Posttraumatic Stress Disorder Part III: Health Effects of Interpersonal Violence Among Women

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**TOPIC.** The aim of this three-part series is to examine the sufficiency of the posttraumatic stress (PTSD) diagnostic construct to capture the full spectrum of human responses to psychological trauma. Part I (Lasiuk & Hegadoren, 2006a) reviewed the conceptual history of PTSD from the nineteenth century to its inclusion in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association, 1980), while Part II (Lasiuk & Hegadoren, 2006b) described subsequent refinements to the original PTSD diagnostic criteria and highlighted subsequent controversies.

**PURPOSE.** This paper focuses on interpersonal violence (sexual, physical, and emotional abuse/assault) and its sequelae in women. We argue in support of Judith Herman's (1992) conceptualization of the human trauma response as a spectrum, anchored at one end by an acute stress reaction that resolves on its own without treatment, and on the other by “complex” PTSD, with “classic” or “simple” PTSD somewhere between the two.

**SOURCES OF INFORMATION.** The existing theoretical, clinical and research literatures related to humans responses to trauma.

**CONCLUSION.** The paper concludes with a call for the need to increase a gendered perspective in all aspects of trauma research and clinical service delivery.


The inclusion of posttraumatic stress disorder (PTSD) in the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM-III; American Psychiatric Association [APA], 1980) reflected a response to social and political pressures of the day more than it did the state of the science on trauma (Cloitre, Koenen, Gratz, & Jakupcak, 2002; Yehuda & McFarlane, 1995). During the 1960s and 1970s, Vietnam War veterans, profoundly affected by their experience of war, returned to a Veterans Administration (V. A.) that was grossly unprepared to meet their needs. This spurred veterans, their families and friends, and mental health practitioners to successfully campaign for changes within the V. A. and influenced the third revision of the DSM in which both combat and civilian (e.g., rape trauma syndrome, battered woman syndrome, abused child syndrome) trauma response syndromes were subsumed under the diagnosis of PTSD.

The PTSD diagnosis was a benchmark in North American psychiatry in that it recognized the psychological effects of horrific life events and established both a language and conceptual framework for the systematic study of trauma and its sequelae. At the same time, because the original PTSD diagnostic criteria drew heavily on Abram Kardiner’s (1978) early works with men in combat, they were inherently gender-biased. Now 26 years later, there is clear evidence that gender influences the type of trauma that individuals experience, social factors that mediate the impact of exposure, and the way that trauma is encoded into meaning (Stewart, Ouimette, & Brown, 2002). Despite this, gender-specific factors are not systematically included across trauma studies or in service delivery.
PTSD: Health Effects of Interpersonal Violence Among Women

Not only does this make cross-study comparisons difficult, but it limits our understanding of the full-range of potential outcomes after trauma and hinders our ability to provide comprehensive and integrated treatment response.

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Interpersonal Trauma

Definition

Ten years ago, the 49th World Health Assembly (1996) declared violence to be a global public health problem. This prompted a closer analysis of the phenomenon worldwide and culminated in the World Health Organization (WHO) World Health Report on Violence and Health (Krug, Dahlberg, Mercy, Zwi, & Lozano, 2002). That document defines violence as

The intentional use of physical force or power, threatened or actual, against oneself, another person, or against a community, that either results in or has a high likelihood of resulting in injury, death, psychological harm, maldevelopment, or deprivation. (p. 5)

The Report goes on to identify three types of violence—self-directed, interpersonal, and collective. Interpersonal violence includes two subcategories—family and intimate partner violence and community violence. As its name suggests, family and intimate partner violence involves family members and intimate partners, generally within the home and includes physical, sexual, psychological, and/or deprivation/neglect. Community violence usually occurs outside of the home, between or among individuals who may or may not know each other.

The WHO (2002) estimates that in the year 2000, 1.6 million individuals worldwide died as the result of violence. “Not-fatal violence” (p. 11)—acts of violence that result in injury but not in death—is as troubling as the number of violence-related deaths. However, given the definitional, reporting, and measurement issues that exist, there is wide agreement that even the best estimates of the scope of interpersonal violence represents the proverbial tip of the iceberg (Krug et al., 2002). In the wider literature, as here, the terms interpersonal violence and interpersonal trauma are used interchangeably.

Epidemiology of Interpersonal Violence

Although the scope of this paper prohibits a detailed summary of the epidemiological literature on interpersonal violence, we will endeavor to highlight the salient themes. Large-scale epidemiological studies are blunt instruments. In part, this is due to the kinds of problems inherent in measurement of any kind. As noted earlier, problems of conceptualization, operationalization, instrumentation, and information collection strategies can affect the nature, quality, and accuracy of data that epidemiologists collect. With these limitations in mind, population studies furnish our best glimpses into the incidence and prevalence of traumatic events. One such study is the U.S. National Comorbidity Survey (NCS; Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995), which employed face-to-face interviews with a representative sample of 5,877 community-dwelling individuals, aged 15–54 years. The findings of the NCS reiterate a now familiar pattern of themes borne out consistently in similar studies.
around the globe. The first of these themes relates to frequency of exposure to traumatic events. In this study, over 50% of the participants reported experiencing at least one traumatic event in their lifetime. Here as elsewhere, men experienced traumatic events more often than women did, with 60.7% of the men and 51.2% of the women in the NCS reporting at least one lifetime trauma. In addition, the majority of respondents who reported trauma exposure experienced more than one type of trauma.

A second theme in the NCS (Kessler et al., 1995) concerns a gender differential across the type of trauma experienced. Men more often report physical attacks, combat experience, and being threatened with a weapon, held captive, or kidnapped. By comparison, higher numbers of women report rape, sexual molestation, childhood parental neglect, and childhood physical abuse. Similarly, in other U.S. studies girls report sexual abuse at rates two to three times higher than boys do (Breslau, Davis, Andreski, Peterson & Schultz, 1997; MacMillan et al., 1997). More than 6 out of 10 of all rapes (61%) in the United States involve girls under the age of 18, with 29% of all forcible rape occurring to girls younger than 11 (National Center for Victims of Crime, 1992). In Canada, girls under the age of 18 were the victims in 79% of the 2,353 family-related sexual assaults reported to police in 2001 (Statistics Canada, 2004).

A third theme in the NCS speaks to the effects of exposure to traumatic events. Using the clinical diagnosis of PTSD as an index, the NCS (Kessler et al., 1995) concludes that women are twice as likely as men to develop PTSD following exposure to a traumatic event (10.4% vs. 5.0%). This last theme is the focus of considerable study and debate in the trauma literature, with the central question being “What makes females so much more vulnerable to PTSD than men?” The answer to this questions is contingent on a number of complex and interrelated features of the individual; the traumatic event itself; and the social and physical environments during the trauma and afterwards. All that said, females—women and girls—experience higher rates of the type of trauma associated with PTSD in both sexes, that is, interpersonal trauma. In the NCS for example, rape is associated with the highest conditional probability of developing PTSD (men 65% and women 45.9%). Stated differently, one explanation for why women develop PTSD twice as often as men do is women’s more frequent exposure to interpersonal trauma. Only prisoner-of-war experiences carry similar risk of PTSD, which supports Herman’s (1992) belief that powerlessness is central to the development of PTSD.

Human Stress Response

Stress Response Systems

From a biological perspective, the perception of any threat to our life or personal integrity triggers an automatic, total-body response that Cannon (1939) dubbed as fight-or-flight response. Within seconds of the perception of a threat, cascades of hormones and neurotransmitters course through the body marshalling every cell into action (Bloom, 1997; Sapolsky, 2004). When the danger subsides, the body returns to its normal state. Two major stress response systems underlie the fight-flight response. The first of these involves release of norepinephrine (also termed noradrenaline) from the brain stem, which activates the adrenal medulla to release epinephrine (also termed adrenaline) and increase sympathetic nervous system tone. This increased tone results in increases in heart rate and blood pressure, bronchodilation, and decreased gastrointestinal activity. The second stress response system is the hypothalamic–pituitary–adrenal (HPA) axis. Stressors stimulate the hypothalamus to release corticotrophin-releasing hormone (CRH), which interacts with receptors in the pituitary to release adrenocorticotrophic hormone (ACTH) into the systemic circulation. ACTH stimulates receptors in the adrenal cortex to release cortisol and negative feedback at the level of the hypothalamus and pituitary limit the amount and duration of increased cortisol levels
PTSD: Health Effects of Interpersonal Violence Among Women

Figure 1. Simplified models of HPA axis activity under varying conditions.

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CRH – corticotrophin releasing hormone; ACTH – adrenocorticotrophic hormone; OT – oxytocin.

(Cortisol has multiple metabolic effects that increase available glucose to skeletal muscles, cognitive effects that limit memory processing during a highly stressful event, and augments the adrenal-releated increases in heart rate and blood pressure. Cortisol also has wide-ranging effects on immune function and metabolic homeostasis and chronic activation of the HPA axis is linked to cardiovascular, endocrine, and autoimmune disorders (Bunker et al., 2003; Okamura et al., 2000; Wittchen, Lieb, Wundrich, & Schuster, 1999).

Neurobiology of Extreme Stress

Despite the prevalence of interpersonal trauma in women and the related risk of PTSD, there have been limited gender-specific studies looking at the full impact of such experiences and few attempts to build well-integrated theoretical frameworks to explain the underlying mechanisms of the various changes observed in biological systems. The majority of the studies examining the basic organization of stress response systems employ male laboratory animals. However, a common theme that may be relevant to PTSD related to interpersonal trauma is powerlessness. In animal models, the lack of control over the stimulus and inability to escape leads to a cluster of behaviors termed learned helplessness (e.g., passive coping behaviors, decreased attempts to escape, hypoactivity) (Edwards, King, & Fray, 1999; Shumaker, Edwards, & Gonzalez-Lima, 2003). Analogous behaviors in those who live in situations of prolonged interpersonal trauma include passive acceptance of the violence, ambivalence around terminating the abusive relationship, and the lack of affect surrounding the violence. The experience of mental defeat (reflecting helplessness) and lower perceived control over responses have been identified as strong predictors of PTSD severity (Brown & Holmes, 2003; Frazier, 2003).

In humans, most trauma research involves combat veterans and motor vehicle crashes (Beckham et al., 2002; Orr, Lasko, Shalev, & Pitman, 1995; Shalev, Schreiber, Galai, & Melmed, 1993). Characteristic findings in PTSD populations include elevated resting heart rate, an exaggerated heart rate response to trauma cues, and increased startle responses to loud tones. Increased plasma cortisol and blunted diurnal variability are consistent findings in depression (Holsboer & Barden, 1996; Nemeroff, 1991) (Figure 1b). However, there have been conflicting findings regarding basal cortisol levels in PTSD, with lower levels (Boscarino 1996; Yehuda, Teicher, Trestman, Levengood, & Siever, 1996) or no change (Halbreicht et al., 1989; Rasmusson et al., 2001; Stein, Yehuda, Keverola, & Hanna, 1997) being reported. Control of factors such as type of trauma, gender, age, and medications partly explains this variability (Bremner & Vermetten, 2001; Rasmussen, Milton, Green, & Puchalski, 2001; Yehuda, 2002).

Neuroendocrine challenge tests to assess HPA axis function add further important information about stress response regulation beyond that of basal measures (King & Hegadoren, 2002). The most common of
these is the dexamethasone (DEX) suppression test (DST). The DST examines the ability of DEX, as a glucocorticoid receptor agonist, to exert negative feedback control over cortisol release (Figure 1b & c). In contrast to the blunted response to DEX observed in major depressive disorder (MDD), researchers have shown an exaggerated degree of cortisol suppression following DEX in PTSD (Yehuda, 2002). However, biological investigations in depression have paid little attention to whether depression in the context of a history of traumatic events comorbid with chronic trauma-related symptoms represents a subgroup with distinct associated biological findings. In addition, many studies did not control for menstrual phase, yet the degree of DEX-induced suppression of cortisol differs between the follicular and luteal phases (Altemus et al., 1997). The few studies that were done with women with interpersonal trauma show an HPA axis profile more similar to PTSD than depression (Shia, Walsh, MacMillan, & Steiner, 2004; Hegadoren, unpublished). Recent evidence from neuroimaging studies suggests that brain activation patterns differ between those with PTSD dominated by arousal symptoms and those with PTSD dominated by dissociative symptoms (Lanius et al., 2002). This is an important area for further research, as dissociation is more common with women after interpersonal trauma and carries with it an increased risk of developing chronic PTSD and for poor response to either drug therapies or specific psychotherapies.

In addition to alterations in central and peripheral HPA function, stress-related disorders may involve changes in other steroid hormones and neuropeptides. A neuropeptide of keen interest in stress research in women is oxytocin (OT). Whilst OT is best known for its roles in mating, parturition, lactation, maternal behavior, and pair bonding (Argiolas & Gessa, 1991; DeVries, Clasper, & Detillion, 2003), OT also inhibits HPA axis activity in animals (Gibbs, 1984; Petersson, Hulting, & Uvnas-Moberg, 1999) and acute injections of OT decrease anxiety and blood pressure (McCarthy, 1995; Page et al., 1996; Petersson, Alster, Landeberg, & Uvnas-Moberg, 1996) (Figure 1d). Given its dual role in social attachment and stress-modulation, OT has been theorized to participate in what has been termed the "tend and befriend" pattern of stress-related behaviors in women, as opposed to the more aggressive behaviors seen typically in men (Taylor et al., 2000).

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The Phenomenology of Interpersonal Violence

Although the experience of interpersonal violence originates in the external world, its effects quickly pervade the body and the mind (van der Kolk, McFarlane, & Weisaeth, 1996). At its centre is the experience of intense fear, helplessness, or horror (Herman, 1992; van der Kolk et al., 1996). Mardi Horowitz (1997) frames these as experiences that cannot be assimilated into a person's schema of their self-in-relation-to-the-world. Interpersonal violence disrupts one's sense of identity, basic trust in other people, and trust in the world as a safe, predictable place. Because of the intensely personal nature of interpersonal violence, victims and survivors often live with debilitating shame and self-blame. When they ask their self "Why did this happen to me?" it is often a small, childlike voice from within that responds, "Bad things happen to bad people." Terrorized by what has happened to them, victims and survivors are desperate for comfort. At the same time, they are often full of self-loathing and doubt and lack faith that others will understand and respond to them. As one rape survivor, put it "...
was terrified of being with people and terrified of being alone" (cited in Warshaw, 1988, p. 68).

When neither resistance nor escape is possible, the human self-defense system becomes overwhelmed and disorganized. Each component of the ordinary response to danger, having lost its utility, tends to persist in an altered and exaggerated state long after the danger is over. (Herman, 1992, p. 34)

These extended and cumulative effects of stress-responsive hormones and neuro-modulators can leave very long-term and perhaps permanent changes in both biological systems and psychological schemas. They live with alterations in consciousness, self-perceptions, systems of meaning, ability to regulate emotion, and ability to relate with others. Clinicians and researchers cluster these changes into three categories: hyperarousal, intrusion, and constriction.

### Traumatic events are set into memory differently than are ordinary experiences.

Hyperarousal keeps the survivors at the edge of terror—they startle easily, are irritable, and sleep poorly. It is as if having glimpsed it once, their mortality remains omnipresent like a dark shadow on the periphery of their awareness. Intrusion is the imprinting of trauma on the mind and body. It is what we see in the images of September 11—faces looking on, frozen and wide-eyed in disbelief. Traumatic events are set into memory differently than are ordinary experiences. "Traumatic memories lack verbal narrative and context; rather they are encoded in the form of vivid sensation and images" (Herman, 1992, p. 38), which flood survivors' consciousness as flashbacks during waking states and as traumatic nightmares during sleep. Flashbacks are triggered by significant reminders of the traumatic event—sights, sounds, odors, tastes, and physical sensations—that transport the survivor back into the trauma. A flashback is not an ordinary memory; it is a vivid reliving of the trauma and engulfs the survivor with the full emotional force of the original event. Constriction is the numbing that is required for surrender. Unable to fight off or escape the threat, survivors dissociate from the traumatic event so that they can endure it. Constriction alters perception, sensation, and time sense. In this dissociated state individuals report feeling detached from their bodies or of viewing the traumatic event dispassionately, as though it was happening in a movie. In these states, events continue to register but they are severed from their ordinary meaning (Herman). It is common, when flashbacks occur, for survivors to go into dissociative states.

### The Health Effects of Interpersonal Trauma

The recognition that relationships exist among stress, illness, and disease is not new. Recent world events serve only as dramatic reminders of what we already know. Several lines of research converge on the conclusion that traumatized individuals have poorer physical and mental health and a lower health-related quality of life (Felitti et al., 1998; Resnick, Acierno, Kilpatrick, & Kilpatrick, 1997; Schnurr & Green, 2004; Walker et al., 1999). Trauma, especially when it occurs early in life, is a major contributor in the development of psychiatric illness, particularly depression (Ballanger et al., 2004; Kendler, Karkowski, & Prescott, 1999). The fact that women with histories of childhood adversity have a fourfold increased risk for depression leads some researchers to endorse a model in which the causal pathways of PTSD and major depression following trauma are not independent (Breslau, Davis, Peterson, & Schultz, 2000; Stein & Kennedy, 2001). Indeed, Cutler and Nolen-Hoeksema (1991) suggest that CSA accounts for 35% of the increased prevalence rates of depression in women.
Survivors of interpersonal trauma utilize health services far beyond those directly related to the treatment of injury sustained during the traumatic event (Schnurr & Green, 2004). Adversity in childhood, including physical and sexual abuse, is associated with chronic pelvic pain (Reiter & Gambone, 1990), gastrointestinal disorders (Scarinci, McDonald-Haile, Bradley, & Richter, 1994), intractable low back pain (Schafferman, Anderson, Hines, Smith, & Keane, 1993), and chronic headache (Felitti, 1991). Compared with women without histories of interpersonal trauma, survivors have greater functional disability, more physical symptoms, more physician-coded diagnoses, and more health risk behaviors, including driving while intoxicated, unsafe sex, and being obese (Walker et al., 1999). Among survivors in one HMO, Felitti and his colleagues (1998) found a direct relationship between the number of childhood adversities and the presence of adult diseases, including ischemic heart disease, cancer, chronic lung disease, skeletal fractures, and liver disease. In addition, survivors often attempt to cope with or avoid their distress through drug and alcohol use, self-mutilation, suicide, and disordered eating, further increasing the risk of physical health problems (Springs & Friedrich, 1992).

Other studies have reported increased rates of psychiatric comorbidity after interpersonal trauma (Kessler et al., 1995), underdiagnoses in physician practices, underestimates of burden of distress and impairment (Kendler, Gardner, & Proscott, 2002; Mayou & Farmer, 2002), and increased hospitalization for poor treatment response (Firsten, 1991; Thase, Rush, Kasper, & Nemeroff, 1995). Depression associated with histories of CSA often carries with it severe psychological burden, a protracted longitudinal course, and an increased risk of poor treatment response to antidepressant therapy (Boudewyn & Liem, 1995; Hall, Sachs, Rayens, & Lutenbacher, 1993).

Studies specific to women who experience domestic violence also support the complexity of responses to such experiences. The most common psychiatric disorder in this population is depression (Hegarty, Gunn, Chondros, & Small, 2004; Kramer, Lorenzon, & Mueller, 2004; Ratner, 1993; Stein & Kennedy, 2001). Rates of MDD in physically abused women range from 66% to 80% (Follingstad, Wright, Lloyd, & Sebastian, 1991; Goodman, Koss, Fitzgerald, Russo, & Keita, 1993). Other associated disorders include PTSD, anxiety, addictions, and chronic pain syndromes (Carlson, Manut, & Choi, 2003; Sharhabili-Arzy, Amir, Kotler, & Liran, 2003). In addition to full syndromal diagnoses of MDD or PTSD, subthreshold symptoms are also associated with considerable functional impairment (Cuijpers & Smil, 2004; Lewinsohn, Shankman, Cai, & Klein, 2004; Zlotnick, Franklin, & Zimmerman, 2002).

"Complex" Posttraumatic Stress Disorder

This mounting evidence informs a growing consensus that using PTSD as the sole index for interpersonal trauma has limited utility. Judith Herman (1992) was among the first to challenge the sufficiency of the PTSD diagnostic construct, to capture the full range of human response to trauma. Based on her extensive clinical work with Holocaust survivors and survivors of sexual assault, she argues that as the nature, severity, and temporal characteristics of traumatic events vary so do individual responses. Herman believes that it is more accurate to conceptualize the human trauma response as a spectrum. This spectrum is anchored at one end by an acute stress reaction that resolves on its own without treatment, and on the other by what Herman calls "complex posttraumatic stress disorder," with "classic or simple" PTSD residing somewhere between the two (p. 119).

In 1994, the diagnostic construct of "complex" PTSD was officially sanctioned by North American psychiatric community when it was included in the DSM-IV (APA, 2000) under the diagnostic label Disorders of Extreme Stress not Otherwise Specified (DESNOS). The diagnostic criteria for DESNOS include:
PTSD: Health Effects of Interpersonal Violence Among Women

(i) alterations in affect regulation, which includes difficulty with modulation of anger and self-destructiveness
(ii) alterations in attention and consciousness leading to amnesias and dissociative episodes and depersonalizations
(iii) alterations in self-perception, such as a chronic sense of guilt and responsibility, chronically feeling ashamed
(iv) alterations in relationship to others, such as not being able to trust, not being able to feel intimate with people
(v) somatization—the expression of somatic symptoms on a somatic level for which no medical explanations can be found
(vi) alterations in systems of meaning

The DSM-IV field trial (Roth, Newman, Pelcovitz, van der Kolk, & Mandel, 1997), which examined whether complex PTSD is a separate entity from PTSD as described in the DSM-III (APA, 1980), failed to provide evidence of mutual exclusivity and DESNOS was not included in the DSM-IV (APA, 1994). However, the field trials added further support that women are at higher risk for interpersonal violence, that CSA and physical abuse carry the most serious risk for psychiatric problems, and that the differences between DESNOS and PTSD were clinically meaningful.

These diagnostic distinctions are important because they orient health practitioners to the fact that they encounter individuals with histories of interpersonal violence frequently in their everyday practice. Survivors are individuals—patients, clients, healthcare consumers—of every age, who seek services in every area of specialty. In some instances, practitioners will have knowledge that the individual before them is a survivor, but in most brief encounters, they will not. Given the intimacy and (often) invasiveness of procedures and treatments, it should be of surprise to no one that for many survivors, health-serving environments are very frightening places. What are simple, routine encounters for health professionals can be very distressing for survivors, because much about health-serving environments is reminiscent of the original trauma, including a sense of powerlessness, lack of control, invasion of personal boundaries, exposure, vulnerability, and pain. A simple touch, even when it is gentle, can trigger a flashback.

Most health professionals will not develop the specialized knowledge and skills needed to work with survivors through their healing. However, it is important for all practitioners to know about the long-term effects of interpersonal trauma so that they can respond sensitively to its survivors and routinely include broader assessment regarding the historical and current contexts in which women live their lives.

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Future Directions

Interpersonal trauma is a serious problem and can have profound effects on women’s health. The diagnosis of PTSD fails to address the complexity and diversity of women’s responses to these types of traumatic experiences. Various attempts have been made to address the limitations of this diagnosis, including the formulation of a distinct diagnostic category. Although complex PTSD or DESNOS was not considered a distinct diagnosis in the latest DSM classification system, it is still used by researchers and clinicians who work with traumatized women. The coexistence of PTSD and depression has led some researchers to suggest that a cascade model, which can meet full or partial criteria for a number of
Axis I and Axis II disorders, might fit better than a distinct two-disorder comorbidity model (Stein & Kennedy, 2001). There is a crucial need to develop integrated models of stress disorders in women that include both contextual factors, as well as gender-specific biological data about stress responses in women across the life span, from premenopausal menstrual cycles to perimenopause through to postmenopause.

Trauma researchers Harvey and Bryant (2002) suggest the need to further delineate the basis for trauma-related symptoms.

Future research needs to specify the way in which the biological, cognitive, and dissociative responses to trauma interact...to proceed beyond the umbrella term of dissociation and specify the processes that lead to alterations in encoding, retrieval, perception and reactivity...to elucidate the role of appraisals in mediating adjustment.

Ongoing gender-specific research is fundamental to the development of gender-sensitive practice guidelines and programs to address the full impact of interpersonal trauma on women's health.

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PTSD: Health Effects of Interpersonal Violence Among Women

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