Biology, Animal Models; Biology, Brain Structure, and Function, Adult; Biology, Brain Structure, and Function, Child; Biology, Neurochemistry; Conditioned Fear; Hippocampus; Limbic System

ANGER

Anger is a negatively toned emotion, subjectively experienced as an aroused state of antagonism toward someone or something perceived to be the source of an aversive event. It is triggered or provoked situationally by events that are perceived to constitute deliberate harm-doing by an instigator toward oneself or toward those to whom one is endeared. Provocations usually take the form of insults, unfair treatments, or thwartings appraised as intended. Anger is prototypically experienced as a justified response to a perceived “wrong.” While anger is situationally triggered by acute, proximal occurrences, it is shaped and facilitated contextually by conditions affecting the cognitive, arousal, and behavioral systems that comprise anger reactions. Anger activation is centrally linked to threat perceptions and survival responding, and thus it has intrinsic relevance for understanding trauma.

As a normal human emotion, anger has considerable adaptive value, although there are sociocultural variations in the acceptability of its expression and the form that such expression

takes. In the face of adversity, it can mobilize psychological resources, energize behaviors for corrective action, and facilitate perseverance. Anger serves as a guardian to self-esteem, operates as a means of communicating negative sentiment, potentiates the ability to redress grievances, and boosts determination to overcome obstacles to our happiness and aspirations. Akin to aggressive behavior, anger has functional value for survival. Anger, an emotion, should be distinguished from hostility, which is an attitudinal disposition, and from aggression, which is behavior intended to do harm.

Despite having multiple adaptive functions, anger also has maladaptive effects on personal and social well-being. Generally, strong physiological arousal impairs the processing of information and lessens cognitive control of behavior. Because heightened physiological arousal is a core component of anger, people are not cognitively proficient when they become angry. Also, because the activation of anger is accompanied by aggressive impulses, anger can motivate harm toward other people, which in turn can produce undesirable consequences for the angered person, either from direct retaliation, loss of supportive relationships, or social censure. An angry person is not optimally alert, thoughtful, empathic, prudent, or physically healthy. Being a turbulent emotion ubiquitous in everyday life, anger is now known to be substantially associated with various stress-related disorders, such as cardiovascular disorders, in addition to its relevance for trauma and for post-traumatic stress disorder (PTSD).

Trauma and Anger Dysregulation

Anger, since at least 1942, has been identified as a component of traumatic reactions in clinical and field studies. Anger is a recognized feature of a range of clinical disorders that may result from psychological trauma exposure, such as dissociative amnesia, dissociative identity disorder, borderline personality disorder, head-trauma dementia, major depressive disorder, and especially PTSD. “Irritability/outburst of anger” is one of five symptoms in the hyperarousal cluster for PTSD diagnosis

(since *DSM-III-R*'s publication). Although typically viewed as merely a symptom of PTSD, anger can alternatively be seen as a dynamic factor bearing on the course of traumatic stress and its treatment. While anger or irritability is a PTSD diagnostic symptom, PTSD diagnostic criteria can be met in the absence of anger or irritability.

The meta-analysis by Orth and Wieland (2006), who reviewed 39 studies between 1985 and 2003, demonstrated that anger is substantially associated with PTSD in trauma-exposed adults, with the largest effect size being obtained for those having military war experience. As well, they found other trauma sources, such as technological disaster, crime victimization, and motor vehicle accidents to have medium to large effects for anger and hostility's association with PTSD.

Beyond this associative linkage, anger has been found to be predictive of PTSD chronicity, severity, and treatment response with various trauma populations. Studies with noncombat populations show anger to be a key long-term symptom resulting from trauma, as found with sexual assault survivors, motor vehicle accident victims, violence-exposed adolescents, domestic violence victims, political prisoners, refugees, and general samples of psychiatric patients. A growing body of research indicates that the relevance of anger is more than that of a mere symptom or diagnostic marker. Multiple studies point to anger's importance for the course of PTSD and for treatment efficacy, including longitudinal studies of Vietnam veterans, adult victims of violent crime, and motor vehicle accident victims. Anger assessed at early stages of trauma has been found to be predictive of later PTSD severity and response to treatment after statistically controlling for initial PTSD and various background and psychosocial factors.

As a result of trauma, anger occurs as part of a dyscontrol syndrome activated by sensing a threat in one's environment. The engagement of anger in PTSD involves hostile appraisal, heightened arousal, and antagonistic behavior as survival responding in contextually inappropriate conditions, such that the person becomes

dysregulated in reacting to the demands of the environment. While anger is situationally triggered by acute, proximal occurrences, it can be primed by trauma-related stimuli and contexts. Threat perception and anger schemas are reciprocally influenced.

Because of the survival function of the threat-sensing aspects of anger schemas, the detection of threat carries urgent priority and rapidly engages anger. Linking anger to survival needs allows for understanding its activation in PTSD and increasing violence risk: (1) its onset carries a coping response urgency that preempts alternative appraisals of the triggering event and considerations of alternative action plans; (2) it engages cognitive processes and behavior that further bias the system toward confirmation of the expectation of threat; (3) the strong arousal and the peremptory nature of the threat schemas suppress inhibitory controls of aggressive behavior; and (4) threat-anger responses are organized as a positive feedback loop—the more threat is perceived, the more anger and aggression are engaged; and, conversely, the more anger and aggression, the greater is the readiness to perceive threat. The cognitive distortions linked to threat perceptions and highly automatized anger have powerful immediacy and validity, and anger is infused with themes of justification.

Why does anger dysregulation occur in the context of trauma? One possibility is that anger serves to suppress both fear and pain in conjunction with activating approach or attack behaviors. Anger counters "loss of control," providing an antidote to the debilitating sense of vulnerability or uncontrollability. Anger keeps the adversary in mind (maintaining attentional focus on that which threatens survival), and it may prolong PTSD because it entails rumination, entraining the reexperiencing of one's trauma through a cognitive network of associations. Anger also causes alarm and has symbolic links to madness and badness. Long after trauma exposure, heightened arousal from everyday provocations might overwhelm self-regulatory capacity and reevoke the trauma experience.

Treatment of Anger in Trauma-Related Disorders

Essential to reinstituting regulatory controls for anger and aggression is treating the patient's self-monitoring deficits. This entails helping the person to (1) monitor cognitions during anger episodes; (2) identifying signs of arousal, as well as its intensity, duration, and lability in response to the perception of danger; (3) recognizing how anger reactions can escalate the threat potential of a situation; and (4) distinguishing impulsive actions from more controlled responses. Anger's cognitive, arousal, and behavioral domains are thus segmented for self-monitoring. In the treatment of anger disorders, cognitive behavioral therapy (CBT) approaches have been found to be effective with a wide range of clinical populations, including combat veterans with severe anger and severe PTSD (Chemtob, Novaco, Hamada, & Gross, 1997).

The CBT psychotherapeutic procedure is an adjunctive treatment involving cognitive restructuring, somatic arousal reduction, and behavioral coping skills enhancement. To facilitate anger regulation, the therapist strives to disconnect anger from the threat system, first through provision of safety, patience, and psychological space for reflection, exploration, and choice. The client's view of anger is normalized, to obviate worries about being a "bad" or unworthy person. The therapist acknowledges the legitimacy of the client's feelings, affirming his or her self-worth. Building trust in the therapeutic relationship is pivotal. As self-regulation hinges on knowledge, education about anger and discovery of the client's personal anger patterns or "anger signature" is facilitated. Much is done to augment self-monitoring and to encourage the moderation of anger intensity. Because tension or strain may surface in the course of treatment, the therapist models and reinforces nonanger alternative responding so as to build replacements for the automatized angry reactions that had been the client's default coping style.

In the CBT stress inoculation approach to anger treatment, provocation is simulated by therapeutically paced exposure to anger incidents created in imaginal visualization and in

role-play. The progressively graduated exposure, directed by the therapist, involves a hierarchy of anger incidents produced by the collaborative work of client and therapist. This graduated, hierarchical exposure, done in conjunction with the teaching of stress coping skills, is the basis for the inoculation metaphor. Anger-control coping skills are rehearsed with the therapist and practiced while visualizing and role-playing progressively more intense anger-arousing scenes from the personal hierarchies. See Novaco and Chemtob (1998) for more detailed discussion.

REFERENCES

- Chemtob, C. M., Novaco, R. W., Hamada, R. S., & Gross, D. M. (1997). Cognitive-behavioral treatment for severe anger in post-traumatic stress disorder. *Journal of Consulting and Clinical Psychology, 65*, 184–189.
- Novaco, R. W., & Chemtob, C. M. (1998). Anger and trauma: Conceptualization, assessment, and treatment. In V. M. Follette, J. I. Rusek, & F. R. Abueg (Eds.), *Cognitive behavioral therapies for trauma* (pp. 162–190). New York: Guilford Press.
- Orth, U., & Wieland, E. (2006). Anger, hostility, and posttraumatic stress disorder in trauma-exposed adults: A meta-analysis. *Journal of Consulting and Clinical Psychology, 74*, 698–706.

RAYMOND W. NOVACO
University of California, Irvine

See also: Aggression; Cognitive Behavior Therapy, Adult; Social Cognitive Theory

ANHEDONIA

See: Depression; Emotional Numbing

ANNIVERSARY REACTIONS

Stimuli that reactivate traumatic memories (i.e., "triggers") often include significant dates or times of the year that are associated with unresolved traumatic experiences. These stimuli can induce particular time-related responses called *anniversary reactions* that range from

mild and transient, to severe and prolonged. Anniversary reactions are typically understood as annual phenomena, but belong to a broader category of reactions that include more frequent time-related triggers, such as months, weeks, days, or even time of day.

Unresolved experiences associated with anniversary reactions include traumatic birth and deaths (Bowlby, 1980), abortions (Franco et al., 1989), abuse, accidents, natural disasters, or war experiences (Morgan, Hill, Fox, Kingham, & Southwick, 1999). Anniversary reactions involve symptoms and features of posttraumatic stress and complicated grief: flashbacks, nightmares, sleep disorders, fear and anxiety, rage, sadness, guilt, shame, suicidality, depression, or manic episodes (Beratis, Gourzis, & Gabriel, 1996), and brief reactive psychoses. Avoidance, numbing, and detachment symptoms may be prominent around the anniversary date, including efforts to avoid any reminders of the event, narrowed focus of attention, reduction in awareness of surroundings, derealization, depersonalization, and obsessive compulsive behaviors that serve to contain anxiety.

One patient reported taking heavy doses of sleeping pills in order to sleep through the entire 24-hour period that triggered her. Significant somatic symptoms associated with the traumatic experience may occur (Cavenar, Nash, & Maltbie, 1978). For example, a woman who as a child was tied up and raped during Christmas holidays had nausea, intense vaginal pain, and numbing in her hands every December.

Symptoms of anniversary reactions may last several days or weeks, and in rare instances they may last months. Many individuals are not consciously aware of the connection between their current distress and the original event. Since the inclusion of posttraumatic stress disorder in *DSM-III* in 1980 (American Psychiatric Association, 1980), anniversary reactions are considered to be a special feature of this disorder. These specific experiences were subsumed under the more general category of posttraumatic triggers, resulting in fewer studies on anniversary reactions in the literature since the mid-1980s. The most widely studied anniversary reactions pertain to the anniversary of the death

of a loved one that was experienced as traumatic and has not been sufficiently mourned and integrated (Bowlby, 1980; Volkan, 1989).

Hilgard (1953; see also Volkan, 1989) distinguished a particular form of anniversary reactions. Parents may experience symptoms when their child reaches the age at which they themselves experienced a traumatic event. She described a man who developed severe headaches and psychosis, and made a suicide attempt when his son was 4 years old. When he had been the same age as his son, the man's father had unexpectedly died and his mother had to return to work, precipitating severe unresolved feelings of abandonment. Conversely, children of parents who died or were traumatized at a certain age may experience anniversary reactions when they reach the same age as the parent. For example, a patient developed severe panic, phobia of bathrooms, and suicidality when she reached the same age as her mother at the time of her suicide. The daughter, a child at the time of her mother's death, had found her mother dead in the bathtub, and had been instructed by the family never to speak of it.

Some religious and cultural traditions have recognized that anniversary reactions involve unresolved mourning and have designated special commemorations of anniversaries of painful or traumatic events (Lamm, 1969). Such observances are helpful in resolving traumatic experiences. There are several essential aspects to commemorations that parallel psychotherapy of anniversary reactions: (1) evocation and narration of a conscious memory of the event, which is an exposure technique; (2) social support, which has shown to be an essential part of overcoming traumatic reactions; and (3) setting aside a specific time for mourning and other emotions, with gradual support to move back to normal life. Psychotherapy also includes reworking maladaptive or incorrect beliefs that maintain overwhelming negative emotions. For example, patients often inappropriately blame themselves for accidents, their own abuse, or the death of a loved one. Final resolution of anniversary reactions occurs when the individual is able to fully realize and integrate the traumatic experience.

REFERENCES

- American Psychiatric Association. (1980). *Diagnostic and statistical manual of mental disorders* (3rd ed.). Washington, DC: Author.
- Beratis, S., Gourzis, P., & Gabriel, J. (1996). Psychological factors in the development of mood disorders with a seasonal pattern. *Psychopathology*, 29, 331–339.
- Bowlby, J. (1980). *Loss: Sadness and depression*. London: Hogarth Press.
- Cavenar, J. O., Nash, J. L., & Maltbie, A. A. (1978). Anniversary reactions presenting as physical complaints. *Journal of Clinical Psychiatry*, 39, 369–374.
- Franco, K., Campbell, N., Tamburrino, M., Jurs, S., Pentz, J., & Evans, C. (1989). Anniversary reactions and due date responses following abortion. *Psychotherapy and Psychosomatics*, 52, 151–154.
- Hilgard, J. R. (1953). Anniversary reactions in parents precipitated by children. *Psychiatry*, 16, 73–80.
- Lamm, M. (1969). *The Jewish way in death and mourning*. Middle Village, NY: Jonathan David.
- Morgan III, C. A., Hill, S., Fox, P., Kingham, P., & Southwick, S. M. (1999). Anniversary reactions in Gulf War veterans: A follow-up inquiry 6 years after the war. *American Journal of Psychiatry*, 156, 1075–1079.
- Volkan, G. H. (1989). *The mourning-liberation process* (Vols. 1 & 2). Madison, CT: International Universities Press.

ONNO VAN DER HART
Utrecht University

KATHY STEELE
Metropolitan Counseling Services

See also: Acute Stress Disorder; Memories of Traumatic Experiences; Posttraumatic Stress Disorder

ANTERIOR CINGULATE CORTEX

Basic Anatomy and Function

The anterior cingulate cortex (ACC) is a medial prefrontal structure located anterior to (in front of) the genu of the corpus callosum (see Figure 1). The ACC differs from other subregions of the cingulate gyrus in its cytoarchitecture, connectivity with other structures,

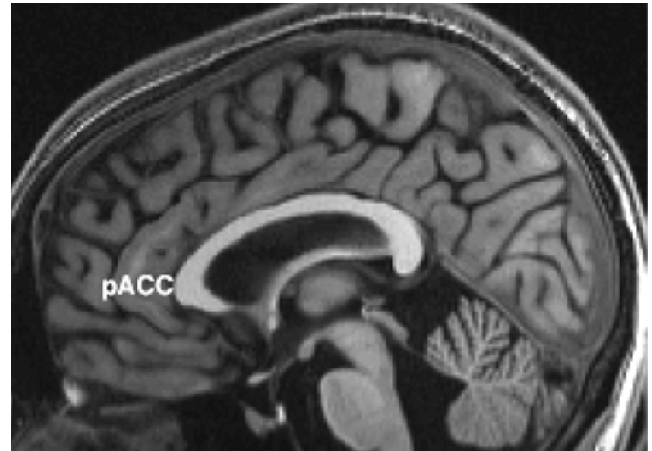


Figure 1. This magnetic resonance image shows the midsagittal (middle) surface of the human brain. Perigenual anterior singulate cortex (pACC) lies anterior to (in front of) the genu of the corpus callosum.

and function (Bush, Luu, & Posner, 2000; Vogt, Berger, & Derbyshire, 2003). Specifically, the perigenual (front portion of the) ACC is reciprocally connected to the amygdala (see: **Amygdala**), sends projections to autonomic centers in the brainstem, and appears to be involved in the processing of emotional information and the regulation of emotional responses. The ventral (lower portion of the) ACC and other ventral medial prefrontal regions appear to play an important role in the retention of fear extinction after fear conditioning.

In contrast, the portion of the cingulate that is dorsal to (behind) the ACC (i.e., the anterior midcingulate cortex [aMCC]) has fewer connections to the amygdala and more connections to parietal and motor areas, and is involved in executive functions including response selection and monitoring of conflict and errors.

The nomenclature used to describe the boundaries and subdivisions of the ACC has evolved over the years. The nomenclature used in this entry reflects the most recent research of Vogt and colleagues (Vogt, in press; Vogt et al., 2003).

ACC Function in Posttraumatic Stress Disorder

The results of many recent functional neuroimaging studies suggest diminished function of

the perigenual ACC and surrounding medial frontal cortex in posttraumatic stress disorder (PTSD) relative to individuals without PTSD (Shin, Rauch, & Pitman, 2006). This finding has been reported when participants view traumatic reminders, recall and imagine their own traumatic events, view fearful facial expressions, recall emotional words, perform emotional Stroop interference tasks, and undergo extinction after fear conditioning. Although a small number of studies have yielded discrepant results, the majority of studies have provided evidence for diminished perigenual ACC function in PTSD. Notably, several studies have reported that the degree of ACC activation is inversely related to symptom severity, such that individuals with lower ACC activation have more severe symptoms. Two treatment studies have reported a relationship between symptomatic response to serotonin reuptake inhibitors and increased activation of medial prefrontal cortical regions. Preliminary data suggests that the function of more dorsal portions of the ACC may not be diminished in PTSD.

According to current neurocircuitry models of PTSD, perigenual ACC and adjacent medial prefrontal regions are less responsive than normal and fail to inhibit an over-responsive amygdala (Shin et al., 2006). There is indeed evidence that the amygdala is overly responsive to reminders of trauma and to emotional, trauma-unrelated stimuli in PTSD. Four studies have reported a functional relationship between ACC and amygdala in PTSD, although the direction of this relationship is not yet clear.

Structural magnetic resonance imaging (MRI) studies have revealed that ACC volumes are smaller in PTSD patients compared to trauma-exposed control participants, even when controlling for alcohol use and total brain volume. Additionally, two studies have found ACC volume to be inversely correlated with severity of PTSD symptoms. One study has found shape differences in the ACC in PTSD.

Complementing the structural MRI studies, a magnetic resonance spectroscopy study has reported diminished N-acetyl aspartate (NAA)/creatine ratios (which is thought to reflect a loss of functional nerve cells) in the perigenual

ACC in PTSD. However, another study failed to replicate this finding. Thus while the exact mechanisms are unclear, PTSD appears to involve a compromise to the perigenual ACC's neural structure. This may be related to diminished function of the ACC and reduced inhibition of the amygdala in PTSD.

Although a wealth of data suggests diminished perigenual ACC function and volumes in PTSD, whether these abnormalities are a concomitant of the disorder or whether they represent a preexisting vulnerability is currently unknown.

REFERENCES

- Bush, G., Luu, P., & Posner, M. I. (2000). Cognitive and emotional influence in anterior cingulate cortex. *Trends in Cognitive Sciences*, 4, 215–222.
- Shin, L. M., Rauch, S. L., & Pitman, R. K. (2006). Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Annals of the New York Academy of Sciences*, 1071, 67–79.
- Vogt, B. A. (Ed.). (in press). *Cingulate neurobiology and disease: Vol. 1. Infrastructure, diagnosis, and treatment*. New York: Oxford University Press.
- Vogt, B. A., Berger, G. R., & Derbyshire, S. W. (2003). Structural and functional dichotomy of human midcingulate cortex. *European Journal of Neuroscience*, 18, 3134–3144.

LISA M. SHIN
Tufts University

KATHRYN HANDWERGER
Tufts University

See also: Biology, Brain Structure, and Function, Adult; Biology, Brain Structure, and Function, Child; Biology, Neurochemistry; Frontal Cortex; Hippocampus; Limbic System

ANTHROPOLOGICAL PERSPECTIVES

There has long been a dialogue between anthropologists and psychologists interested in trauma, dating back at least to the work of physician/anthropologist W.H.R. Rivers in the 1920s. Rivers early on recognized trauma as a legitimate way for soldiers to suffer from combat experiences. Abraham Kardiner, another key

figure in early trauma studies, who wrote an important volume on combat stress (Shepherd, 2001), had an association with one of the titans of American anthropology, Franz Boas, and his influential student Ruth Benedict at Columbia University in New York City.

Even before the term “culture bound syndrome” (Yap, 1951) was coined to describe “exotic” disorders found in the non-Western world, such as the much-debated Indonesian *Latah* (Winzeler, 1995), anthropologists have questioned the universality of Western diagnostic categories. For many years, anthropologists were concerned with seemingly bizarre disorders in non-Western contexts, largely ignoring the cultural nature of Western diagnostics. Recently, as many anthropologists have moved from a focus on culture bound syndromes to seeing all “idioms of distress” (Nichter, 1981) as culture bound, students of psychological trauma have increasingly been interested in the interactions between PTSD and globalization. This has led to an emphasis on social suffering, structural violence, and collective memory rather than solely intrapsychic distress (Kleinman, Das, & Lock, 1997).

Anthropological versus Psychological Perspectives

The key difference between anthropology and psychology is one of emphasis—culture or biology. Clifford Geertz famously wrote (2000) that people often see biology as the cake and culture as the icing. Anthropologists argue that culture is also the cake. Thus, while psychology has typically been concerned with discovering universal aspects of human behavior, anthropology has focused on the culturally imbedded nature of human experience. Diagnostic categories have often been criticized by anthropologists for their bias toward Western perspectives, and successive generations of anthropologists have tried to develop ways to describe distress within the context of locally distinct cultures. Cultural anthropology tends to either dismiss biological knowledge as reductionistic (Good, 1992; Kirmayer, 2007), avoiding challenges researchers face in acquiring expertise in biological sciences,

and the complexity involved in developing models accounting for both biological and sociocultural processes relating to trauma.

Current Perspectives on Trauma

It was not until the 1990s that anthropology began to approach trauma as an object of study. Several lines of inquiry have developed: (1) analyses of the *social construction* of and *biomedical discourses* on trauma and PTSD; (2) *critiques of the mass-trauma paradigm* used to justify humanitarian disaster interventions; and (3) *ethnographies* of collective violence, cultural trauma, identity, and memory.

In the first category (*social construction*), Allan Young’s work on the social construction of PTSD and its antecedents has become foundational and widely cited in both anthropological and psychiatric literature. His research locates and identifies the historical emergence, evolution, and logic of this psychiatric category within specific social practices and institutions, including the science of epidemiology, clinical settings, and legal and bureaucratic sites (e.g., Young, 1995). A group of researchers in England (e.g., Kilshaw, 2006) has examined the changing face of trauma for war veterans within shifting contexts of compensation, and related literature in this category has criticized the assumption that PTSD is a timeless and universal psychiatric illness by examining its historical and cultural-specificity (e.g., Bracken, Giller, & Summerfield, 1995). This work also links with the second category (*critiques of the mass-trauma paradigm*), and stems from a concern for the implications of exporting the PTSD model and associated psychosocial interventions to resource-poor countries during humanitarian emergencies. These critiques have focused on the medicalization and de-politicization of suffering, the cultural economy of victimhood, and the construction of new forms of sovereignty and governance as aspects of humanitarian aid (e.g., Breslau, 2004; Pupavac, 2004).

Research in the third category (*ethnographies*) has been the most wide-ranging. The bulk of this third line of literature examines the politics of collective memory and idioms of distress in

conflict and postconflict societies. Two notable contributions include one edited volume dedicated to the intersection between psychology and anthropology in regard to historical trauma (Robben & Suárez-Orozco, 2000) and another volume that focuses on culture, trauma, memory-making, and identity (Antze & Lambek, 1996).

Conclusion

Recently, efforts have been made to integrate the findings of neuroscience and other biological sciences with the social and cultural experiences of trauma and its aftermath (e.g., Kirmayer, Lemelson, & Barad, 2007). This promising direction takes into account anthropological critiques of diagnostic categories while maintaining the importance of biological factors.

REFERENCES

- Antze, P., & Lambek, M. (Eds.). (1996). *Tense past: Cultural essays in trauma and memory*. New York: Routledge.
- Bracken, P., Giller, J., & Summerfield, D. (1995). Psychological responses to war and atrocity: The limitations of current concepts. *Social Science and Medicine*, 40, 1073–1082.
- Breslau, J. (2004). Cultures of trauma: Anthropological views of posttraumatic stress disorder in international health. *Culture, Medicine, and Psychiatry*, 28, 113–126, 211–220.
- Geertz, C. (2000). *Available light: Anthropological reflections on philosophical topics*. Princeton, NJ: Princeton University Press.
- Good, B. (1992). Culture and psychopathology: Directions for psychiatric anthropology. In T. Schwartz, G. M. White, & C. A. Lutz (Eds.), *New directions in psychological anthropology* (pp. 181–206). Cambridge: Cambridge University Press.
- Kilshaw, S. (2006). On being a gulf veteran: An anthropological perspective. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 361, 697–706.
- Kirmayer, L., Lemelson, R., & Barad, M. (2007). *Understanding trauma: Integrating biological, clinical and cultural perspectives*. Cambridge: Cambridge University Press.
- Kleinman, A., Das, V., & Lock, M. (Eds.). (1997). *Social suffering*. Berkeley: University of California Press.
- Nichter, M. (1981). Idioms of distress, alternatives in the expression of psychosocial distress: A case study from South India. *Culture, Medicine and Psychiatry*, 5, 379–408.
- Pupavac, V. (2004). International therapeutic peace and justice in Bosnia. *Social and Legal Studies*, 13, 377–401.
- Robben, A. C. G. M., & Suárez-Orozco, M. M. (Eds.). (2000). *Cultures under siege: Collective violence and trauma*. Cambridge: Cambridge University Press.
- Shephard, B. (2001). *A war of nerves: Soldiers and psychiatrists in the twentieth century, 1914–1994*. Cambridge, MA: Harvard University Press.
- Winzeler, R. L. (1995). *Latah in Southeast Asia: The ethnography and history of a culture-bound syndrome*. Cambridge: Cambridge University Press.
- Yap, P. M. (1951). Mental diseases peculiar to certain cultures: A survey of comparative psychiatry. *Journal of Mental Science*, 97, 313–327.
- Young, A. (1995). *The harmony of illusions: Inventing posttraumatic stress disorder*. Princeton, NJ: Princeton University Press.

JOSHUA MOSES
City University of New York

KELLY MCKINNEY
McGill University

See also: Culture and Trauma; Culture-Bound Syndromes

ANTISOCIAL BEHAVIOR

High rates of both childhood trauma exposure (including physical and sexual abuse) and post-traumatic stress disorder (PTSD) have been observed among individuals who are incarcerated or who engage in antisocial behaviors such as drug and alcohol abuse, interpersonal aggression, and domestic violence. Indeed, childhood trauma and PTSD are two of the most robust and frequently examined risk factors for later antisocial behavior, and have been found to be associated with increased risk for these behaviors among both male and female

samples. However, the causal link between trauma/PTSD and later antisocial behavior remains controversial.

In addition to traumatic experiences such as physical and sexual abuse, however, other aspects of the family environment during childhood may also increase the risk for later antisocial behaviors. In particular, evidence suggests that parental neglect or rejection, inconsistent patterns of discipline, family instability (e.g., divorce or death of a parent), disruptions in the attachment relationship, and family dysfunction in general are predictive of adult perpetration of antisocial behavior. These developmental, contextual factors are thought to contribute to an intergenerational cycle of violence.

In addition to these environmental and interpersonal risk factors, individual (i.e., intrapersonal) risk factors have also been implicated in the development of antisocial behavior, including genetics, biological factors (e.g., reduced serotonin levels), and personality traits (e.g., impulsivity, affective instability, and insecure attachment). Further, recent research suggests that emotion dysregulation and distress intolerance may underlie antisocial behaviors, with these behaviors (in particular, intimate partner abuse perpetration, aggressive behaviors, and substance abuse) functioning to escape, avoid, or otherwise regulate unwanted feelings and emotional distress. In fact, research suggests that emotion dysregulation may mediate the relationship between childhood trauma and other distressing experiences and later antisocial behaviors.

Much attention has been paid to PTSD and antisocial behavior among returning combat veterans reintegrating into society. Pre-military factors (e.g., family environment, childhood conduct disorder, the presence of personality disorders [especially borderline and antisocial]) have demonstrated a direct association with postwar antisocial behaviors and represent the best predictors of interpersonal aggression following combat service. However, factors such as war zone combat exposure or negative homecoming experiences also may be related to antisocial behaviors through their relationship with PTSD, with PTSD serving as a mediating variable.

RECOMMENDED READINGS

- Bushman, B. J., Baumeister, R. F., & Phillips, C. M. (2001). Do people aggress to improve their mood? Catharsis beliefs, affect regulation opportunity, and aggressive responding. *Journal of Personality and Social Psychology*, 81, 17–32.
- Dixon, A., Howie, P., & Franzcp, J. S. (2005). Trauma exposure, posttraumatic stress, and psychiatric comorbidity in female juvenile offenders. *Journal of the American Academy of Child and Adolescent Psychiatry*, 44, 798–806.
- Dutton, D. G. (1995). Trauma symptoms and PTSD-like profiles in perpetrators of intimate abuse. *Journal of Traumatic Stress*, 8, 299–316.
- Egeland, B., Jacobvitz, J., & Paptola, K. (1989). Intergenerational continuity of abuse. In J. Lancaster & R. Gelles (Eds.), *Biosocial aspects of child abuse* (pp. 255–266). New York: Jossey-Bass.
- Fontana, A., & Rosenheck, R. (2005). The role of war-zone trauma and PTSD in the etiology of antisocial behavior. *Journal of Nervous and Mental Disease*, 193, 203–209.
- Heim, A., & Westen, D. (2005). Theories of personality and personality disorders. In J. M. Oldham, A. E. Skodol, & D. E. Bender (Eds.), *Textbook of personality disorders* (pp. 17–33). Washington, DC: American Psychiatric Publishing.
- Jang, K. L., Stein, M. B., Taylor, S., Asmundson, G. J., & Livesley, W. J. (2003). Exposure to traumatic events and experiences: Aetiological relationships with personality function. *Psychiatry Research*, 120, 61–69.
- Paris, J. (1997). Antisocial and borderline personality disorders: Two separate diagnoses or two aspects of the same psychopathology? *Comprehensive Psychiatry*, 38, 237–242.
- Stuart, G. L., Moore, T. M., Coop Gordon, K., Ramsey, S. E., & Kahler, C. W. (2006). Psychopathology in women arrested for domestic violence. *Journal of Interpersonal Violence*, 21, 376–389.

MATTHEW JAKUPCAK
Puget Sound Health Care System

KIM L. GRATZ
University of Maryland

See also: Aggression; Anger; Disruptive Behavior Disorders

ANXIETY DISORDERS

Anxiety disorders include posttraumatic stress disorder (PTSD), panic disorder (PD), generalized anxiety disorder (GAD), social anxiety disorder (SAD; or social phobia), obsessive compulsive disorder (OCD) and specific phobias (*DSM-IV*; American Psychiatric Association, 1994). This entry reviews the potential relationship of traumatic event exposure to anxiety disorders. Although many people who have anxiety disorders may have had traumatic event exposure, the causal relationships may vary by disorder.

Traumatic event exposure may serve as a primary cause, a predisposing cause, a precipitating cause, or a reinforcing cause in the development of an anxiety disorder. *Primary causes* are the conditions necessary for the disorder to occur. It is a necessary, but not always sufficient factor in the development of a disorder. *Predisposing causes* are conditions that occur prior to the onset of a disorder, which pave the way for the disorder to occur under certain conditions. These are often referred to as vulnerability factors. *Precipitating causes* are conditions that overwhelm the individual's resources to cope and trigger the disorder; and a *reinforcing cause* is a condition that maintains the disorder once it develops. These differing levels of causality will be considered.

The assessment of traumatic event exposure in people with anxiety disorders other than PTSD has not always been considered important or even relevant in prior literature. As a result, it has only been within the past 15 years that traumatic event exposure has been studied in any depth in relationship to anxiety disorders other than PTSD, which by definition includes the experience of a traumatic event. Because of this, the types of traumatic stressors that have been assessed have varied greatly from study to study and the instruments used to assess trauma have also varied widely. Some studies have used only brief questionnaires, whereas others have used well-validated instruments. The type of sample assessed has also greatly affected results. Some studies have assessed the anxiety of college students, while others have assessed specific anxiety disorders

in patient populations. Other studies have been epidemiological or have investigated large samples after mass traumas such as the September 11, 2001, terrorist attacks or major natural disasters. Still others have examined veterans returning from war.

The original National Comorbidity Survey Study (NCS) indicated that 50% to 60% of the U.S. population, ages 15 to 54, has experienced a traumatic event within their lifetime (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). Rates of anxiety disorders in the U.S. population are also quite high, with a lifetime prevalence of 29% (Kessler, Chiu, Demler, & Walters, 2005) and a 12-month prevalence of 18% for any anxiety disorder (Kessler, Berglund, et al., 2005).

It is estimated that approximately 4% of the population suffers from PTSD, 3% from PD, 3% from GAD, 7% from SAD, 1% from OCD, and 9% with specific phobias within a 12-month period (Kessler, Berglund, et al., 2005). Lifetime prevalence rates are considerably higher, with estimates of 7% for PTSD, 5% for PD, 6% GAD, 12% SAD, 2% from OCD, and 13% for specific phobias (Kessler, Chiu, et al., 2005). The relationship between traumatic event exposure and each anxiety disorder is explored next.

Posttraumatic Stress Disorder

A diagnosis of PTSD requires the presence of a traumatic stressor. Traumatic event exposure is a primary cause of PTSD by definition. Several types of stressors have been found to be most associated with PTSD, with military combat and rape having the highest probability of resulting in a PTSD diagnosis (Kessler et al., 1995). Studies have indicated that women are more likely to develop PTSD following trauma than men are, and that the more traumatic events that a person experiences, the more likely that they are to develop PTSD. It has been found that approximately 32% of women who have experienced rape, 38% of physical assault victims, 22% of those who have had a loved one murdered (Resnick, Kilpatrick, Dansky, Saunders, & Best, 1993), and 30% of

male and 27% of female veterans suffer from lifetime PTSD (Kulka, Schlenger, Fairbank, Hough, & Jordan, 1990).

Several pre-traumatic event, peri-event, and postevent characteristics have been found to influence the development of PTSD. These include factors such as previous psychiatric history, perceived life threat during the event, physical injury resulting from the event, and social support after the event (Brewin, Andrews, & Valentine, 2000).

Individuals with PTSD often present with a variety of comorbid psychiatric conditions including substance abuse, major depression, obsessive compulsive disorder, dysthymic disorder, bipolar disorder, and panic disorder (Back et al., 2000; Falsetti & Resnick, 1997; Hamner et al., 2000; Kessler, 2000; Kilpatrick et al., 2000). In fact, psychiatric comorbidity is between 2 to 6 times more likely to occur in adults with PTSD compared to adults without PTSD. PTSD when comorbid with other disorders may also negatively influence treatment response (Green et al., 2006). Data strongly support that traumatic event exposure is a primary cause of PTSD.

Panic Disorder

Traumatic event exposure is quite high in PD samples and is often comorbid with PTSD. Resnick, Falsetti, Kilpatrick, and Foy (1994) found that 90% of rape victims assessed within 72 hours post-assault met full criteria for panic attacks during that time frame. Similarly, Bryant and Panasetis (2001) found that 53% of civilian trauma survivors reportedly met criteria for panic attacks during traumatic event exposure. Reported panic symptoms during the traumatic event were significantly positively correlated with measures of acute stress disorder and panic assessed between 2 and 28 days post-event.

Data from a random sample of 1,008 residents of Manhattan at the time of the September 11 terrorists attacks indicated that a relatively large percentage of those near the disaster reportedly experienced panic attacks during the

terrorist attacks or soon after learning of the them; in fact 12% reported panic attacks acutely following the event (Galea, Ahern, Resnick, Kilpatrick, & Bucuvalas, 2002). Importantly, report of a peri-event panic attack was a significant predictor of current PTSD diagnosis in the 5 to 8 weeks following the attacks. Estimated prevalence of PTSD in that study was 8%, while 10% of the sample reported symptoms consistent with a diagnosis of current major depression. Further, those reporting a panic attack during the terror attacks were eight times more likely than those without panic to later have a PTSD diagnosis, after controlling for race, prior stressors in the previous 12 months, loss of possessions due to the attacks, and residence below or within close proximity to the World Trade Center. Report of a peri-event panic attack was also a significant predictor of current depression, after controlling for other predictors (Galea et al., 2002). Finally, these data were corroborated by Pfefferbaum, Stuber, Galea, and Fairbrother (2006), who reported similar associations of PTSD and peri-event panic attacks in adolescents. Based on research conducted thus far, it appears that trauma may be a predisposing cause of PD.

Employing Barlow's concept that panic reactions are triggered by alarms, Falsetti, Resnick, Dansky, Lydiard, and Kilpatrick (1995) proposed that for many people, a "true alarm," such as a physical assault or rape, may trigger the first panic attack. Subsequently, the panic attack is signaled by both external and internal stimuli associated with the traumatic event. Examples of external cues include places, situations, objects, smells, and sounds associated with the trauma, while internal cues include emotions, the physiological arousal experienced during traumatic events, as well as cognitions about dying or going crazy. Over time, these cues elicit a conditioned response (i.e., panic attack) that in effect becomes a "learned alarm."

Similar to Foa and Kozak's (1986) theory, Falsetti et al. (1995) proposed that external cues associated with the event, and internal cues, such as physiological arousal, thoughts, and emotions experienced at the time of the event,

comprise a fear network in one's cognitions. Activation of any component in the network may lead to an anxiety response, including panic attacks. Given that panic attacks may be triggered by internal cues, this accounts for why panic symptoms may seem to come from "out of the blue," since individuals may not perceive the symptoms as directly connected to any particular event. Thus, panic may not only develop directly from a past traumatic experience, but may be a consequence of the chronic hyperarousal noted in PTSD, which can increase vulnerability to panic by decreasing the amount of further arousal needed to reach the threshold for a panic attack to occur.

Falsetti et al. (1995) also proposed that although individuals may possess a biological predisposition for anxiety, this is not considered to be a necessary condition for development of either a conditioned emotional response or avoidance symptoms. Finally, the avoidance symptoms are hypothesized to further strengthen the associational network of conditioned cues and responses through escape-avoidance learning and lack of extinction, thereby maintaining a cycle of chronic hyperarousal and panic attacks.

Because there may be no actual physical danger at the time of these future attacks, it is also possible that when such physiological symptoms do occur when the individual becomes frightened and focuses on the arousal symptoms, thinking he or she is having a heart attack, going crazy, or dying (Barlow, 1988). In fact, evidence suggests that many people with PD demonstrate a specific hypervigilance to signs of threat (Mathews & McLeod, 1986) and are excessively preoccupied with fears of physical danger (Hibbert, 1984). In addition, Litz and Keane (1989) found that anxious subjects have an attentional bias toward threat cues. If such a theory is applied to the understanding of panic in individuals with a traumatic event history, then it would be expected that physiological cues would be considered part of that fear network and vigilance toward such cues would further increase the likelihood of future panic attacks. Falsetti and Resnick developed a treatment specifically for comorbid PTSD and panic attacks called multiple channel exposure

therapy (Falsetti, Resnick, & Davis, 2005, in press; Falsetti, Resnick, & Gibbs, 2001).

Generalized Anxiety Disorder

Research on the relationship of traumatic event exposure and GAD is sparse. Most studies that have assessed stressful life events in relation to GAD have included life events that would not meet Criterion A for PTSD in terms of traumatic event exposure. Few studies were found that examined traumatic event exposure. One study (Smith, North, McCool, & Shea, 1990) conducted with hotel workers who survived a jet plane crash into a hotel where they worked found that more than half the sample met criteria for a psychiatric disorder 4 to 6 weeks following the crash. Of the employees who were on site at the time of the crash, 29% were diagnosed with PTSD, 12% with alcohol abuse/dependence, 41% with depression, and 29% with GAD. Diagnoses for employees who were offsite at the time of the crash included 17% with PTSD, 14% with alcohol abuse/dependence, 41% with depression, and 14% with GAD. However, two-thirds of these disorders were predicted by prior psychiatric history, making it unclear to what extent the stressor contributed to the development or reemergence of these disorders.

Another study (Roemer, Molina, Litz, & Borkovec, 1997) examined 94 patients with a principal diagnosis of GAD and compared these patients to 48 nonanxious participants. They found that patients were significantly more likely to report a past potentially traumatic event than nonpatients. However, the types of events that were experienced were not reported.

In another study, the types and rates of traumatic event exposure and differences in symptom endorsement in a clinical sample of patients diagnosed with GAD were reported (Brawman-Mintzer, Monnier, Wolitzky, & Falsetti, 2005). Results indicated that 95% of the GAD sample reported a traumatic event. The most common events were natural disaster (64%), serious accident (29%), childhood sexual assault (26%), and adulthood sexual assault (21%). Trauma exposure preceded onset of GAD in 65% of patients.

At this time, given the limited data, the extent of causality of traumatic event exposure in the development of GAD is unclear.

Social Anxiety Disorder, Obsessive Compulsive Disorder, and Specific Phobias

It has long been believed that people who suffer from social anxiety disorder may have suffered from early social experiences that were traumatic (Wolpe, 1958) or aversive (Trower, Bryant, & Argyle, 1978). Despite this belief, there have been very few studies conducted that have examined traumatic event exposure in patients who suffer from social anxiety. David, Giron, and Mellman (1995) assessed childhood traumatic event histories of 51 patients with PD with agoraphobia and/or social phobia and a nonclinical comparison group of 51 participants. They found that 63% of the patient group was positive for childhood trauma compared to 3% of the nonclinical comparison group. Fifty percent of the social phobia group had a history of physical or sexual abuse compared to 2% of the patients without social phobia. The social phobia fear and avoidance subscale ratings were higher in patients with physical/sexual abuse than in patients without. The authors hypothesized that trauma exposure may interact with a genetic vulnerability and other factors to influence how phobic symptoms are expressed. This study did not assess for PTSD, so it is unknown as to how many patients may have also had comorbid PTSD.

A study of early traumatic events, parental rearing styles, family history of mental disorders, and birth risk factors in patients with social anxiety disorder found higher rates of traumatic childhood experiences in patients with social anxiety (88%) compared to health controls (48%; Bandelow et al., 2004). When the other factors assessed were statistically adjusted though, there was only a trend toward a significant contribution of childhood sexual abuse.

Social phobia does appear to be highly comorbid with PTSD when PTSD is the principal diagnosis. Zayfert, DeViva, and Hofman (2005) reported that in a sample of 443 patients seek-

ing treatment of PTSD, SP, or both, that 43% of those with a principal diagnosis of PTSD had SP. In comparison in those patients with a primary diagnosis of SP, only 7% had a comorbid diagnosis of PTSD. Another study compared traumatic event histories of patients with OCD and SAD (Fontenelle et al., 2007). They found that patients with OCD reported significantly lower rates of exposure to traumatic events compared to patients with SAD.

Finally, a twin study that evaluated the stress-diathesis model in the development of phobias did not find trauma to be associated with higher risk of developing phobias (Kendler, Myers, & Prescott, 2002). There is insufficient evidence at this time to determine a causal role of trauma in the development of social anxiety disorder, OCD, and specific phobias.

REFERENCES

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- Back, S., Dansky, B. S., Coffey, S. F., Saladin, M. E., Sonne, S., & Brady, K. T. (2000). Cocaine dependence with and without posttraumatic stress disorder: A comparison of substance use, trauma history and psychiatric comorbidity. *American Journal on Addictions*, 9, 51–62.
- Bandelow, B., Torrente, A. C., Wedekind, D., Broocks, A., Hajak, G., & Ruther, E. (2004). Early traumatic life events, parental rearing styles, family history of mental disorders, and birth risk factors in patients with social anxiety disorder. *European Archives of Psychiatry Clinical Neuroscience*, 254, 397–405.
- Barlow, D. H. (1988). *Anxiety and its disorders*. New York: Guilford Press.
- Brawman-Mintzer, O., Monnier, J., Wolitzky, K. B., & Falsetti, S. A. (2005). Patients with generalized anxiety disorder and a history of trauma: Somatic symptom endorsement. *Journal of Psychiatric Practice*, 212–215.
- Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology*, 68, 748–766.

- Bryant, R. A., & Panasetis, P. (2001). Panic symptoms during trauma and acute stress disorder. *Behavior Research and Therapy*, 39, 961–969.
- David, D., Giron, A., & Mellman, T. A. (1995). Panic-phobic patients and developmental trauma. *Journal of Clinical Psychiatry*, 56, 113–124.
- Falsetti, S. A., & Resnick, H. S. (1997). Frequency and severity of panic attack symptoms in a treatment seeking sample of trauma victims. *Journal of Traumatic Stress*, 10(4), 683–689.
- Falsetti, S. A., Resnick, H. S., Dansky, B. S., Lydiard, R. B., & Kilpatrick, D. G. (1995). The relationship of stress to panic disorder: Cause or effect. In C. M. Mazure (Ed.), *Does stress cause psychiatric illness?* (pp. 111–147). Washington, DC: American Psychiatric Press.
- Falsetti, S. A., Resnick, H. S., & Davis, J. L. (2005). Multiple channel exposure therapy: Combining cognitive behavioral therapies for the treatment of posttraumatic stress disorder with panic attacks. *Behavior Modification*, 29, 70–94.
- Falsetti, S. A., Resnick, H. S., Davis, J. (in press). An investigation of the long-term effectiveness of multiple channel exposure therapy for the treatment of PTSD with comorbid panic attacks. *Depression and Anxiety*.
- Falsetti, S. A., Resnick, H. S., & Gibbs, N. A. (2001). Treatment of PTSD with panic attacks combining cognitive processing therapy with panic control treatment techniques. *Group Dynamics: Theory, Research and Practice*, 5, 252–260.
- Foa, E. B., & Kozak, M. J. (1986). Emotional processing of fear: Exposure to corrective information. *Psychological Bulletin*, 99, 20–35.
- Fontenelle, L. F., Domingues, A. M., Souza, W. F., Mendlowicz, M. V., de Menezes, G. B., & Figueira, I. L. (2007). History of trauma and dissociative symptoms among patients with obsessive-compulsive disorder and social anxiety disorder. *Psychiatric Quarterly*, 78, 241–250.
- Galea, S., Ahern, J., Resnick, H., Kilpatrick, D., & Bucuvalas, M. (2002). Psychological sequelae of the September 11 terrorist attacks in New York City. *New England Journal of Medicine*, 346, 982–987.
- Green, B. L., Krupnick, J. L., Chung, J., Siddique, J., Krause, E. D., Revicki, D., et al. (2006). Impact of PTSD comorbidity on one-year outcomes in a depression trial. *Journal of Clinical Psychology*, 62, 815–835.
- Hamner, M. B., Frueh, B. C., Ulmer, H. G., Huber, M. G., Twomey, T. J., Tyson, C., et al. (2000). Psychotic features in chronic posttraumatic stress disorder and schizophrenia: Comparative severity. *Journal of Nervous and Mental Disease*, 188, 217–221.
- Hibbert, G. A. (1984). Ideational components of anxiety: Their origin and content. *British Journal of Psychiatry*, 144, 618–624.
- Kendler, K. S., Myers, J., & Prescott, C. A. (2002). The etiology of phobias: An evaluation of the stress-diathesis model. *Archives of General Psychiatry*, 59, 242–248.
- Kessler, R. C. (2000). The epidemiology of pure and comorbid generalized anxiety disorder: A review and evaluation of recent research. *Acta Psychiatrica Scandinavica Supplement*, 406, 7–13.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62, 593–602.
- Kessler, R. C., Chiu, W. T., Demler, O., & Walters, E. E. (2005). Prevalence, severity, and comorbidity of 12-month DSM-IV disorders in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 62, 617–709.
- Kessler, R. C., Sonnega, A., Bromet, E., Hughes, M., & Nelson, C. B. (1995). Posttraumatic stress disorder in the National Comorbidity Survey. *Archives of General Psychiatry*, 52, 1048–1060.
- Kilpatrick, D. G., Acierno, R., Saunders, B., Resnick, H. S., Best, C. L., & Schnurr, P. P. (2000). Risk factors for adolescent substance abuse and dependence: Data from a national sample. *Journal of Consulting and Clinical Psychology*, 68, 19–30.
- Kulka, R. A., Schlenger, W. E., Fairbank, J. A., Hough, R. L., & Jordan, B. K. (1990). *Trauma and the Vietnam war generation: Report of findings from the National Vietnam Veterans Readjustment Study*. New York: Brunner/Mazel.
- Litz, B. T., & Keane, T. M. (1989). Information processing in anxiety disorders: Application to the understanding of post-traumatic stress disorder. *Clinical Psychology Review*, 9, 243–257.
- Mathews, A. M., & MacLeod, C. (1986). Discrimination of threat cues without awareness in anxiety states. *Journal of Abnormal Psychology*, 95, 131–138.
- Pfefferbaum, B., Stuber, J., Galea, S., & Fairbrother, G. (2006). Panic reactions to terrorist attacks and

- probable posttraumatic stress disorder in adolescents. *Journal of Traumatic Stress*, 19, 217–228.
- Resnick, H. S., Falsetti, S. A., Kilpatrick, D. G., & Foy, D. W. (1994, November). *Associations between panic attacks during rape assaults and follow-up PTSD or panic attack outcomes*. Paper presented at the 10th annual meeting of the International Society for Traumatic Stress, Chicago.
- Resnick, H. S., Kilpatrick, D. G., Dansky, B. S., Saunders, B. E., & Best, C. L. (1993). Prevalence of civilian trauma and posttraumatic stress disorder in a representative national sample of women. *Journal of Consulting and Clinical Psychology*, 61, 984–991.
- Roemer, L., Molina, S., Litz, B. T., & Borkovec, T. D. (1997). Preliminary investigation of the role of previous exposure to potentially traumatizing events in generalized anxiety disorder. *Depression and Anxiety*, 4, 134–138.
- Smith, E. M., North, C. S., McCool, R. E., & Shea, J. M. (1990). Acute postdisaster psychiatric disorders: Identification of persons at risk. *American Journal of Psychiatry*, 147, 202–218.
- Trower, P., Bryant, B. M., & Argyle, M. (1978). *Social skills and mental health*. London: Methuen.
- Wolpe, J. (1958). *Psychotherapy and reciprocal inhibition*. Stanford, CA: Stanford University Press.
- Zayfert, C., DeViva, J. C., & Hofman, S. G. (2005). Comorbid PTSD and social phobia in a treatment seeking population. *Journal of Nervous and Mental Disease*, 193, 93–101.

SHERRY A. FALSETTI

University of Illinois College of Medicine at Rockford

See also: Anxiety Management Training; Comorbidity; Posttraumatic Stress Disorder

ANXIETY MANAGEMENT TRAINING

The conceptual framework underlying anxiety management training is rooted in theories of stress and coping. In this model, stress is neither an environmental event nor the person's emotional and behavioral response to the environment. Rather, stress results from the *interaction* of the person and the environment when events are experienced as exceeding or

taxing one's coping resources and thus causing a threat to one's welfare. In this transactional view, stress is also seen as an inevitable aspect of life that cannot be eliminated. Anxiety is a normal response to stress and is often useful in motivating coping behavior. However, anxiety can become excessive and disruptive, as is the case for people who have developed persistent posttraumatic stress symptoms.

Anxiety management training is a set of cognitive-behavioral interventions that have been applied with psychological trauma survivors for several decades (Rothbaum, Meadows, Resick, & Foy, 2000). Its use increased significantly when posttraumatic stress disorder (PTSD) was formally codified as an anxiety disorder by the American Psychiatric Association in 1980. At that time, some cognitive behavioral researchers and clinicians viewed PTSD as a complex phobia with extensive generalization (i.e., in which fear responses spread, or "generalized" to stimuli or circumstances beyond the original stressful experiences), and thus applied interventions that were known to be effective for pathological anxiety. These included exposure therapy procedures (see: **Exposure Therapy, Adult**) that had been found successful in the treatment of phobias, and anxiety management procedures, known to be successful for those with generalized anxiety. Participants in anxiety management programs were often female survivors of assault or military combat veterans.

One such program that has been commonly employed in the past 30 years is stress inoculation training (SIT; Meichenbaum, 1975, 1985). SIT, which provides techniques or skills that the person can use to manage and reduce anxiety, is a relatively well-researched program that was used in early studies of treatments for female rape and crime victims. The goal of stress inoculation treatment is to teach the person to understand the dynamics of stress and to develop or improve intrapersonal and interpersonal skills for managing stress reactions. These coping skills, which typically include breathing and relaxation training, cognitive restructuring, guided (task-enhancing) self-dialogue, assertiveness training, role-playing, and covert modeling, help the person to manage

her reactions to trauma-related cues or situations. Treatment usually begins with discussing the person's stress and anxiety from the transactional perspective, and the rationale or conceptual groundwork for the skills training is laid down. The next phase includes training and practice of coping skills. The core skills of SIT are central to most other anxiety management programs, and are briefly described next.

Breathing Training

The therapist gives specific instructions for reducing tension by slowing the rate of respiration and pairing breathing with a cue for calming and relaxing the mind (e.g., silently and slowly drawing out the word "calm" or "relax" while exhaling very slowly). Typically, the therapist models this slow breathing pattern, and then observes the person practice and provides appropriate feedback. Sometimes a tape recording is made of the therapist guiding the person through a number of respiratory cycles for the person to practice at home. Homework is given to practice the skill several times daily in order to develop its usefulness in reducing tension and managing anxiety.

Relaxation Training

There are many methods of relaxation, but the one that is utilized in SIT and was commonly used in early programs for trauma survivors is progressive muscle relaxation (PMR). During PMR training the person is taught to systematically tense and then relax specific muscle groups throughout the body, while focusing attention on the contrast, or how these muscles feel when tense and when relaxed. The goal is to learn to identify excess muscular tension and to eliminate it when detected. The therapist typically records the relaxation training instructions for the client's use in daily practice at home.

Cognitive Restructuring

Cognitive restructuring is based on cognitive theory, which hypothesizes that it is the *interpretation* of events, rather than events

themselves, which leads to specific emotional responses such as anxiety. Using an example that is commonly experienced by psychological trauma survivors, when "safe" or harmless events are viewed as threatening, unrealistic or excessive anxiety results. The aim of cognitive restructuring is to help the client to understand the role of his or her beliefs and interpretations in influencing emotional reactions, to identify the ways that the traumatic event or experience has influenced his or her beliefs and expectations (especially those that trigger excessive negative emotions such as fear, anger, and shame), and to learn to challenge and modify these beliefs and expectations in a rational, evidence-based manner.

Guided Self-Dialogue

The way we think and messages that we give ourselves can help or hinder our management of stressful situations. Guided self-dialogue teaches the person to focus on her or his internal dialogue, or on what he or she "is saying" to her/himself, with the objective of replacing irrational, self-critical, or negative dialogue with rational, facilitative, and task-enhancing dialogue. The therapist and client focus on coping with stressful events in a series of steps: (1) preparation, (2) confrontation and management, (3) coping with feelings of being overwhelmed, and (4) reinforcement. For each step, the patient and therapist generate a series of questions and/or statements which encourage the patient to: (1) assess the actual probability of a negative event happening, (2) manage overwhelming avoidance behavior, (3) control self-criticism and self-devaluation, (4) engage in a feared behavior, and (5) reinforce her/himself for attempting the behavior and for following the plan.

Behavioral Rehearsal

Behavioral rehearsal is a means of learning new or developing existing behaviors to replace old (less effective) ways of responding to stressful situations, and provides a chance to practice the new behaviors before the "real-life" event occurs. The client and therapist act out scenes (either in

imagination or in role-play) in which the client confronts a difficult or stressful situation. The premise is that repeated practice of a behavior reduces anxiety and makes it more likely that a new behavior will be used when it is called for.

Covert modeling is a behavioral rehearsal technique that permits practice of the desired behaviors or coping response via imagination (i.e., covert). First the therapist and client select a stressful or difficult situation to work on. Often the therapist will then model successful coping: closing her or his eyes and describing aloud what he or she is visualizing, the therapist explains how he or she is utilizing anxiety management skills during the difficult situation and successfully works through it. The client then takes a turn and visualizes her/himself coping successfully with the situation, describing aloud her/his imaginal use of the skills. If suitable, scenes used for covert modeling are sometimes those later utilized for role-play practice in session.

Role-play involves the acting out of behaviors, and rehearsal of responses, while pretending to be in a particular situation. During role-play, as in covert modeling, it is common for the therapist to first play the client's role and model appropriate social skills. Next roles are reversed with the client playing himself. Each role-play is discussed and the client is encouraged to point out positive aspects of his performance as well as areas that can be improved. Role-plays are repeated with the goal of shaping desired behavior and developing better skill through practice.

Empirical Research on Anxiety Management Training

Randomized controlled research studies comparing various forms of cognitive behavior therapy (CBT) for psychological trauma survivors with PTSD have shown that stress inoculation training is efficacious at reducing PTSD as well as other trauma-related symptoms (e.g., depression), particularly among women who have survived sexual assault (Foa et al., 1999; Foa, Rothbaum, Riggs, & Murdock, 1991). These studies have found stress inoculation training to be fairly comparable to exposure

therapy and more effective than control conditions such as waitlist and supportive counseling. In two studies conducted with veterans, stress inoculation training was not as strongly supported, because one study found that veterans treated with stress management in group format did not improve in terms of reductions in PTSD (and neither did the trauma-focused group in that study; Monson, Rodriguez, & Warner, 2005) although the other study found stress management (focused heavily on anger management) more effective than routine clinical care in reduction of anger and PTSD intrusive reexperiencing symptoms (Chemtob, Novaco, Harnada, & Gross, 1997). Several other studies have used single anxiety management skills such as relaxation training, bio-feedback, and assertion training as comparison conditions, but failed to find them efficacious. For example, three randomized trial research studies have found relaxation training to be less efficacious than exposure therapy, cognitive therapy, and their combination.

Conclusion

Research studies have generally found that anxiety management skills training is effective at reducing psychological trauma-related pathology in female assault survivors but perhaps less effective when used with male military veterans. Stress inoculation training has been studied less extensively than other forms of CBT, however, and most studies involving SIT were conducted in the 1980s and 1990s. This may be in part due to a shift from earlier, heavily behavioral conceptualizations of PTSD to formulations that emphasize the role of cognition in explaining the development and maintenance of chronic traumatic stress-related disorders.

Thus, while anxiety management approaches showed early promise at reducing traumatic stress-related symptoms, most psychological trauma researchers moved on to investigating treatments that address the factors that are thought to maintain posttraumatic symptoms and difficulties, and thus may offer deeper or longer-lasting change in traumatic stress-related symptoms. These treatments—exposure therapy

(see: **Exposure Therapy, Adult**) and cognitive therapy (see: **Cognitive Behavior Therapy, Adult**)—are designed to help the survivor to emotionally process his or her memories of traumatic experiences and to modify the unhelpful or inaccurate beliefs about the world and the self that may be maintaining traumatic stress-related fears and other symptoms.

REFERENCES

- Chemtob, C. M., Novaco, R. W., Harnada, R. S., & Gross, D. M. (1997). Cognitive-behavioral treatment for severe anger in PTSD. *Journal of Consulting and Clinical Psychology*, 65(1), 184–189.
- Foa, E. B., Dancu, C. V., Hembree, E., Jaycox, L. H., Meadows, E. A., & Street, G. P. (1999). The efficacy of exposure therapy, stress inoculation training and their combination in ameliorating PTSD for female victims of assault. *Journal of Consulting and Clinical Psychology*, 67, 194–200.
- Foa, E. B., Rothbaum, B. O., Riggs, D., & Murdock, T. (1991). Treatment of PTSD in rape victims: A comparison between cognitive-behavioral procedures and counseling. *Journal of Consulting and Clinical Psychology*, 59, 715–723.
- Meichenbaum, D. (1975). Self-instructional methods. In F. H. Kanfer & A. P. Goldstein (Eds.), *Helping people change* (pp. 357–391). New York: Pergamon Press.
- Meichenbaum, D. (1985). *Stress inoculation training*. New York: Pergamon Press.
- Monson, C. M., Rodriguez, B. F., & Warner, R. (2005). Cognitive-behavioral therapy for PTSD in the real world: Do interpersonal relationships make a real difference? *Journal of Clinical Psychology*, 61, 751–761.
- Rothbaum, B. O., Meadows, E. A., Resick, P., & Foy, D. W. (2000). Cognitive-behavioral therapy. In E. Foa, T. Keane, & M. Friedman (Eds.), *Effective treatments for PTSD: Practice guidelines from the International Society for Traumatic Stress Studies* (pp. 320–325). New York: Guilford Press.

ELIZABETH A. HEMBREE
University of Pennsylvania

See also: **Anxiety Disorders; Cognitive Behavior Therapy, Adult; Cognitive Behavior Therapy, Child Abuse; Coping Skills Training; Exposure Therapy, Adult; Exposure Therapy, Child; Psychoeducation**

ARTISTIC DEPICTIONS OF PSYCHOLOGICAL TRAUMA

The human experience of psychological trauma finds expression in many forms. The very symptoms of posttraumatic stress disorder (PTSD) themselves are often described as a form of expression because they are understood by some to communicate the complexities of the traumatic experience in ways that are physical, behavioral, emotional, and cognitive. The verbal expression of the traumatic experience can be terribly difficult and frustrating to accomplish. Because of the intensely disturbing and degrading aspects of traumatic events, and the intimately personal nature of some of the concomitant violations, verbally expressing traumatic experiences may require finding words for the ineffable and speaking the unspeakable (see: **Literary Depictions of Psychological Trauma**). Nonverbal artistic forms of creative expression therefore have been an important set of media for depictions of the nature and effects of psychological trauma.

Dating back to the earliest cave paintings, art has stood as a means for capturing and expressing the human experience with a power beyond words. Artistic depictions of events display more than a setting and scene. They convey emotions and layers of meaning in a manner that is both veridical and symbolic, while avoiding some of the confusions, clichés, and reductions to which words are so vulnerable. This is not to demean all written and verbal depictions of psychological trauma, but rather to admire the courage and skill of those who find with words and phrases a means of expressing to others what is so deeply painful and so painfully necessary to convey. But among the most powerful characteristics of the visual arts is their ability to convey the depths of the human experience beyond the limits of words and the impediments of language. The prehistoric paintings preserved in France on the cavernous walls beneath Lascaux have long outlived whatever languages may have been spoken by their creators and demonstrate the profound capacity for artistic depictions of the human experience to transcend time, language, and culture.

When portraying psychological trauma, artists and photographers work at the intersection of mind and emotion to visually represent what is by definition an unimaginable and unbearable shock. The visual artist applies paint, line, color, a lens, or a blend of media to the subject, expressing either a personal traumatic experience, or attempting to depict the traumatic experiences or reactions of others. These artistic depictions have the latitude for conveying the psychologically traumatic experience from any angle of view and within any imaginable frame of reference. The depicted scene may be broad in scope, or narrowed to a very fine point. Viewers may be drawn into small details and variations of texture, or cast back to vantage points where the scene's entirety can be viewed in full. Moreover, the psychological impact of the trauma on a previously organized perspective can be conveyed through varying degrees of distortion, as is evident in the works of the Cubists and Surrealists.

To whom does the artist address this visual depiction of psychological trauma? Victims? Perpetrators? Bystanders? Few artists reveal their explicit purpose in bringing to the public eye a work that is ultimately about personal horror. And many people consider psychological trauma a private matter owned only by the victims, as for instance in cases of child abuse, rape, or domestic violence. These subjects are found less often in art galleries than in very personal works meant more often for expressing than for sharing. But Plutarch's tale of the public abduction of conquered women by Romans seeking wives, *The Rape of the Sabine Women*, was rendered in Giambologna's sixteenth-century writhing sculpture and painted in classically stylized scenes of chaotic frenzy a century later by Poussin and Rubens, and in the eighteenth century by David. Much later, in the early 1960s, Pablo Picasso took inspiration from David's vision of this event, which he transformed by stripping away its classic patina to reveal a scene of crushing violence and domination. Working in a related vein in the twenty-first century, Chinese artist Li Hu created the *Rape of Nanking; The Forgotten Holocaust* (2005), a large and powerful exhibition of his artwork

depicting the horrors that ensued for the Chinese people when the Imperial Japanese Army laid siege to the city in 1937.

Art across time and cultures shows a long fascination with psychological trauma. From depictions of inhuman evil forces in early allegorical paintings of demons and devils, to the real blood and gore of the battlefield, artists and photographers have recorded what is most feared and most vicious. American photographer and writer Susan Sontag pointed out in her essay *Regarding the Pain of Others* (2003) that in the art of trauma, the primary focus is on the gruesome abuses humans inflict on each other, rather than on the miseries of the human condition itself—such as illness, poverty, or natural disasters. “In each instance, the gruesome invites us to be spectators or cowards, unable to look” (p. 42), Sontag wrote, emphasizing the classical role of torment as spectacle in art, where often onlookers depicted in the painting itself are either horrified or impervious to the scene. While the “evil that men do” may be the most recurrent theme in artistic depictions of psychological trauma, they also include portrayals of traumatic events that are the result of larger forces such as natural disasters. For example, English painter J. M. W. Turner's graphic *Slavers Throwing Overboard the Dead and Dying, Typhoon Coming On* (1840), shows the torment of drowning slaves abandoned to a raging sea. Traumatic art and photography objectify, bringing a grand scale event to within the imagination of an individual viewer, condensing all that has transpired in the event to a single, instantaneous shock.

This is made strikingly evident in Picasso's *Guernica*, a massive painting commissioned by the Spanish government for its Pavilion at the 1937 World's Fair. Picasso (1881–1973), distressed by personal problems and the ugly civil war in his native Spain, was appalled by a vicious air attack on the village of Guernica that murdered or wounded some 1,600 civilians. He began work the day he learned of the bombing. The seven-meter wide, horizontal painting is executed only in black and white in an angular, modernist style, with central images of a bull, wounded horse, and harsh

light bulb over wounded and dying people in agony. Sympathizers exhibited the piece widely to call attention to the Fascist conspiracy that massacred the people of Guernica. Both as an artistic wonder and as a political instrument, the piece became an icon of twentieth century art as protest.

Picasso was certainly influenced by his countryman and predecessor Francisco Goya (1746–1828), who chronicled in excruciating realism humanity gone mad during the Napoleonic invasion of Spain. Torn between the morality of liberating Spain from a despotic monarchy and the brutalities with which it was done, he painted and etched a prolific number of works, as if to empty his own mind of emotions in turmoil. His disturbingly graphic series entitled *Disasters of War*, begun in 1810, numbers over 100 prints and drawings. Goya produced it after the conflict was long over and throughout a decade, as if his memory of the savagery would not stop churning out new evidence. His painting, *The Third of May 1808* (1814), stands among the greatest works of art in its depiction of a lantern-lit scene where rebels stand helpless as soldiers execute them by firing squad in the dark of night.

Working without the restrictions that the state and church imposed on early painters, Fernando Botero (b. 1932), a Columbian artist, tackled the highly controversial incident of prisoner torture in his 2005 series *Abu Ghraib*. Botero's unique neo-figurative style typically presents bulbous high-society people in prettified contexts, a presumed commentary on the excesses of the moneyed class. In *Abu Ghraib*, Botero carries his overfed characters into prison, with wrenching scenes he built from newspaper photographs and accounts of actual abuse inflicted by American troops on Iraqi prisoners. Beware, he seems to say, this can happen to any of us if we let it.

Using experiential art forms, many contemporary artists have moved from simply depicting traumatic scenes to actually placing the viewer in them, a shift Australian Professor Jill Bennett (2005) describes as a move from communicative to transactive art fostering "empathic vision." In part, this answers the

development of the photographic image and subsequent ascension of live television as recorded truth in the matters of war and violence rather than artistic imagination. The camera lens installs the viewer in the scene, or brings it home, so to speak. This artistic trend grew in parallel to the recognition of traumatic stress disorders such as PTSD by the mental health and medical fields as a diagnosis of incapacitating mental anguish. In art as well as psychology, the notion of "secondary or vicarious trauma" (see: **Vicarious Traumatization**) appeared, recognizing that bystanders and viewers, too, could be affected by the force of the images of psychological trauma.

The American artist Jasper Johns (b. 1930) produced a series of paintings and mixed media works in the 1950s featuring prominent, ringed targets that exemplify the beginning of this trend. Art critic Robert Hughes (1997) points out that this was the era of the Cold War, when the threat of nuclear apocalypse haunted the world, and particularly Americans, toward whom—they were relentlessly reminded by the press and politicians—the Soviet Union pointed its missiles. In *Target with Four Faces* (1955), half-hidden plaster faces peer out from tight boxes with hinged doors above a large bulls-eye, evoking collective fear and a sense of being trapped and inevitably doomed. But it is the viewer who stands in the position of aiming the weapon at the target, becoming the aggressor, inflicting the fear on the hiding faces.

A similar message of a public and private conscience for mass traumatic loss infuses visitors to the Vietnam veterans memorial, known as The Wall, in Washington, DC. It is a prime example of a *countermonument*—a term coined by American Professor James Young to describe a memorial in structural form that is marked by its context, a feeling of absence completed by the viewer's participation and thus constant redefinition. Maya Lin, a Chinese-American artist, was 21 years old at the time she submitted her drawings in a blind competition for the memorial design. In antithesis of many towering war monuments, the memorial is cut into the earth and simply carries on its wall of tablets the carved names of the 58,000 U.S.

soldiers lost in the protracted conflict. As a visitor descends the pathway to walk alongside The Wall, the black polished marble reflects his or her own face rippling across the names, thus uniting witness and victim, and drawing strong emotions in the process.

The personalization of psychological trauma in art is a process enveloping many artists in their own work. In a struggle to live with their own suffering, or the pain of their loved ones or community, they are both witness and portrayer as they commit to an image. Joerg Bose (2005), in discussing the disturbing self-portraits of Mexican painter Frida Kahlo (1917–1954), refers to this intrapsychic duality. He believes that for a painter such as Kahlo, who suffered immeasurable physical and emotional pain in her life, her ability to paint her pain into images to express the horror of her existence may have supported her ability to survive. She often painted dual images in her self-portraits, such as in *The Two Fridas* (1939). The painting depicts her two selves dressed beautifully, seated formally side-by-side and holding hands, turned toward the viewer without expression. The power of the painting comes from the juxtaposition of this emotionless gaze with the two figures' hearts depicted anatomically on their chests as if surgically removed and placed there, with veins intertwined and one Frida holding scissors that sever the end of a vein which is bleeding onto her white dress. It is a painting that captures not only the overpowering presence of psychological trauma but also the emotional emptiness that is often left behind in its wake, a common PTSD symptom. Translating this psychic void into a work of art gives form and substance to the feelings of loss in two ways, both of which can ease suffering, according to psychoanalysts Dori Laub and Daniel Podell (1995). It allows the individual to shift from being consumed by the memories of the traumatic experience to entering into an internal dialogue with the experience as represented. The representation of the lived experience can also become a shared burden with others as witnesses, as much of the art of the Holocaust has served to do, in enhancing the *knowing* of psychological trauma.

Tanzanian artist and writer Everlyn Nicodemus (b. 1954) finds ways large and small to bring the subject of cultural trauma into this knowing. In her *Reference Scroll on Genocide, Massacre and Ethnic Cleansing* (2004), she draws on I. W. Charny's *Encyclopedia of Genocide* (1999) to meticulously record on a seemingly unending scroll the atrocities rained down upon peoples of vulnerable ethnic, social and cultural communities. The force of the work originates in the choice of a scroll's form, suggesting a religious inscription and thus a deeply held and timeless human value. Nicodemus also addresses her personal knowing of one person's trauma, represented hauntingly in *Birthmask*, a work in mixed media showing a frozen scream of horror on a traditionally dressed doll figure. The doll is flattened, with outstretched arms bound by a taut mesh web barring escape. Below the doll's waist, a large round hole gapes with only slightly torn edges, as if punched out methodically. While the meaning of the work is indeterminate because of its entirely nonverbal presentation, Nicodemus clearly conveys a sense of both psychic and physical entrapment and damage that is consistent with the impact of psychological trauma.

Around the world, the processing of strong emotion underlies the use of art to give voice to traumatized individuals and communities. In South Africa, women of the impoverished Mapula region embroider quilts with scenes of domestic violence, child abuse, and AIDS, challenging the taboos of public discussion (Schmahmann, 2005). Polish artist and architect Krzysztof Wodiczko films interviews with survivors of rape, police brutality, and the nuclear holocaust of World War II and projects the faces large-scale onto public monuments, where they demand attention. In the Philippines, an exhibit by Asian women artists entitled "Trauma, Interrupted" (Manila, 2007) displays art drawing attention to forced sexual slavery of some 200,000 Asian women during World War II.

In a very different style, two contemporary artists have used the form of graphic novels, or drawn stories similar to cartoons, to depict psychological trauma in a way that blends

visual images with verbal expressions of satire and irony to traumatic experiences of the Holocaust and political upheaval in Iran. Art Spiegelman (b. 1948), the American son of a Holocaust survivor, won the 1992 Pulitzer Prize with his *Maus: A Survivor's Tale* (1986), which used cartoon animals to symbolize the Germans, Jews, Russians, Americans, and other historical participants in the Holocaust. The tale recounts his father's struggles, but his own as well, as a generation once removed but still very much impacted by the Holocaust. Marjane Satrapi (b. 1969), an Iranian woman who lived through the violent revolution from the end of the Shah's regime to Islamic fundamentalists, has also drawn her memories into an autobiographical cartoon in *Persepolis: The Story of a Childhood* (2003), which was later animated for a film. In both cases, the subject matter is deeply experienced trauma, but the injection of humorous affect holds it at arm's length, as if to better examine it, manipulate it with symbols to attenuate the shock, and see what can be understood when the overwhelming emotional burden is temporarily relieved.

When the artist gives us a visual language for psychological trauma, a point of view is involved. Similarly, a photographer may crop a photo, or otherwise frame a point of view. Botero painted his *Abu Ghraib* series to show the point of view of the prisoner and his agony, contrary to the newspaper photos that concentrated on the soldiers delivering the abuse. Spiegelman and Satrapi give us new ways of seeing old pain, and Johns and Lin compel us to participate in the scene. Thus, through creative application of image, tonality, texture, and light, artists render personal and historic horrors that illuminate humanity's darkest recesses, with a power to pierce the hidden depths of our vulnerability.

REFERENCES

- Bennett, J. (2005). *Empathic vision: Affect, trauma, and contemporary art*. Stanford, CA: Stanford University Press.
- Bose, J. (2005, Spring). Images of trauma: Pain, recognition, and disavowal in the works of Frida Kahlo and Francis Bacon. *Journal of the American Academy of Psychoanalysis and Dynamic Psychiatry*, 33(1), 51–70.
- Charny, I. W. (Ed.). (1999). *Encyclopedia of genocide* (Vols. 1 & 2). Santa Barbara, CA: ABC-CLIO.
- Hughes, R. (1997). *American visions: The epic history of art in America*. New York: Random House.
- Laub, D., & Podell, D. (1995). Art and trauma. *International Journal of Psychoanalysis*, 76, 995–1005.
- Satrapi, M. (2003). *Persepolis: The story of a childhood*. New York: Pantheon Books.
- Schmahmann, B. (2005, Autumn). Stitches as sutures: Trauma and recovery in works by women in the Mapula embroidery project. *African Arts*, 38(33), 52–65.
- Sontag, S. (2003). *Regarding the pain of others*. New York: Picador.
- Spiegelman, A. (1986). *Maus: A survivor's tale*. New York: Pantheon Books.
- JANET DE MERODE
Fielding Graduate University
- GILBERT REYES
Fielding Graduate University
- See also: Literary Depictions of Psychological Trauma; Movie Depictions of Psychological Trauma; Vicarious Traumatization

ASSESSMENT, PSYCHOMETRIC, ADULT

The use of standardized, psychometrically sound self-report measures and structured interviews is the foundation of the evidence-based assessment of trauma exposure and posttraumatic stress disorder (PTSD). Such measures provide an efficient, cost-effective means for collecting essential information regarding an individual's unique responses to catastrophic life events, and are administered routinely to accomplish a wide range of clinical and research assessment tasks, including screening for trauma exposure and PTSD, establishing a PTSD diagnosis, quantifying PTSD symptom severity, evaluating comorbid emotional problems, evaluating response bias, and gathering information relevant for case conceptualization and treatment planning.

The development and empirical evaluation of psychometric measures has been one of the most active and productive areas in the field of traumatic stress. Dozens of checklists, questionnaires, and interviews are now available, a growing number of which have been extensively validated in diverse settings and trauma populations. Recent reviews of the various measures and guidelines for their application are available in Briere (2004) and Wilson and Keane (2004). Also of interest, Elhai, Gray, Kashdan, and Franklin (2005) recently reported the prevalence of use of the various measures among trauma professionals. In the next two sections, several of the most widely used interviews and self-report measures of PTSD and trauma exposure are briefly described. In the last section, recommendations for conducting evidence-based assessments of PTSD are provided.

Assessment of PTSD

PTSD measures vary substantially in format, most notably with respect to method of administration (interview versus self-report), number of items, item content, response dimension (e.g., symptom frequency, subjective distress), number of response or rating options, time frame (e.g., past week, past month), and whether symptoms are linked explicitly to a specific traumatic event. A key difference among measures is the extent to which they correspond to the diagnostic criteria for PTSD in the *DSM-IV-TR* (American Psychiatric Association, 2000). All of the interviews and many of the self-report measures are *DSM-IV*-correspondent (American Psychiatric Association, 1994), with items conforming to the 17 *DSM-IV* (American Psychiatric Association, 1994) symptoms of PTSD, but several self-report measures are not, although they tap trauma-relevant symptoms.

The choice of a PTSD measure for a given application depends primarily on the purpose of the assessment. Structured interviews, particularly those that yield both a diagnosis and a continuous measure of symptom severity, are essential whenever PTSD is the major focus of a study or clinical assessment and

whenever a formal PTSD diagnosis is required. A structured interview administered by an experienced clinician is considered the “gold standard” or most widely accepted criterion measure for PTSD assessment. When an interview is not feasible, *DSM-IV* correspondent (American Psychiatric Association, 1994) self-report measures can be substituted, and are particularly useful for screening, large-scale surveys, and ongoing monitoring of symptom severity, as in treatment outcome research. PTSD-focused but non-*DSM-IV*-correspondent (American Psychiatric Association, 1994) self-report measures can be useful supplements as part of a battery of measures, but whenever possible should be used in conjunction with interviews and *DSM-IV*-correspondent (American Psychiatric Association, 1994) self-report measures.

Structured Interviews

There are several widely used, well-validated structured interviews for PTSD. They vary in format, administration, and scoring, as well as in the nature of the assessment information they yield. Valid administration and scoring of these interviews requires a strong background in psychopathology, proficiency in diagnostic interviewing and differential diagnosis, and a thorough understanding of psychological trauma and the clinical phenomenology of PTSD. Therefore, an adequate description of an interview, or explicit operational definition of its use in a given context, must include not only the features of the interview *per se*, but also the qualifications of the interviewer.

Structured Clinical Interview for DSM-IV

The Structured Clinical Interview for *DSM-IV* (SCID; First, Spitzer, Gibbon, & Williams, 1996; *see: Structured Clinical Interview for DSM-IV—Posttraumatic Stress Disorder Module*) is a comprehensive structured interview that assesses all the major *DSM-IV* (American Psychiatric Association, 1994) disorders. Its PTSD module can be administered in the context of a complete administration of the

SCID, but is often administered as a stand-alone module. As with other SCID modules, the PTSD module directly assesses each of the *DSM-IV-TR* (American Psychiatric Association, 2000) diagnostic criteria. The module begins with a brief screening question that evaluates possible exposure to traumatic life events. This screening is followed by two questions that identify an index event for symptom inquiry and determine whether that event meets the definition of a trauma according to *DSM-IV* (American Psychiatric Association, 1994) PTSD's Criterion A. Next is the symptom inquiry section, which consists of single prompt questions for each of the 17 PTSD symptoms, although interviewers are encouraged to generate additional spontaneous prompts as needed to clarify responses. If clinically significant symptoms are present, the module concludes with questions regarding their onset, course, and severity. Following SCID conventions, symptoms are rated as ? = *Inadequate information*, 1 = *Absent*, 2 = *Subthreshold*, or 3 = *Threshold*, and symptom cluster criteria and the PTSD diagnosis are rated dichotomously as 1 = *Absent* or 3 = *Threshold*.

The advantages of the SCID PTSD module are that it is relatively brief, it follows the well-established, user-friendly structure of the SCID, and it appears to have adequate psychometric properties (see: **Structured Clinical Interview for DSM-IV—Posttraumatic Stress Disorder Module**). However, there are two main disadvantages. First, the trauma screening question is brief and likely insufficient for assessing lifetime trauma exposure. Second, it yields essentially dichotomous ratings for individual symptoms and for a PTSD diagnosis. It does not yield a continuous measure of PTSD symptom severity, which significantly limits its utility.

PTSD Symptom Scale-Interview

The PTSD Symptom Scale-Interview (PSS-I; Foa, Riggs, Dancu, & Rothbaum, 1993; see: **Posttraumatic Stress Disorder Symptom Scale**) consists of 17 questions corresponding to the symptom criteria for PTSD. The severity of each symptom over the past 2 weeks is

rated on a 4-point scale. In the original version the rating scale anchors were 0 = Not at all, 1 = A little bit, 2 = Somewhat, and 3 = Very much. In the current version the anchors include combined frequency and severity ratings, for example, 1 = Once per week or less/A little and 3 = 5 or more times per week/Very much, which allows the interviewer to apply whichever dimension, frequency or severity, is more appropriate for a given symptom (Foa & Tolin, 2000). The PSS-I thus yields a severity score for each of three PTSD symptom clusters as well as a total PTSD severity score. It also yields a dichotomous PTSD diagnosis, which is derived by means of a rationally derived scoring rule whereby items are counted as symptoms toward a diagnosis if they are rated at least as 1 = Once per week or less/A little or higher.

The advantages of the PSS-I are that it is relatively brief, it yields continuous severity scores for the three symptom clusters and the full syndrome as well as a dichotomous PTSD diagnosis, and it has excellent psychometric properties. One disadvantage is that the diagnostic scoring rule, which was rationally rather than empirically derived, may be too lenient in that it yields substantially higher PTSD prevalence rates than even the most lenient diagnostic rule for the Clinician-Administered PTSD Scale (Foa & Tolin, 2000).

Clinician-Administered PTSD Scale

The Clinician-Administered PTSD Scale (CAPS; Blake et al., 1990, 1995; see: **Clinician-Administered PTSD Scale**) is a 30-item structured interview for PTSD that assesses all of the *DSM-IV-TR* (American Psychiatric Association, 2000) criteria for PTSD, including trauma exposure, the 17 core symptoms, onset and duration, and degree of subjective distress and functional impairment. The CAPS also assesses five associated symptoms, including trauma-related guilt and dissociation, as well as overall response validity, symptom severity, and symptom improvement. The Life Events Checklist component of the CAPS is used to first screen for exposure to traumatic events, and a trauma inquiry section is administered

to evaluate both parts of Criterion A and identify an index event for symptom inquiry.

The CAPS differs from other PTSD interviews in several important ways. First, the CAPS assesses the frequency and intensity of each of the core and associated symptoms on separate 5-point (0 to 4) rating scales. Second, CAPS items include initial prompt questions as well as a number of standardized follow-up prompts. Third, CAPS prompts and rating scale anchors contain explicit behavioral referents to increase the reliability of the inquiry and ratings. Fourth, for the numbing and hyperarousal symptoms the CAPS requires interviewers to assess the link between symptoms and the index event and make an explicit “trauma-related” rating. Fifth, the CAPS provides explicit guidelines for assessing lifetime PTSD diagnostic status. Finally, a number of rationally and empirically derived scoring rules have been developed for generating a dichotomous PTSD diagnosis from CAPS frequency and intensity scores (Weathers, Ruscio, & Keane, 1999).

The advantages of the CAPS are that it provides a comprehensive evaluation of PTSD and associated features, it yields continuous measures of symptom severity as well as a dichotomous PTSD diagnosis, and it has excellent psychometric properties (Weathers, Keane, & Davidson, 2001). The CAPS has been studied extensively and has become the most widely PTSD interview. The main disadvantages of the CAPS are that relative to other PTSD interviews it typically takes longer to administer and requires more extensive training.

Self-Report Measures

DSM-Correspondent Measures

PTSD Checklist The PTSD Checklist (PCL; Weathers et al., 1993; see: **Posttraumatic Stress Disorder Checklist**) is a 17-item self-report measure of PTSD. The PCL is directly *DSM-IV*-correspondent (American Psychiatric Association, 1994) in that the 17 items reflect the 17 *DSM-IV* (American Psychiatric Association, 1994) symptoms of PTSD. PCL

ratings are based on the response dimension of subjective distress. Respondents indicate how much they were bothered by each symptom over the past month using a five-point scale ranging from 1 = Not at all to 5 = Extremely. There are three versions of the PCL, which differ only in the description of the index event in the first eight items (i.e., the five reexperiencing symptoms, two effortful avoidance symptoms, and amnesia). The civilian version (PCL-C), which refers to “a stressful experience from the past,” and the military version (PCL-M), which refers to “a stressful military experience,” are appropriate when a specific stressor has not been identified. In contrast, the specific version (PCL-S) is appropriate when a specific stressor has been identified. On the PCL-S respondents are instructed to write in a brief label for their target stressor and indicate the date it occurred, then answer all items with reference to this stressor.

The PCL yields a continuous measure of PTSD symptom severity for each of the three PTSD symptoms clusters and for the whole syndrome. It also yields a dichotomous PTSD diagnosis, which is obtained by considering items rated 3 = Moderately or higher as a symptom and following the *DSM-IV-TR* (American Psychiatric Association, 2000) diagnostic rule. The PCL is one of the most widely used self-report measures of PTSD. It has been evaluated extensively and has excellent psychometric properties across a wide variety of trauma populations. One disadvantage is that the PCL only measures the 17 symptoms of PTSD and does not measure trauma exposure, course and duration, or functional impairment.

Posttraumatic Stress Diagnostic Scale The Posttraumatic Stress Diagnostic Scale (PDS; Foa, 1995; Foa, Cashman, Jaycox, & Perry, 1997) is a 49-item self-report measure of PTSD that assesses all of the *DSM-IV-TR* (American Psychiatric Association, 2000) criteria for PTSD. The PDS consists of four sections. The first two sections assess PTSD’s Criterion A and identify an index event for symptom inquiry, the third section assesses the frequency of the 17 PTSD symptoms over the past month, and

the last section assesses functional impairment associated with the symptoms. Respondents rate symptoms on a 4-point frequency scale, with 0 = Not at all or only one time, 1 = Once a week or less/once in a while, 2 = 2 to 4 times a week/half the time, and 3 = 5 or more times a week/almost always. The PDS yields a continuous measure of symptom severity for each of the three symptom clusters, as well as a total severity score which ranges from 0 to 51 and is classified into one of four severity categories: mild (10 or lower), moderate (11 to 20), moderate to severe (21 to 35), and severe (36 or higher). The PDS also yields a dichotomous PTSD diagnosis, which is obtained by considering items rated at least 1 = Once a week or less/once in a while as symptoms and following the *DSM-IV-TR* (American Psychiatric Association, 2000) diagnostic rules.

The advantages of the PDS are that it assesses all the PTSD diagnostic criteria, it yields both a continuous measure of symptom severity and a PTSD diagnosis, and it has good psychometric properties. The PDS has been widely adopted in a variety of settings and has been translated into a number of languages for use in cross-cultural trauma research. One disadvantage is that the PTSD diagnosis it yields is based on a single, rationally derived scoring rule, and other rules have not been proposed or empirically evaluated.

Detailed Assessment of Posttraumatic Stress

The Detailed Assessment of Posttraumatic Stress (DAPS; Briere, 2001) is a 104-item, comprehensive self-report measure of trauma and PTSD. Like the PDS, the DAPS assesses all *DSM-IV-TR* (American Psychiatric Association, 2000) criteria for PTSD, including trauma exposure, the 17 PTSD symptoms, and the degree of functional impairment. However, the DAPS also includes scales assessing peri-traumatic distress and dissociation, trauma-specific dissociation, substance abuse, and suicidality. In addition, the DAPS is one of the only dedicated PTSD measures to include scales assessing response bias. DAPS scales are reported as T scores derived from a normative sample of approximately 400

trauma-exposed adults. On the clinical scales, T score elevations of 65 and above are considered clinically significant. In addition, the DAPS provides decision rules that yield a probable diagnosis for PTSD and acute stress disorder.

The DAPS is a very promising newer measure of PTSD. It has a number of advantages including the assessment of response validity, the assessment of all PTSD diagnostic criteria, the assessment of peri-traumatic responses and associated features of PTSD, and the use of norms to generate T scores. A potential disadvantage is that it is longer than other self-report PTSD measures. Also, it is a relatively new instrument and to date little psychometric work beyond that presented in the manual has appeared in the literature.

PTSD-Focused Measures

Impact of Event Scale The Impact of Event Scale (IES; Horowitz, Wilner, & Alvarez, 1979) is one of the most widely used self-report measures in the field of traumatic stress and is routinely included in clinical and research assessment batteries. Based on Horowitz's biphasic model of stress response, the IES consists of 15 items, 7 of which assess intrusive symptoms and 8 of which assess avoidance. Respondents rate the frequency of each symptom over the past week on a 4-point scale ranging from 0 = Not at all, 1 = Rarely, 3 = Sometimes, and 5 = Often. The IES has been extensively evaluated and has excellent psychometric properties (Sundin & Horowitz, 2002).

However, because the IES does not assess hyperarousal symptoms, it does not provide complete coverage of the PTSD symptom criteria. To address this limitation and to refashion the rating scale to bring it more in line with a typical Likert format, Weiss and Marmar (1997) developed a 22-item revised version of the IES (IES-R; see: **Impact of Event Scale—Revised**). They added six hyperarousal items and one dissociative item and substantially modified the rating scale, changing the response dimension from frequency to subjective distress, expanding it to a five-point

scale, and relabeling the anchors such that 0 = Not at all, 1 = A little bit, 2 = Moderately, 3 = Quite a bit, and 4 = Extremely. Even with these changes, though, the IES-R is still not completely *DSM-IV* correspondent (American Psychiatric Association, 1994). Some *DSM-IV* (American Psychiatric Association, 1994) PTSD symptoms are not assessed at all (diminished interest, estrangement, foreshortened future), and others are assessed somewhat ambiguously (amnesia, restricted range of affect). Nonetheless, the IES-R is a valuable measure and its use has grown steadily since its introduction. Also, as Sundin and Horowitz (2002) have noted, the introduction of IES-R did not render the IES obsolete, and both measures continue to be used effectively.

Mississippi Scale for Combat-Related PTSD

The Mississippi Scale for Combat-Related PTSD (Mississippi Scale; Keane, Caddell, & Taylor, 1988; see: **Mississippi Combat PTSD Scale**) is a 35-item self-report measure of PTSD symptoms and associated features. Items are rated on a 5-point scale with anchors that vary according to item content (e.g., 1 = Never to 5 = Very Frequently; 1 = Never True to 5 = Always True). The Mississippi Scale is the most widely used measure of combat-related PTSD. It has been extensively investigated and has excellent psychometric properties. Although it is not directly *DSM-IV* correspondent (American Psychiatric Association, 1994) it assesses many core and associated symptoms of PTSD and is a valuable component of a multimethod approach to PTSD assessment.

Because of the success of the Mississippi Scale, a civilian version (CMS) was developed (see: **Mississippi Civilian Scale for PTSD—Revised**). The most important modifications involved revising items that referred to the military, and adding four items to provide better coverage of the PTSD diagnostic criteria. Despite additional modifications involving item content and rating scale format (e.g., Inkelas, Loux, Bourque, Widawski, & Nguyen, 2000; Norris & Perilla, 1996) the CMS has not performed as well as the original combat version.

Multiscale Personality Inventories

Multiscale inventories such as the Minnesota Multiphasic Personality Inventory—2nd edition (MMPI-2; Butcher et al., 2001; see: **Minnesota Multiphasic Personality Inventory-2**) and the Personality Assessment Inventory (PAI; Morey, 1991) are valuable additions to a PTSD assessment battery. They complement dedicated PTSD interviews and self-report measures by providing crucial information regarding response validity, comorbidity, clinical management issues, and general personality factors, all of which can greatly facilitate differential diagnosis, case conceptualization, and treatment planning. These instruments also include specialized PTSD scales, which, although they are not *DSM-IV*-correspondent (American Psychiatric Association, 1994), provide useful converging evidence as part of a multimethod approach to assessing PTSD.

Minnesota Multiphasic Personality Inventory

The Minnesota Multiphasic Personality Inventory MMPI/MMPI-2 (Butcher et al., 2001; see: **Minnesota Multiphasic Personality Inventory-2**) is one of the oldest and most popular measures of personality and psychopathology and has been used extensively in the assessment of PTSD (Penk, Rierdan, Losardo, & Robinowitz, 2006). The original investigations of the MMPI in male combat veterans identified a mean *F-2-8* PTSD profile and led to the development of a specialized PTSD scale, the Keane PTSD scale (*PK* scale; Fairbank, Keane, & Malloy, 1983; Keane, Malloy, & Fairbank, 1984). Mean elevations on *F*, 2, and 8 have generally been replicated in other PTSD samples, although other scales often are elevated and there appears to be considerable heterogeneity in mean profile and prevalence of individual profiles across studies (e.g., Glenn, Beckham, & Sampson, 2002; Wise, 1996).

Similarly, the utility of the *PK* scale for discriminating individuals with and without PTSD has generally been replicated, although performance has varied across studies and optimal cutoff scores have generally been lower

than originally reported (e.g., Cannon, Bell, Andrews, & Finkelstein, 1987; Watson, Kucala, & Manifold, 1986). The PK scale has also been used to assess PTSD in civilian trauma samples (e.g., Koretzky & Peck, 1990), although there is some evidence that it may function more as a measure of general distress than as a measure of PTSD specifically (Scheibe, Bagby, Miller, & Dorian, 2001). The PK scale has also been evaluated as a stand-alone measure and appears to perform equally well in this format (Herman, Weathers, Litz, & Keane, 1996; Lyons & Scotti, 1994).

In addition to PTSD-specific information, the MMPI-2 provides information regarding two other vital domains, comorbidity and response bias. The MMPI-2 includes a number of empirically and conceptually derived scales that assess a wide variety of conditions commonly comorbid with PTSD, and thus contributes to a richly detailed assessment of the full clinical presentation in trauma survivors. In addition, the MMPI-2 includes an extensive set of response validity indicators. Given the concerns regarding malingering in the PTSD literature (e.g., Rosen, 2004), the MMPI-2 scales that detect a fake-bad response style, especially *F*, *Fp*, and *Ds* (Rogers, Sewell, Martin, & Vitaco, 2003), are particularly relevant. Beyond these general indicators of malingering, a new scale specific to detecting feigned PTSD, *Fptsd*, was recently developed and found to outperform existing MMPI-2 scales (Elhai, Ruggiero, Frueh, Beckham, & Gold, 2002). However, Marshall and Bagby (2006) recently questioned the incremental validity of *Fptsd*, so additional studies are needed to clarify the relative performance of these scales.

Personality Assessment Inventory Developed in 1991, the Personality Assessment Inventory (PAI; Morey, 1991) is a newer multiscale inventory designed to address some of the limitations of previous instruments. The PAI was developed in a construct validation framework that emphasized conceptual explication of the constructs to be assessed, the importance of content validity in generating items, and the use of multiple empirical parameters for evaluating items and retaining them for the

final scale. The PAI consists of 344 items, which are rated on a 4-point scale with anchors of False, Not At All True; Slightly True; Mainly True; and Very True. There are 22 primary scales, including four response validity scales, 11 clinical scales, five treatment scales, and two interpersonal scales. In addition, nine clinical scales and one treatment scale have subscales to assess specific components of the parent scale. The response validity scales detect random responding and overly positive and overly negative self-presentation. The clinical scales assess well-established clinical syndromes, the treatment scales assess several areas relevant to clinical management, and the interpersonal scales assess key aspects of normal personality.

Although relatively few studies have been conducted to date, the PAI appears to have considerable promise for the assessment of PTSD and represents a viable alternative to the MMPI-2. The PAI includes an 8-item specialized PTSD scale, the Traumatic Stress Scale of the Anxiety-Related Disorders Scale (*ARD-T*), which assesses reexperiencing, effortful avoidance, loss of interest in usual activities, and guilt. Although it does not provide full coverage of the *DSM-IV* (American Psychiatric Association, 1994) PTSD symptom criteria, *ARD-T* does assess many of the distinctive aspects of PTSD and typically is the most elevated PAI scale in PTSD profiles. In addition, recent studies have found that several other PAI scales differentiate those with and without PTSD, including scales measuring depression, anxiety, somatic complaints, and borderline features (McDevitt-Murphy, Weathers, Adkins, & Daniels, 2005; McDevitt-Murphy, Weathers, Flood, Eakin, & Benson, 2007). Further, in one recent study the PAI outperformed the MMPI-2 in discriminating PTSD from depressive disorders (McDevitt-Murphy et al., 2007).

Assessment of Trauma Exposure

In contrast to the remarkable progress that has been made in the development of psychometrically sound measures of PTSD, considerably less progress has been made in the development of measures of trauma exposure

(see Weathers & Keane, 2007 for a full discussion of issues regarding the definition and measurement of trauma). Numerous trauma exposure measures have been developed but few have been adequately investigated or widely adopted. This is due in large part to the fact that trauma is difficult to define, both in a broad conceptual sense with respect to delineating traumatic stressors from ordinary stressors, and in a more specific, practical sense of considering key aspects of trauma exposure such as event type (e.g., combat, sexual assault, natural disaster), dimensions of stressor severity and burden (e.g., life threat, physical harm, interpersonal loss, frequency, duration), and exposure level (e.g., directly experienced, witnessed, learned about).

Trauma measures vary considerably in scope and format in accordance with the assessment questions they are intended to address (see Norris & Hamblen, 2004 for a recent review of trauma exposure measures). Often the main purpose of the trauma assessment is to determine if an individual has experienced at least one event that would meet both parts of the definition of a trauma in Criterion A of the *DSM-IV-TR* (American Psychiatric Association, 2000) PTSD criteria. Some measures, such as the Criterion A component of the SCID PTSD module, consist of a few questions that screen for exposure to possible traumatic stressors, identify an index event for symptom inquiry, and determine if the event satisfies Criterion A. Others, such as the Criterion A section of the PDS and DAPS, provide a list of traumatic event types and direct respondents to check all that apply, then select the worst event and write a brief narrative. The Life Events Checklist (LEC), the trauma assessment component of the CAPS, combines a self-report screener with an interview-based inquiry. The screener consists of a list of 17 event categories with five response options to indicate exposure level, including “happened to me,” “witnessed it,” “learned about it,” “not sure if it happened,” and “did not happen.” The interview component identifies up to three events for symptom inquiry, then determines if the events meet both parts of Criterion A. The LEC may also be

used in conjunction with self-report measures of PTSD. In our own research, for example, we often administer an extended form of the LEC along with the PCL.

In addition to identifying an index event for PTSD symptom inquiry, measures have also been developed to quantify the severity of trauma exposure within a single domain of exposure and to quantify trauma exposure across the life span. An example of a domain-specific measure is the Combat Exposure Scale (CES; Keane et al., 1989), a widely used, 7-item self-report measure that quantifies the severity of exposure to war-zone stress. Examples of carefully constructed measures that are useful for evaluating trauma across the life span include the Traumatic Life Events Questionnaire (TLEQ; Kubany et al., 2000), Life Stressor Checklist—Revised (McHugo et al., 2005), and Evaluation of Lifetime Stressors (ELS; Krinsley, Gallagher, Weathers, Kutter, & Kaloupek, 2003).

Conclusion

Considerable progress has been in the development and evaluation of standardized measures of trauma exposure PTSD, and such measures are essential for the evidence-based assessment of PTSD. There are a number of issues that need to be addressed when selecting measures for a given clinical or research application. First, the goals for the assessment must be clearly established. Self-report measures are appropriate for screening and quantifying symptom severity, but structured interview are best for establishing a PTSD diagnosis and should be administered whenever possible. Second, the overall size and scope of the assessment battery must be determined. A comprehensive assessment would include a thorough evaluation of lifetime trauma exposure, a structured interview, one or more self-report measures, and a multiscale inventory. Such a battery would provide information regarding PTSD diagnostic status, PTSD symptom severity, comorbidity and differential diagnosis, clinical management concerns, and response validity. When resources are limited a smaller battery could suffice for some applications. However, adding additional

measures would in many cases unnecessarily increase respondent burden and raise the issue of incremental validity.

Third, the target population must be taken into account. Measures should be selected only if they have been shown to be valid for the intended purpose in the specific population in which they are to be used. When a relevant empirical literature does not yet exist, measures should be used and interpreted with caution. Last, given the controversy surrounding trauma and PTSD and the potential for malingering it is crucial to formally evaluate response bias whenever possible. This can be accomplished with the use of multiscale inventories such as the MMPI-2 or PAI, or through the use of specialized measures of malingering such as the Structured Interview of Reported Symptoms (SIRS; Rogers, Bagby, & Dickens, 1992).

REFERENCES

- American Psychiatric Association. (1994). *Diagnostic and statistical manual of mental disorders* (4th ed.). Washington, DC: Author.
- American Psychiatric Association. (2000). *Diagnostic and statistical manual of mental disorders* (4th ed., text rev.). Washington, DC: Author.
- Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Gusman, F. D., Charney, D. S., et al. (1995). The development of a clinician-administered PTSD scale. *Journal of Traumatic Stress*, 8, 75–90.
- Blake, D. D., Weathers, F. W., Nagy, L. M., Kaloupek, D. G., Klauminser, G., Charney, D. S., et al. (1990). A clinician rating scale for assessing current and lifetime PTSD: The CAPS-1. *Behavior Therapist*, 18, 187–188.
- Briere, J. (2001). *Detailed Assessment of Posttraumatic Stress (DAPS)*. Odessa, FL: Psychological Assessment Resources.
- Briere, J. (2004). *Psychological assessment of adult posttraumatic states: Phenomenology, diagnosis, and measurement* (2nd ed.). Washington, DC: American Psychological Association.
- Butcher, J. N., Graham, J. R., Ben-Porath, Y. S., Tellegen, A. M., Dahlstrom, W. G., & Kaemmer, B. (2001). *Minnesota Multiphasic Personality Inventory-2: Manual for administration, scoring, and interpretation* (Rev. ed.). Minneapolis: University of Minnesota Press.
- Cannon, D. S., Bell, W. E., Andrews, R. H., & Finkelstein, A. S. (1987). Correspondence between MMPI PTSD measures and clinical diagnosis. *Journal of Personality Assessment*, 51, 517–521.
- Elhai, J. D., Gray, M. J., Kashdan, T. B., & Franklin, C. L. (2005). Which instruments are most commonly used to assess traumatic event exposure and posttraumatic effects?: A survey of traumatic stress professionals. *Journal of Traumatic Stress*, 18, 541–545.
- Elhai, J. D., Ruggiero, K. J., Frueh, B. C., Beckham, J. C., & Gold, P. B. (2002). The Infrequency-Posttraumatic Stress Disorder Scale (Fptsd) for the MMPI-2: Development and initial validation with veterans presenting with combat-related PTSD. *Journal of Personality Assessment*, 79, 531–549.
- Fairbank, J. A., Keane, T. M., & Malloy, P. F. (1983). Some preliminary data on the psychological characteristics of Vietnam veterans with post-traumatic stress disorders. *Journal of Consulting and Clinical Psychology*, 51, 912–919.
- First, M. B., Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structured Clinical Interview for DSM-IV Axis I Disorders, Clinician Version (SCID-CV)*. Washington, DC: American Psychiatric Press.
- Foa, E. B. (1995). *Posttraumatic Stress Diagnostic Scale* [Manual]. Minneapolis, MN: National Computer Systems.
- Foa, E. B., Cashman, L., Jaycox, L., & Perry, K. (1997). The validation of a self-report measure of posttraumatic stress disorder: The Posttraumatic Diagnostic Scale. *Psychological Assessment*, 9, 445–451.
- Foa, E. B., Riggs, D. S., Dancu, C. V., & Rothbaum, B. O. (1993). Reliability and validity of a brief instrument for assessing post-traumatic stress disorder. *Journal of Traumatic Stress*, 6, 459–473.
- Foa, E. B., & Tolin, D. F. (2000). Comparison of the PTSD Symptom Scale-Interview version and the Clinician-Administered PTSD Scale. *Journal of Traumatic Stress*, 13, 181–191.
- Glenn, D. M., Beckham, J. C., & Sampson, W. S. (2002). MMPI-2 profiles of Gulf and Vietnam combat veterans with chronic posttraumatic stress disorder. *Journal of Clinical Psychology*, 58, 371–381.
- Herman, D. S., Weathers, F. W., Litz, B. T., & Keane, T. M. (1996). Psychometric properties of the embedded and stand-alone versions of the MMPI-2 Keane PTSD Scale. *Assessment*, 3, 437–442.

- Horowitz, M. J., Wilner, N., & Alvarez, W. (1979). Impact of Event Scale: A measure of subjective stress. *Psychosomatic Medicine*, 41, 209–218.
- Inkelas, M., Loux, L. A., Bourque, L. B., Widawski, M., & Nguyen, L. H. (2000). Dimensionality and reliability of the Civilian Mississippi Scale for PTSD in a postearthquake community. *Journal of Traumatic Stress*, 13, 149–167.
- Keane, T. M., Caddell, J. M., & Taylor, K. L. (1988). Mississippi Scale for combat-related posttraumatic stress disorder: Three studies in reliability and validity. *Journal of Consulting and Clinical Psychology*, 56, 85–90.
- Keane, T. M., Fairbank, J. A., Caddell, J. M., Zimering, R. T., Taylor, K. L., & Mora, C. A. (1989). Clinical evaluation of a measure to assess combat exposure. *Psychological Assessment*, 1, 53–55.
- Keane, T. M., Malloy, P. F., & Fairbank, J. A. (1984). Empirical development of an MMPI subscale for the assessment of combat-related posttraumatic stress disorder. *Journal of Consulting and Clinical Psychology*, 52, 888–891.
- Koretzky, M. B., & Peck, A. H. (1990). Validation and cross-validation of the PTSD Subscale of the MMPI with civilian trauma victims. *Journal of Clinical Psychology*, 46, 296–300.
- Krinsley, K. E., Gallagher, J. G., Weathers, F. W., Kutter, C. J., & Kaloupek, D. G. (2003). Consistency of retrospective reporting about exposure to traumatic events. *Journal of Traumatic Stress*, 16, 399–409.
- Kubany, E. S., Haynes, S. N., Leisen, M. B., Owens, J. A., Kaplan, A. S., Watson, S. B., et al. (2000). Development and preliminary validation of a brief broad-spectrum measure of trauma exposure: The Traumatic Life Events Questionnaire. *Psychological Assessment*, 12, 210–224.
- Lyons, J. A., & Scotti, J. R. (1994). Comparability of two administration formats of the Keane Posttraumatic Stress Disorder Scale. *Psychological Assessment*, 6, 209–211.
- Marshall, M. B., & Bagby, R. M. (2006). The incremental validity and clinical utility of the MMPI-2 Infrequency Posttraumatic Stress Disorder Scale. *Assessment*, 13, 417–429.
- McDevitt-Murphy, M. E., Weathers, F. W., Adkins, J. W., & Daniels, J. B. (2005). Use of the Personality Assessment Inventory in assessment of posttraumatic stress disorder in women. *Journal of Psychopathology and Behavioral Assessment*, 27, 57–65.
- McDevitt-Murphy, M. E., Weathers, F. W., Flood, A. M., Benson, T., & Eakin, D. E. (2007). A comparison of the MMPI-2 and PAI for discriminating PTSD from depression and social phobia. *Assessment*, 14, 181–195.
- McHugo, G. J., Caspi, Y. Y., Kammerer, N., Mazelis, R., Jackson, E. W., Russell, L., et al. (2005). The assessment of trauma history in women with co-occurring substance abuse and mental disorders and a history of interpersonal violence. *Journal of Behavioral Health Services and Research*, 32, 113–127.
- Morey, L. C. (1991). *Personality Assessment Inventory: Professional manual*. Lutz, FL: Psychological Assessment Resources.
- Norris, F. H., & Hamblen, J. L. (2004). Standardized self-report measures of civilian trauma and PTSD. In J. P. Wilson & T. M. Keane (Eds.), *Assessing psychological trauma and PTSD* (2nd ed., pp. 63–102). New York: Guilford Press.
- Norris, F. H., & Perilla, J. L. (1996). The revised Civilian Mississippi Scale for PTSD: Reliability, validity, and cross-language stability. *Journal of Traumatic Stress*, 9, 285–298.
- Penk, W. E., Rierdan, J., Losardo, M., & Robinowitz, R. (2006). The MMPI-2 and assessment of posttraumatic stress disorder (PTSD). In J. N. Butcher (Ed.), *MMPI-2: A practitioner's guide* (pp. 121–139). Washington, DC: American Psychological Association.
- Rogers, R., Bagby, R. M., & Dickens, S. E. (1992). *Structured Interview of Reported Symptoms (SIRS) and professional manual*. Odessa, FL: Psychological Assessment Resources.
- Rogers, R., Sewell, K. W., Martin, M. A., & Vitacco, M. J. (2003). Detection of feigned mental disorders: A meta-analysis of the MMPI-2 and malingering. *Assessment*, 10, 160–177.
- Rosen, G. M. (2004). Malingering and the PTSD data base. In G. M. Rosen (Ed.), *Posttraumatic stress disorder: Issues and controversies* (pp. 85–99). Hoboken, NJ: Wiley.
- Scheibe, S., Bagby, R. M., Miller, L. S., & Dorian, B. J. (2001). Assessing posttraumatic stress disorder with the MMPI-2 in a sample of workplace accident victims. *Psychological Assessment*, 13, 369–374.
- Sundin, E. C., & Horowitz, M. J. (2002). Impact of Event Scale: Psychometric properties. *British Journal of Psychiatry*, 180, 205–209.
- Watson, C. G., Kucala, T., & Manifold, V. (1986). A cross-validation of the Keane and Penk MMPI

Scales as measures of post-traumatic stress disorder. *Journal of Clinical Psychology*, 42, 727–732.

Weathers, F. W., & Keane, T. M. (2007). The Criterion A problem revisited: Controversies and challenges in defining and measuring psychological trauma. *Journal of Traumatic Stress*, 20, 107–121.

Weathers, F. W., Keane, T. M., & Davidson, J. R. (2001). Clinician-Administered PTSD Scale: A review of the first ten years of research. *Depression and Anxiety*, 13, 132–156.

Weathers, F. W., Litz, B. T., Herman, D. S., Huska, J. A., & Keane, T. M. (1993, October). *The PTSD Checklist (PCL): Reliability, validity, and diagnostic utility*. Paper presented at the annual meeting of the International Society for Traumatic Stress Studies, San Antonio, TX.

Weathers, F. W., Ruscio, A. M., & Keane, T. M. (1999). Psychometric properties of nine scoring rules for the Clinician-Administered Posttraumatic Stress Disorder Scale. *Psychological Assessment*, 11, 124–133.

Weiss, D. S., & Marmar, C. R. (1997). The Impact of Event Scale—Revised. In J. P. Wilson & T. M. Keane (Eds.), *Assessing psychological trauma and PTSD* (pp. 399–411). New York: Guilford Press.

Wilson, J. P., & Keane, T. M. (Eds.). (2004). *Assessing psychological trauma and PTSD* (2nd ed.). New York: Guilford Press.

Wise, E. A. (1996). Diagnosing posttraumatic stress disorder with the MMPI clinical scales: A review of the literature. *Journal of Psychopathology and Behavioral Assessment*, 18, 71–82.

FRANK W. WEATHERS
Auburn University

TERENCE M. KEANE
National Center for Posttraumatic Stress Disorder

See also: Clinician-Administered PTSD Scale; Diagnosis of Traumatic Stress Disorders (*DSM* & *ICD*); Impact of Event Scale—Revised; Minnesota Multiphasic Personality Inventory-2; Mississippi Civilian Scale for PTSD—Revised; Mississippi Combat PTSD Scale; Posttraumatic Stress Disorder Checklist; Posttraumatic Stress Disorder, Diagnosis of; Posttraumatic Stress Disorder Symptom Scale; Structured Clinical Interview for *DSM-IV*—Posttraumatic Stress Disorder Module; Trauma, Definition

ASSESSMENT, PSYCHOMETRIC, CHILD

A thorough clinical assessment is crucial to effective treatment interventions for traumatized children and adolescents. Comprehensive evaluation of psychological trauma and its effects typically includes information from a number of sources, including the child's or adolescent's self-report, caretaker reports of the child's functioning, and collateral reports from other providers. The primary targets of assessment are the child's trauma exposure history and current emotional symptoms. However, information may also be collected on caretaker and family functioning, the child's developmental history, primary attachment relationships, child protective services involvement and placement history, current school functioning, history of losses, medical status, coping skills, and environmental stressors such as community violence. Once consent for release of information is provided, the clinician can gather more complete background information from agencies interacting with the child and family, such as child protective services, law enforcement, and other mental health agencies.

Evaluation of Trauma Exposure History

The most common types of traumatic events evaluated are child abuse (physical, sexual, and psychological), emotional neglect, assaults by peers (both physical and sexual), community violence, witnessing violence done to others, traumatic loss, exposure to accidents (e.g., motor vehicle accidents) and disasters, and serious medical illness or injury. Assessment typically involves determining not only the nature of these various traumas, but also their frequency, type, and age of onset.

Unfortunately, the child or adolescent may not report all significant instances of trauma exposure during the initial assessment session or early in treatment. Instead, important historical events may be disclosed later in treatment, as the child engages more fully with the therapist and experiences a greater sense of trust and safety. The manner in which children

and adolescents, as well as caretakers, are questioned regarding trauma exposure will also determine the extent to which a more complete account is provided (Lanktree, Briere, & Zaidi, 1991). Clinical sensitivity is often required regarding particularly difficult, embarrassing, traumatic, or shame-inducing experiences. If a child or caretaker has fears regarding the repercussions of the disclosure (e.g., removal of the child from the home), this may also further limit the accuracy of abuse or violence-related disclosures. Finally, the child's or adolescent's tendency to avoid thinking about and disclosing distressing material may decrease his or her participation in the assessment, thereby leading to underreporting of trauma exposure (Elliott & Briere, 1994).

The context in which the assessment is conducted also can affect the extent of trauma-related information that is disclosed by the child and/or family, whether by interview or on psychological tests. For example, in school settings, the child may not feel as free to divulge information due to concerns about confidentiality, including fear that his or her trauma history or symptoms will be shared with school personnel or other students. In hospital settings, where a child may be assessed for psychological trauma following a serious medical illness or condition (e.g., HIV infection, cancer, surgeries) or traumatic injury (e.g., an automobile accident), the child's and family's need to cope with urgent or chronic medical issues may lead them to overlook or suppress information regarding prior (or current) abuse or violence.

In forensic settings, issues of blame, punishment, and authority may cause the child to fear (a) retribution from those he or she implicates in a crime by virtue of disclosing the trauma, or (b) maltreatment, loss, or family disruption as a result of criminal justice and child welfare system involvement in his or her life—each of which may motivate underreporting of traumatic experiences. On the other hand, if a child has been able to disclose abuse or violence-related exposures in a forensic interview, he or she may be able to more easily express symptoms and discuss traumatic exposure during the psychological assessment, especially if all

services are delivered at the same center. This assessment should be conducted separately in a clinical environment that is not part of the forensic investigation, with ongoing consideration of the current safety of the child or adolescent (e.g., possible ongoing exposure to child abuse, domestic violence, or risk related to gang-related community violence).

Because the words used to label traumatic events may confuse or intimidate children (e.g., asking about "rape" or "abuse"), evaluation of trauma exposure is often more effective when behavioral descriptions of these events are employed. This is often best accomplished by using a structured measure or interview that assesses exposure to the major types of traumatic events in a standardized and behaviorally specific way. Trauma exposure measures on which either the child or a caretaker can report about traumatic experiences include the KID-SAVE (Flowers, Hastings, & Kelley, 2000), Traumatic Events Screening Inventory (TESI; Ford et al., 2000), and Violence Exposure Scale for Children—Revised (VEX-R; Fox & Leavitt, 1995). These and similar measures facilitate a relatively complete review of the child's lifetime trauma exposure, and typically have been evaluated psychometrically to determine their effectiveness and validity.

Evaluation of Trauma-Relevant Symptoms

An optimal assessment of children's trauma-related symptomatology includes evaluation of immediate safety issues, such as suicidality, substance abuse, and involvement in high-risk behaviors, as well as a preliminary estimation of current emotional functioning and potential targets for treatment. The results of such assessment, in turn, will determine whether an immediate clinical response is indicated (e.g., crisis intervention, hospitalization, harm reduction activities), as well as which specific treatment modalities (e.g., play therapy, cognitive interventions, therapeutic exposure, family therapy) might be most helpful. Further, when the same tests are administered on multiple occasions (e.g., every 2 or 3 months), the ongoing effects of clinical intervention can be ascertained,

allowing the clinician to make mid-course corrections in strategy or focus when specific symptoms are seen to decrease or exacerbate.

For some children and adolescents, multiple trauma exposures such as abuse, neglect, family and community violence, relational losses, and injuries or illnesses may occur concomitantly, resulting in a more complex clinical picture. For example, a child with early, sustained, and multiple traumatic experiences may evidence significant disturbances in emotional, behavioral, developmental, cognitive, and relational domains, as well as presenting with significant posttraumatic stress (Briere & Spinazzola, 2005; Cook et al., 2005). In addition, gender-related, developmental, and cultural factors may affect how any given symptom manifests. For this reason, it is usually preferable to administer multiple tests tapping a variety of different symptoms, rather than a single measure, and to take mediating demographic, social, and cultural issues into account.

As is true in other areas of assessment, standardized assessment measures of trauma-related symptomatology are almost always preferable to those without norms or validation studies. Such measures may be either generic (i.e., tapping symptoms that occur in both traumatized and nontraumatized children, such as anxiety, depression, or aggression) or trauma-specific (i.e., evaluating symptoms that are more commonly associated with trauma exposure, such as posttraumatic stress, dissociation, or reactive sexual behavior).

These tests commonly involve either caretaker reports of the child's symptoms and behaviors or child self-reports of their own distress and/or behavioral disturbance. The choice of whether to use child or caretaker reports of child symptoms can be difficult, since each approach has its own potential benefits and weaknesses.

Child self-report measures allow the child to directly disclose his or her internal experience or problems, as opposed to the clinician relying on "second-hand" reports of a parent or caretaker. However, the child's report may be affected by his or her fears of disclosure, denial of emotional distress, or—especially in younger children—inability to report on complex internal

states (Friedrich, 2002). Caretaker report of the child's symptomatology has the potential benefit of providing a more objective report of the child's symptoms and behaviors, yet may be compromised by parental denial, guilt, or preoccupation with the child's trauma, as well as parental/caretaker difficulties in accurately assessing the child's internal experience, especially if the child avoids describing those experiences to the caretaker (Lanktree et al., in press; Reid, Kavanaugh, & Baldwin, 1987). For these reasons, it is recommended that the assessment of traumatized children use *both* child- and caretaker-report measures, so that the advantages of each methodology can be maximized, and the child's actual clinical status can be triangulated by virtue of multiple sources of information (Lanktree et al., in press; Nader, 2004).

Just as effective assessment often involves both caretaker and child reports, it is also important to use both generic and trauma-specific tests when evaluating traumatized children. Reliance on solely generic tests (e.g., of depression or behavior problems) may result in inadequate information on more trauma-specific responses (e.g., posttraumatic stress or dissociation). In contrast, solely administering trauma-specific measures can easily lead to an underestimation of important, yet less trauma-specific, clinical conditions (e.g., severe depression in a sexually abused child or adolescent).

Perhaps the most commonly used generic measure in the assessment of traumatized children and youth is the Child Behavior Checklist (CBCL; Achenbach, 1991), which has separate Parent Report, Teacher Report, and Youth Self-Report versions. This test evaluates the extent of internalizing (e.g., anxiety or depression) and externalizing (e.g., behavior problems) symptomatology, as well as measuring some resilience or adaptive functions. General emotional functioning can also be evaluated using measures such as the Child Depression Inventory (CDI; Kovacs, 1992).

Trauma-specific measures, completed by a child or adolescent, include the UCLA PTSD Index for *DSM-IV* (UPID; Pynoos, Rodriguez, Steinberg, Stuber, & Frederick, 1998; *see: UCLA PTSD Reaction Index*), Child Dissociative

Checklist (CDC; Putnam, Helmers, & Trickett, 1993), and the Trauma Symptom Checklist for Children (TSCC; Briere, 1996; see: **Trauma Symptom Checklist for Children**). Trauma-specific measures completed by the primary caretaker on behalf of the child include the Child Sexual Behavior Inventory (CSBI; Friedrich, 1998) and Trauma Symptom Checklist for Young Children (TSCYC; Briere, 2005).

A carefully selected psychological test battery can help determine the extent of the child's trauma-related symptomatology, as well as any other emotional difficulties (e.g., depression) that also may be present. This insight into the child's internal emotional experience and behavioral responses, in turn, can help the clinician devise an effective treatment regimen that is relevant to the child's specific clinical presentation and needs.

When assessment is repeated over time, psychometric evaluation can also signal the need to change or augment the treatment focus as necessary. For example, ongoing assessment may suggest a shift in therapeutic focus when post-traumatic stress symptoms begin to respond to treatment but other symptoms continue relatively unabated (e.g., Briere, 2001; Lanktree & Briere, 1995). Finally, repeated administration of measures can increase accountability and quality control, and can add to the clinical knowledge-base regarding the effectiveness of various trauma-related psychotherapies. When administered at the end of psychotherapy (and, potentially, several months later, at follow-up), such pre-post data can help the clinician or agency determine the effectiveness of a given treatment for a given child or group of children. Generally, we suggest a test-retest interval of at least 2 or 3 months, in order to avoid possible biasing or sensitizing effects of repeated testing over shorter periods of time.

Conclusion

Psychological assessment is an important component of effective treatment for traumatized children. An initial assessment should ideally include both child self-reports and caretaker reports on the child, and should address history

of trauma exposure and subsequent trauma-related emotional symptoms. At the same time, less trauma-related (i.e., more generic) symptoms also should be evaluated, since they, too, may require clinical intervention. Because of the breadth of symptoms potentially associated with childhood trauma exposure, effective psychometric assessment often will include a number of different tests that tap a range of potential symptoms and problems. Repeated assessment is recommended for those children undergoing trauma-focused therapy, so that changes in symptomatology over time can be detected and the focus of therapy can be adjusted accordingly. Repeat assessment also increases accountability and quality control, since the effectiveness of treatment can be tracked within and across child clients.

REFERENCES

- Achenbach, T. M. (1991). *Manual for the Child Behavior Checklist/4-18 and 1991 Profile*. Burlington: University of Vermont, Department of Psychiatry.
- Briere, J. (1996). *Trauma Symptom Checklist for Children (TSCC)*. Odessa, FL: Psychological Assessment Resources.
- Briere, J. (2001). Evaluating treatment outcome. In M. Winterstein & S. R. Scribner (Eds.), *Mental healthcare for child crime victims: Standards of care task force guidelines*. Sacramento: California Victims Compensation and Government Claims Board, Victims of Crime Program, State of California.
- Briere, J. (2005). *Trauma Symptom Checklist for Young Children (TSCYC)*. Odessa, FL: Psychological Assessment Resources.
- Briere, J., & Spinazzola, J. (2005). Phenomenology and psychological assessment of complex post-traumatic states. *Journal of Traumatic Stress, 18*, 401-412.
- Cook, A., Spinazzola, J., Ford, J., Lanktree, C., Blaustein, M., Cloitre, M., et al. (2005). Complex trauma in children and adolescents. *Psychiatric Annals, 35*, 390-398.
- Elliott, D. M., & Briere, J. (1994). Forensic sexual abuse evaluations of older children: Disclosures and symptomatology. *Behavioral Sciences and the Law, 12*, 261-277.

- Flowers, A. L., Hastings, T. L., & Kelley, M. L. (2000). Development of a screening instrument for exposure to violence in children: The KID-SAVE. *Journal of Psychopathology and Behavioral Assessment*, 22(1), 91–104.
- Ford, J. D., Racusin, R., Ellis, C., Daviss, W. B., Reiser, J., Fleischer, A., et al. (2000). Child maltreatment, other trauma exposure, and post-traumatic symptomatology among children with oppositional defiant and attention deficit hyperactivity disorders. *Child Maltreatment*, 5, 205–217.
- Fox, N. A., & Leavitt, L. A. (1995). *The Violence Exposure Scale for Children-VEX-R*. College Park: University of Maryland, Department of Human Development.
- Friedrich, W. N. (1998). *The Child Sexual Behavior Inventory professional manual*. Odessa, FL: Psychological Assessment Resources.
- Friedrich, W. N. (2002). *Psychological assessment of sexually abused children and their families*. Thousand Oaks, CA: Sage.
- Kovacs, M. (1992). *Children's Depression Inventory*. New York: Multi-Health Systems.
- Lanktree, C. B., & Briere, J. (1995). Outcome of therapy for sexually abused children: A repeated measures study. *Child Abuse and Neglect*, 19, 1145–1155.
- Lanktree, C. B., Briere, J., & Zaidi, L. Y. (1991). Incidence and impacts of sexual abuse in a child outpatient sample: The role of direct inquiry. *Child Abuse and Neglect*, 15, 447–453.
- Lanktree, C. B., Gilbert, A. M., Briere, J., Taylor, N., Chen, K., Maida, C. A., et al. (in press). Multi-informant assessment of maltreated children: Convergent and discriminant validity of the TSCC and TSCYC. *Child Abuse and Neglect*.
- Nader, K. O. (2004). Assessing traumatic experiences in children and adolescents: Self-reports of DSM PTSD criteria B-D symptoms. In J. P. Wilson & T. M. Keane (Eds.), *Assessing psychological trauma and PTSD* (2nd ed., pp. 513–537). New York: Guilford Press.
- Putnam, F. W., Helmers, K., & Trickett, P. K. (1993). Development, reliability, and validity of a child dissociation scale. *Child Abuse and Neglect*, 17, 731–741.
- Pynoos, R., Rodriguez, N., Steinberg, A., Stuber, M., & Frederick, C. (1998). *The UCLA PTSD Index for DSM-IV*. Los Angeles: UCLA Trauma Psychiatry Program.
- Reid, J. B., Kavanagh, K. A., & Baldwin, D. V. (1987). Abusive parents' perceptions of child problem

behaviors: An example of parental bias. *Journal of Abnormal Child Psychology*, 15, 457–466.

CHERYL LANKTREE
Miller Children's Hospital

JOHN BRIERE
University of Southern California

See also: Child Development; Diagnosis of Traumatic Stress Disorders (DSM & ICD); Posttraumatic Stress Disorder, Diagnosis of; Trauma, Definition; Trauma Symptom Checklist for Children; UCLA PTSD Reaction Index

ASSESSMENT, PSYCHOPHYSIOLOGICAL

Psychophysiological assessment (PA) is a generic term often substituted for psychological challenge testing or (emotion) provocation testing. Psychophysiological procedures involve monitoring biological systems that are reactive to psychologically meaningful events, in contrast to physiological measurement that is concerned with biological state per se. It is common for PA to include nonphysiological measures as well.

Measurement for PA occurs during controlled presentation of stimulus material that has known impact on psychological state. One format of PA examines differences between measures recorded during a target state (e.g., fear) and the same measures recorded during a reference state (e.g., engagement in a nonfearful task). This approach assumes that differences reflect the degree to which the state of interest has been evoked, and the magnitude of differences can be used as one source of evidence regarding the severity of a condition or disorder. Another format of PA examines differences in reactions to the same stimulus material on two or more occasions in order to evaluate change. This approach can be used to demonstrate the effect of treatment (e.g., reduction in fear after therapy)

Rationale

Trauma-related PA is conceptually tied to the fight or flight response. It involves presentation of an evocative cue—a trauma-related reminder—that is expected to trigger a negative emotional

state. The ability of the cue to evoke emotion is commonly attributed to classical conditioning, a learning process believed to occur during the traumatic experience. The negative emotion is expected to involve reactions in at least three domains: (1) biological mobilization, (2) subjective distress, and (3) behavioral withdrawal.

The biological component most often involves measures of bodily systems that are activated by the sympathetic nervous system in preparation for self-protective behavior. For example, increases in heart rate and blood pressure provide more blood as muscles are readied to power escape, and increases in sweating on the palms (the basis for skin conductance measurement) may reduce the risk of skin abrasion and blood loss during attack or self-defense.

It is common to measure the subjective component of PA on a simple rating scale with endpoints indicating high and low distress. Subjective ratings are sometimes obtained on two dimensions thought to be primary constituents of global distress: valence (positive-negative) and arousal (quiescent-activated).

While PA procedures generally limit opportunity for measuring overt escape behavior (e.g., due to electrode attachments), a person may still disengage from the task by behavior such as turning away from a video monitor displaying the trauma reminder. Self-report of behavioral intentions (e.g., the strength of desire to escape from the situation) is another potential index. Measures in this domain have received less attention in the published PA literature than have physiological and subjective measures.

History and Evidence

Trauma-related PA can be traced to two lines of research in the mid-twentieth century. One began in the 1940s and examined physiological reactions of combat-exposed military veterans when they were presented reminders of war. Individuals conducting this work included Abraham Kardiner and M. A. Wenger. The second line began in the 1960s and examined psychophysiological reactions of phobic individuals when presented their self-identified feared object (e.g., a snake). Peter Lang provided both theoretical impetus and methodological innovation

for these efforts (e.g., Lang, Melamed, & Hart, 1970).

Contemporary PA with traumatized populations began in the early 1980s after the PTSD diagnosis was adopted. Studies variously used auditory, audiovisual, and imaginal formats to present reminders of the war zone to groups of combat-exposed military veterans, comparing reactions between those who did and did not meet criteria for PTSD. Typically, these studies demonstrated greater physiological reactivity and distress for the individuals with PTSD.

Since then, numerous traumatized populations have been tested using PA methods and have replicated the essential findings. Examples include individuals who experienced motor vehicle accidents, sexual assault, childhood sexual abuse, and occupational trauma (e.g., police; firefighters), as well as additional military veterans. Other studies using PA with various PTSD populations also have demonstrated that successful trauma-related treatment leads to reductions in physiological and subjective distress reactions.

REFERENCE

- Lang, P. J., Melamed, B. G., & Hart, J. (1970). A psychophysiological analysis of fear modification using an automated desensitization procedure. *Journal of Abnormal Psychology*, 76, 220–234.

RECOMMENDED READINGS

- Keane, T. M., Kolb, L. C., Kaloupek, D. G., Orr, S. P., Blanchard, E. B., Thomas, R. G., et al. (1998). Utility of psychophysiological measurement in the diagnosis of post-traumatic stress disorder: Results from a Department of Veterans Affairs Cooperative Study. *Journal of Consulting and Clinical Psychology*, 66, 914–923.
- Orr, S. P., Metzger, L. J., Miller, M. W., & Kaloupek, D. G. (2004). Psychophysiological assessment of posttraumatic stress disorder. In J. P. Wilson & T. M. Keane (Eds.), *Assessing psychological trauma and PTSD: A handbook for practitioners* (2nd ed., pp. 289–343). New York: Guilford Press.

DANNY G. KALOUPEK

Veterans Affairs Boston Healthcare System

See also: Biofeedback; Biology, Physiology

ASYLUM SEEKERS

Refugees, applying for protection in a foreign country, are called asylum seekers. The 1951 UN Refugee Convention still provides the central legal framework across most of the world and asserts that, “No Contracting State shall expel or return (*refouler*) a refugee in any manner whatsoever to the frontiers of territories where his life or freedom would be threatened on account of his race, religion, nationality, membership of a particular social group or political opinion.” This is intended to provide an important safeguard for refugees who may fear imprisonment, torture, or death if they are captured by their state authorities. These protections may also be supplemented by other local frameworks such as the European Convention on Human Rights.

Asylum seekers who are not recognized as refugees may still have fled civil war or violent oppression, and thus are likely to have high trauma exposure and an elevated rate of physical and psychiatric morbidity. In addition to dealing with their past histories, asylum seekers face the challenges and difficulties of dealing with different cultural expectations as they transition into an alien world. Moreover, they must often accomplish this alone without the benefits of family and other social ties. They may feel adrift from a former political activism, isolated from their friends, colleagues, and communities.

It is a testimony to human resilience that many asylum seekers and refugees successfully adapt to their new circumstances, without developing persistent psychiatric disorder. There is a debate about the degree to which there is an elevation of psychiatric illness in asylum seekers. Different methodologies have been used to survey different populations of refugees with different cultural backgrounds and different experiences (Turner, 2004). However, the general conclusion is that there is a greater likelihood of psychiatric disorder in refugees and asylum seekers. In one study (Steel, Silove, Phan, & Bauman, 2002), evidence was found that this effect is still detectable many years later; although by then the absolute prevalence

figures for psychiatric disorder had fallen. Significantly, trauma exposure was found to be the most important predictor of mental health status in these refugees.

REFERENCES

- Steel, Z., Silove, D. M., Phan, T., & Bauman, A. (2002). Long-term effects of psychological trauma on the mental health of Vietnamese refugees resettled in Australia: A population-based study. *Lancet*, 360, 1056–1062.
- Turner, S. W. (2004). Emotional reactions to torture and organized state violence. *PTSD Research Quarterly*, 15(2), 1–7.

STUART TURNER

The Trauma Clinic, London, UK

See also: Laws, Legislation, and Policy; Refugees; Torture

ATTACHMENT

According to attachment theory (Bowlby, 1969), humans and other primates are biologically inclined to establish close affiliative bonds—that is, emotional attachments—beginning soon after birth. The most fundamental attachment relationship is formed between the biological mother and child, though there are various other pairings that qualify as primary attachments, both in early childhood and at various stages across the lifespan. Attachment theory is rooted in the premise from evolutionary theory that natural selection conferred an adaptive advantage to parents and infants who maintained close physical proximity to each other, which helped both to decrease infant mortality by reducing the risks of accidents and predation and to support the social maturation of the infant. Thus, attachment is thought to provide increased objective security and support for psychosocial development in the developing child. Therefore, the best types of attachments are those that reliably provide protection while also encouraging the child to engage in behaviors that foster learning and the development of skills for relating to other people (e.g., exploration and mastery of environments, active interaction with the primary

caregiver and other persons, and increasingly autonomous functioning).

While infants are biologically inclined to form attachment relationships with parents and other *attachment figures* (that is, persons who provide emotional and physical caregiving to the infant), the quality of these attachments varies depending on how caregivers interact with the infant. When caregivers are able to consistently ensure the physical safety and comfort of the infant, and also interact verbally and nonverbally in ways that are loving and responsive to the infant's biological and psychological needs, the emotional attachment tends to be beneficial to the infant's physical and psychosocial development. It is important to note that the caregiver need not be "perfect" in protecting and facilitating the infant's comfort and growth, but simply "good enough" to enable the infant to be and feel safe, cared for, and encouraged to explore the world (Winnicott, 1971).

The most commonly observed variations in infants' responses to adult caregiving are labeled *attachment styles*, which have been classified into typologies describing relational dispositions (that is, patterns of behavior by the infant in interacting with the caregiver) and working models (that is, beliefs and expectations that the infant develops about relationships). Attachment styles are first seen in early childhood, but tend to extend throughout childhood and adulthood in the person's primary family and peer relationships and intimate (e.g., marital, parenting) and friendship adult relationships. Thus, measures have been developed to assess both infants' and children's attachment styles, and also adolescent and adult attachment styles.

Developmental psychologist Mary Ainsworth and her coworkers (1978) developed an observational protocol known as the *Strange Situation Test*, the first and foremost method for assessing and classifying infants' and toddlers' attachment styles. The test involves a series of apparently simple, but theoretically and relationally powerful, activities that challenge the infant's ability to *tolerate separation* from a primary caregiver (e.g., the caregiver first plays with the infant and then leaves the infant with

another adult whom the infant does not know [the "stranger," who actually is a well-trained researcher who interacts pleasantly with the infant]). Most infants, particularly around the age of 12 to 18 months old, become distressed when their primary caregiver leaves, but the extent of their distress differs and provides an indication of their ability to tolerate the separation. Equally important, the test assesses the infant's capacity to *emotionally accept reunion* with the caregiver (i.e., the caregiver returns to the room and begins to console and play with the infant again). Attachment style has as much or more to do with the infant's ability to regain a calm and happy emotional state, and the caregiver's ability to "soothe" the child so as to facilitate that emotional regrouping, as it does with distress during separation. This line of research produced ample evidence of attachment behaviors and patterns that were consistent with and supportive of Bowlby's theoretical framework.

The most frequently occurring and advantageous attachment style is labeled as *secure attachment* because the child appears to feel a sense of emotional security before and after the separation, by demonstrating a balance between seeking and maintaining adequate proximity to the attachment figure (e.g., getting the caregiver's attention, moving closer to the caregiver) and engaging in developmentally appropriate exploratory behavior (e.g., leaving the caregiver's immediate area to play with toys) without showing signs of undue distress. When circumstances trigger increased activation of the attachment behavioral repertoire (such as after playing autonomously for a short time, or when the caregiver leaves the room), the child physically seeks contact with the attachment figure and will protest and resist separation. Upon reunion with the attachment figure, the securely attached child's distress can be readily soothed by sensitive and responsive behavior on the part of the attachment figure. Thus, a secure attachment style provides a reliable and responsive base both in the infant-caregiver relationship and in the child's inner sense of confidence in both self and the relationship that enables the infant to develop

trust and security both in relationships and in her or himself. The responsive and facilitative caregiver thus provides effective protection for the infant without inhibiting the infant's maturation as an autonomous individual.

Three variations on *insecure attachment*—a pattern of attachment characterized by fearfulness and distress rather than security and trust—have also been identified. An *ambivalent* attachment style describes a relationship in which the child shows distress upon separation from the attachment figure, but signs of ambivalence upon their reunion (e.g., anger, resistance to being soothed). An *avoidant* attachment style describes a relationship in which the child shows little or no response to separation from the attachment figure and little if any response upon reunion. A more recently identified attachment style is characterized as being *disorganized* (Main & Solomon, 1986) because the child appears not to have any consistent strategy for coping with separation from attachment figure and upon reunion may demonstrate combinations of freezing, avoidance, disorientation, anger, depression, and a variety of apparently odd behaviors (such as alternately clinging to or ignoring both the caregiver and the stranger). This disorganized attachment style is often observed among children who have been maltreated or whose parents have a psychiatric disorder (Main, 1995), and it is associated with significant psychosocial problems (such as chronic dissociation) decades later in adulthood (Lyons-Ruth et al., 2006). Disorganized attachment thus has special significance in relation to psychological trauma, as will be discussed later.

While these attachment styles are evident and classifiable in early childhood, and appear to remain relatively stable across time, they are also malleable and may change as the child or adult adapts to changing environments, relationships, and experiences. Bowlby (1969) hypothesized that attachments in infancy produce *internal working models* that are the person's fundamental (and typically unquestioned) assumptions guiding her or his lifelong expectations and behavior regarding close personal relationships. These internal working

models combine durability with plasticity—that is, they are relatively enduring yet can be modified—and serve to influence and limit subsequent relational behavior while undergoing persistent elaboration in the face of actual relational transactions. Attachment is thus considered to be an important aspect of personality across the life span with implications for social and relational functioning in a wide range of contexts (Bohlin, Hagekull, & Rydell, 2000; Hazan & Shaver, 1994).

Attachment and Psychological Trauma

Studies of attachment across the life span indicate that attachment styles are related to a variety of important aspects of social and psychological functioning. Among these are findings indicating that attachment styles predict symptoms of various mental disorders (e.g., Atkinson & Zucker, 1997; Greenberg, 1999; Pianta, Egeland, & Adam, 1996), with insecure and disorganized styles of attachment demonstrating significant linkages to psychological trauma.

Attachment theory provides an explanatory framework for those linkages because of the significant role that attachment relationships play in helping the child to cope with or recover from threats to optimal development. By definition, potentially traumatic events constitute major threats to physical and psychological integrity and development. Thus, when children experience psychological trauma, this may affect their style or working models of attachment in several ways. Moreover, the child's attachment style may affect how the child responds to the traumatic events. First, the traumatic events may alter formative attachment (bonding) experiences, such as by interfering with or preventing caregivers from protecting and providing responsive care to the child. Thus, traumatic events may influence the child's development of internal working models of attachment, potentially leading the child to adopt more insecure, ambivalent, or disorganized attachment styles.

Second, traumatic events also test the adaptive effectiveness of attachment models and behaviors that the child previously has

developed, potentially shifting a child from secure toward insecure attachment styles and working models.

Third, the child's preexisting attachment working models may reduce the adverse impact of traumatic stressors if the working models are predominantly secure, and this may "buffer" or protect the child from developing severe or persistent posttraumatic stress symptoms. However, if a child's attachment experiences and working models were primarily insecure or disorganized prior to exposure to potentially traumatic events, the child may be more likely to develop traumatic stress symptoms that are severe or persistent (as well as potentially shifting toward more insecure attachment styles) than a child who was securely attached.

Thus, while secure attachment may confer substantial resilience for children in the face of traumatic stress, and insecure attachment appears to constitute a risk factor for undesirable posttraumatic outcomes, the child's attachment style and working models also are vulnerable to the pernicious effects of acute and chronic exposure to traumatic stressors. Traumatic stressors experienced in early childhood place the individual at risk for problems with basic forms of self-regulation (such as difficulties in regulating emotions or managing physical injury or pain) and cognitive development (such as impairments in memory or judgment) which may be, in part, related to the disruption of secure attachment (Ford, 2005). Early life experiences with caregivers that promote the development of a secure style and working models of attachment teach children not only how to feel safe and trusting in relationships, but also how to develop skills for self-regulation and cognitive processing that the child needs in order to explore the world and achieve autonomy.

Secure attachment develops through experiences that have been labeled "co-regulation" (Schoore, 2001; Siegel, 2001), in which the infant learns to regulate her or his emotions, thinking, and body by experiencing the caregiver's own self-regulation. The caregiver thus "jump starts" the child's self-regulatory abilities by using her or his ability to reduce or increase

physical arousal level to directly alter the infant's arousal and affective states. The infant thus learns to self-regulate when the caregiver takes the lead and helps the infant to intuitively adopting similar self-regulatory strategies. For example, as a caregiver soothes a distressed infant, the infant learns experientially (that is, simply by experiencing the changes) how to use certain comforting forms of physical contact, tones of voice, and facial expressions to soothe her or himself.

When traumatic stressors occur in the life of a child or caregiver, this often makes it more difficult either for the caregiver to provide the intimate modeling of self-regulation that is needed or for the infant to attend to and "take in" the experience of co-regulating. The result in either case is that the caregiver has difficulty in protecting and being responsive to the child, which leads the child to have difficulty both in developing a secure sense of attachment and to learn how to self-regulate physically (Sethre-Hofstad, Stansbury, & Rice, 2002) and emotionally. The result can be a long-term impairment in the infant's relational and self-regulatory functioning that can undermine the child's effective use of social support, which is often cited as an essential aspect of resilience to traumatic stress. Thus, when exposure to traumatic stressors in early childhood disrupt the development of secure attachment, the child is less likely to be able to cope with traumatic stress reactions because she or he is likely to have difficulties with both self-regulation (that is, adjusting her or his bodily and emotion state) and getting help from others (that is, seeking and utilizing helpful social support).

Attachment and Treatment of Traumatic Stress Disorders

Attachment relationships and internal working models of attachment are promising targets for interventions with adults and children who exhibit signs of posttraumatic stress reactions. The purpose of attachment-oriented therapeutic interventions is to bolster resilience by improving existing relationships while increasing the potential for developing additional relationships

that can support the child's or adult's psychosocial functioning. Relational interventions informed by attachment theory may be conducted at any stage of life, beginning with traumatized young children and their caregivers (Van Horn & Lieberman, in press), but also including traumatized adolescents and adults (Cloitre, Cohen, & Koenen, 2006; Lamagna & Gleiser, 2007; Ogden, Minton, & Pain, 2006). Parents and others who provide care for infants and toddlers exposed to potentially traumatic events, even when these same caregivers may have been involved in abusing or neglecting the child, may improve their caregiving and relational skills with the benefit of careful training and feedback (*see: **Parent-Child Intervention***). Attachment-based dyadic (parent-child) interventions are designed to prevent further harm to the child while supporting the development of more effective attachment relationships with additional positive effects of the internal working models of children and their caregivers.

Attachment-based psychotherapies for adolescents and adults focus on providing a trustworthiness and responsive therapeutic relationship (*see: **Psychotherapeutic Processes***) and assisting the client(s) in developing increased recognition of and ability to regulate bodily reactions (Ogden et al., 2006), emotions (Lamagna & Gleiser, 2007), and beliefs (or *schemas*) about oneself and the world (Cloitre et al., 2006). Through the combination of experiencing security in the therapeutic relationship—which includes knowing that the therapist both cares about but also will not be excessively or intrusively involved with oneself—and learning by example and practice to develop self-regulation and interpersonal effectiveness skills, the attachment-based approaches to psychotherapy seek to help the client to develop a sense of internal security that was interrupted (Cloitre et al., 2006) or prevented by prior experiences of psychological trauma. An attachment approach to psychotherapy thus is consistent with all models of PTSD psychotherapy, and may be particularly important with clients who suffered early life betrayal traumas (*see: **Betrayal Trauma***) in the form of childhood maltreatment or family

violence (*see: **Complex Posttraumatic Stress Disorder***).

Conclusion

Attachment theory has received strong research support and gained widespread acceptance as an explanatory framework for understanding close relationships across the span of human development. A critical function of attachment is to support survival and adaptive development in situations ranging from optimal conditions to those of extreme adversity, with more secure attachment styles being predictive of more resilient adaptations. Thus, in the face of potentially traumatic experiences, attachment activation is likely and may provide a degree of protection against the impact of traumatic stress. In turn, since attachment styles reflect relational experiences, exposure to some types of traumatic stressors (e.g., intimate abuse, emotional betrayal) may influence the development of internal working models of attachment in ways that generally undermine relational functioning and decrease resilience to potentially traumatic events. Interventions informed by attachment theory show promise for strengthening attachment bonds with traumatized young and older children and adults, and of improving relational quality in ways that could improve resilience to stressful life events and assist in resolving the residual effects of prior exposure to traumatic stress.

REFERENCES

- Ainsworth, M. D. S., Blehar, M. C., Waters, E., & Wall, S. N. (1978). *Patterns of attachment: A psychological study of the strange situation*. Hillsdale, NJ: Erlbaum.
- Atkinson, L., & Zucker, K. J. (Eds.). (1997). *Attachment and psychopathology*. New York: Guilford Press.
- Bohlin, G., Hagekull, B., & Rydell, A-M. (2000). Attachment and social functioning: A longitudinal study from infancy to middle childhood. *Social Development*, 9, 24–39.
- Bowlby, J. (1969). *Attachment and loss: Attachment* (Vol. 1). New York: Basic Books.

- Cloitre, M., Cohen, L., & Koenen, K. (2006). *Treating survivors of childhood abuse: Psychotherapy for the interrupted life*. New York: Guilford Press.
- Ford, J. D. (2005). Treatment implications of altered neurobiology, affect regulation and information processing following child maltreatment. *Psychiatric Annals*, 35, 410–419.
- Greenberg, M. T. (1999). Attachment and psychopathology in childhood. In J. Cassidy & P. R. Shaver (Eds.), *Handbook of attachment: Theory, research, and clinical applications* (pp. 469–496). New York: Guilford Press.
- Hazan, C. & Shaver, P. R. (1994). Attachment as an organizational framework for research on close relationships. *Psychological Inquiry*, 5, 1–22.
- Lamagna, J., & Gleiser, K. (2007). Building a secure internal attachment: An intra-relational approach to ego strengthening and emotional processing with chronically traumatized clients. *Journal of Trauma and Dissociation*, 8, 25–52.
- Lyons-Ruth, K., Dutra, L., Schuder, M., & Bianchi, I. (2006). From infant attachment disorganization to adult dissociation: Relational adaptations or traumatic experiences? *Psychiatric Clinics of North America*, 29, 63–86.
- Main, M. (1995). Attachment: Overview, with selected implications for clinical work. In S. Goldberg, R. Muir, & J. Kerr (Eds.), *Attachment theory: Social, developmental, and clinical perspectives* (pp. 407–474). Hillsdale, NJ: Analytic Press.
- Main, M., & Solomon, J. (1986). Discovery of an insecure disorganized/disoriented attachment pattern: Procedures, findings and implications for classification of behavior. In M. W. Yogman & T. B. Brazelton (Eds.), *Affective development in infancy* (pp. 95–124). Norwood, NJ: Ablex.
- Ogden, P., Minton, K., & Pain, C. (2006). *Trauma and the body: A sensorimotor approach to psychotherapy*. New York: Norton.
- Pianta, R. C., Egeland, B., & Adam, E. K. (1996). Adult attachment classification and self-reported psychiatric symptomatology as assessed by the Minnesota Multiphasic Personality Inventory-2. *Journal of Consulting and Clinical Psychology*, 64, 273–281.
- Schore, A. (2001). Effects of a secure attachment relationship on right brain development, affect regulation, and infant mental health. *Infant Mental Health Journal*, 22, 7–66.
- Sethre-Hofstad, L., Stansbury, K., & Rice, M. (2002). Attunement of maternal and child adrenocortical response to child challenge. *Psychoneuroendocrinology*, 27, 731–747.
- Siegel, D. (2001). Toward an interpersonal neurobiology of the developing mind. *Infant Mental Health Journal*, 22, 67–94.
- Van Horn, P., & Lieberman, A. (in press). Using dyadic therapies to treat traumatized children. In D. Brom, R. Pat-Horenczyk, & J. D. Ford (Eds.), *Treating traumatized children: Risk, resilience, and recovery*. London: Routledge.
- Winnicott, D. W. (1971). *Playing and reality*. London: Routledge.

GILBERT REYES

Fielding Graduate University

JULIAN D. FORD

University of Connecticut School of Medicine

See also: Betrayal Trauma; Child Development; Child Maltreatment; Complex Posttraumatic Stress Disorder; Family Systems; Parent-Child Intervention; Psychodynamic Psychotherapy, Child; Psychotherapeutic Processes

ATTENTION

See: Cognitive Impairments; Information Processing

ATTRIBUTIONS

Attribution theorists contend that individuals who experience unexpected, unwanted, or otherwise unusual events are motivated to create explanations that lend meaning to those events. Further, the explanations they construct (i.e., attributions) may have implications for subsequent adjustment.

Attributions have generally been considered with reference to four dimensions—locus, stability, controllability, and generalizability. Locus refers to assigning causality to either oneself (internal attribution) or to other persons or environmental factors (external attributions). The dimension of stability refers to whether the cited cause of an event is enduring and unremitting, or whether it is transient in nature. By way of example, attributing a motor vehicle accident to a malfunctioning traffic

signal would be relatively unstable, whereas attributing an accident to a perceived increase in bad drivers would be a much more stable attribution. Generalizability (also referred to in the literature as globality) involves the degree to which the individual attributes the event to factors that pervade many aspects of their life (e.g., personality, intellect) or very few. The dimension of controllability relates to whether the causal factors culminating in an event are deemed by the individual to be personally modifiable. Clearly, this dimension is not independent of locus and some have suggested that controllability is an aggregate of locus and stability.

The role of causal attributions has been examined within the area of posttraumatic stress disorder (PTSD) and it has been proposed that the type of causal explanations an individual creates to explain the occurrence of an event may serve as a risk factor for developing the disorder. Evidence from the literature has supported the hypothesis that certain attributional styles may be more adaptive than others. An attributional style refers to habitual or dispositional tendencies to invoke certain types of causal explanations for varied life events. For example, a pessimistic attributional style, comprised of internal, global, and stable attributions, has been shown to be associated with more severe PTSD symptoms. Recent research indicates that certain attributional tendencies may differ in adaptiveness depending on the type of trauma experienced. For example, there is general consensus in the literature that internal attributions (i.e., self-blame) for trauma are associated with poorer adjustment among sexual assault survivors. Among combat survivors and motor vehicle accident victims, however, some studies have documented poorer adjustment when an external attributional style (i.e., other blame) is maintained.

As is often the case, apparently discordant findings in the literature may result from methodological discrepancies as opposed to modest or unstable associations among the variables of interest. By way of example, some studies purporting to evaluate attributional tendencies as

they relate to posttraumatic distress have utilized third parties to corroborate responsibility for the event (e.g., motor vehicle accident). In so doing, these studies inadvertently focus on true causality that is less relevant from an attributional perspective. There are many maladaptive posttraumatic attributions (e.g., self-blame following a sexual assault) that are objectively erroneous but nevertheless have significant implications for subsequent adjustment.

In terms of other methodological considerations, most studies to date have been cross-sectional in nature, which renders causal statements premature at this time. Also, dispositional attributional style and event-specific attributions have typically not been examined concurrently with known risk factors for PTSD. Accordingly, the relative importance of causal attributions as a posttraumatic risk factor is largely unknown. For a more detailed review of the role of causal attributions in PTSD, the reader is referred to Massad and Hulsey's (2006) examination of attributions and their implications for clinical research and practice.

REFERENCE

- Massad, P. M., & Hulsey, T. L. (2006). Causal attributions in posttraumatic stress disorder: Implications for clinical research and practice. *Psychotherapy: Theory, research, practice, training*, 43(2), 201–215.

CHRISTINA M. HASSIJA
University of Wyoming

MATT J. GRAY
University of Wyoming

See also: Cognitive Behavior Therapy, Adult; Cognitive Behavior Therapy, Child Abuse; Cognitive Behavior Therapy, Childhood Traumatic Grief; Social Cognitive Theory

AVOIDANCE

Avoidance is commonly observed among people with elevated anxiety and is even more pronounced among people with anxiety disorders.

Avoiding situations that are reminiscent of a traumatic event is a diagnostic feature of post-traumatic stress disorder (PTSD). Avoidance can be more or less apparent, because some forms of avoidance include avoiding thoughts or feeling that are associated with traumatic experiences.

The current diagnostic criteria for PTSD classify emotional numbing into the avoidance symptom cluster, although it is questionable that numbing is a form of avoidance. Recent research, moreover, suggests that avoidance and numbing are better explained as separate factors that differ in their clinical correlates, prognostic significance, and response to treatment (Asmundson, Stapleton, & Taylor, 2004). These findings are consistent with theories suggesting that numbing arises from a biological mechanism associated with a response called *conditioned analgesia*, which dampens arousal. In contrast, avoidance appears to be a more conscious and intentional strategy for regulating distressing emotional states.

Some forms of avoidance are obvious, such as when a survivor of a serious motor vehicle accident goes to considerable effort to avoid passing by the site of the accident, or when a combat veteran avoids watching news pertaining to war. Other forms of avoidance are not so obvious. For example, a physically abused child might refrain from making eye contact with the abusive parent in an effort to avert provocation of another beating. Likewise, to circumvent anxiety-related bodily sensations (e.g., palpitations, pain) that can serve as reminders of traumatic events, a person may refrain from physical exertion, such as household chores. Perhaps most subtle of all, some people try to avoid trauma-related thoughts by deliberately suppressing these thoughts by means of distraction.

Avoidance can be an effective strategy for managing fear or distress and, in some cases, can be adaptive. An example of adaptive avoidance might include staying away from parts of the city where violent crimes are known to be high. But avoidance in people with PTSD is generally not adaptive because, while it does alleviate distress in the short-term, it may also

prevent the person from experiencing the corrective information necessary for learning how to distinguish between safe and risky situations. Deliberate attempts at thought suppression can also have the paradoxical effect of increasing trauma-related thoughts, which in turn may increase the severity and frequency of re-experiencing symptoms. Therefore, avoidance is more likely to exacerbate and perpetuate PTSD symptoms than promote adaptive behavior.

A potent alternative to avoiding trauma-related stimuli is to intentionally expose oneself to safe but distressing reminders under controlled conditions. Studies have demonstrated that the most effective means of treating avoidance is through the application of one or more forms of exposure therapy (Taylor, 2006). These include imaginal, interoceptive (i.e., to fear-evoking but harmless bodily sensations), and situational exposure. Each form of exposure is designed to allow the traumatized person to re-establish the capacity for distinguishing between situations that are reasonably safe and those that pose real danger. This in turn allows patients to resume activities consistent with their pre-trauma level of functioning and promotes more adaptive and progressive improvements in functioning.

REFERENCES

- Asmundson, G. J. G., Stapleton, J. A., & Taylor, S. (2004). Avoidance and numbing are distinct PTSD symptom clusters. *Journal of Traumatic Stress, 17*, 467–475.
- Taylor, S. (2006). *Clinician's guide to PTSD: A cognitive-behavioral approach*. New York: Guilford Press.

GORDON J. G. ASMUNDSON
University of Regina

STEVEN TAYLOR
University of British Columbia

See also: Conditioned Fear; Emotional Numbing; Exposure Therapy, Adult; Exposure Therapy, Child; Posttraumatic Stress Disorder

