Understanding PTSD and Sexual Assault

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Posttraumatic stress disorder (PTSD) is one of the most common mental health consequences of sexual assault (SA) (Dworkin, Menon, Bystrynski, & Allen, 2017). In this chapter, we begin by introducing the diagnosis of PTSD and reviewing prominent theories regarding how PTSD develops after SA. Then, we discuss the prevalence of PTSD after SA and factors that may increase risk. After discussing best clinical practices for assessing and treating PTSD after SA, we conclude with future directions for research, policy, and practice.

What is PTSD?

According to the Diagnostic and Statistical Manual of Mental Disorders, fifth Edition (DSM-5), PTSD involves a set of symptoms that develop in the wake of exposure to specific qualifying events, including SA (American Psychiatric Association, 2013). DSM diagnostic criteria for PTSD includes four "clusters" of symptoms experienced in reaction to traumatic events. *Reexperiencing* symptoms involve persistently reliving the SA, including nightmares, flashbacks, and intrusive memories. *Avoidance* symptoms include avoidance of trauma-related stimuli, such as situations that remind the survivor of the SA, and SA-related emotions, thoughts, and memories. *Negative changes in mood or cognition* can involve persistent negative beliefs about oneself and the world, excessively blaming oneself or others for the SA or its aftermath, persistent negative emotions, and a lack of positive emotions. *Alterations in arousal and reactivity* symptoms include problems with irritability, hypervigilance, sleep, and concentration. Such symptoms are observed in most survivors in the first weeks after SA and are considered a natural reaction to trauma (Rothbaum, Foa, Riggs, Murdock, & Walsh, 1992). Their failure to resolve within 1 month is what characterizes a pathological trauma response.

The construct of PTSD was first applied to SA when researchers and clinicians recognized that a set of symptoms observed in SA survivors, then called "rape trauma syndrome" (Burgess & Holstrom, 1974), had similarities to conditions observed in survivors of other forms of trauma, such as combat, then called "shell shock" or "combat fatigue." These syndromes were

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classified as a single condition, called PTSD, in 1980 in DSM-III (American Psychiatric Association, 1980). Aspects of the diagnosis have changed since that time over the various iterations of the DSM, including what specific symptoms are included and how trauma is defined. Generally, the newest iteration of the DSM has narrowed the scope of what is included as a traumatic event and has expanded both the number of symptoms and scope of symptoms to include a wider variety of emotion changes and a stronger emphasis on cognitive changes.

Several critiques have been levied against the diagnostic construct of PTSD. Feminist scholars argue that PTSD divorces survivors' psychological suffering from the patriarchal social context that facilitated the SA (Burstow, 2005; Gilfus, 1999; Wasco, 2003). For example, self-blame cognitions are framed as arising from the trauma itself, which is argued to ignore sociocultural norms that encourage survivors to blame themselves for victimization (Wasco, 2003). In addition, some argue that the accepted construction of PTSD may not account for distinct symptom presentations among marginalized groups (Wasco, 2003). For example, Latinx and Asian Americans appear to be more likely to express distress in somatic terms, and Latinx individuals with PTSD are more likely to report auditory hallucinations than members of other ethnic groups (Pole, Gone, & Kulkarni, 2008). Understanding the cross-cultural relevance of PTSD is thus an important task for future research. Finally, there is debate regarding whether exposure to repeated, prolonged, and severe stressors can produce a variant of PTSD, termed "complex PTSD," that involves symptoms such as somatic, affective, self-perceptual, attentional, and interpersonal dysfunction (Resick et al., 2012). Complex PTSD was considered for inclusion in both the DSM-IV and DSM-5, but empirical studies suggested that there was insufficient evidence to demonstrate that complex PTSD was a distinct phenomenon from PTSD. As a result, complex PTSD is not currently recognized as a diagnostic construct by the DSM.

In addition to critiques of the diagnosis of PTSD, there are critiques regarding the types of events that are considered by the DSM to be potentially traumatic, and therefore have the potential to elicit PTSD (Brewin, Lanius, Novac, Schnyder, & Galea, 2009). Qualifying traumas are termed "criterion A" events in the DSM. In DSM-5, criterion A stipulates that the trauma must involve "exposure to actual or threatened death, serious injury, or sexual violence," and the following examples are provided of sexual violence: "forced sexual penetration, alcohol/drug-facilitated sexual penetration, abusive sexual contact, noncontact sexual abuse, sexual trafficking." Criterion A further specifies that the event must have been experienced either (1) directly, (2) through observation, (3) by learning of its occurrence to a close family member or friend, or (4) through repeated exposure to trauma details (e.g., therapists hearing extensively about client's traumas; APA, 2013, pp. 271). The inclusion of the last two forms of trauma exposure is recent, and some scholars suggest that it has resulted in an overly inclusive PTSD diagnosis that includes individuals with highly distinct symptom profiles (McNally, 2003). Alternatively, many (especially feminist scholars) have asserted that criterion A's definition of trauma is too narrow. They argue that events that do not involve threatened death, serious injury, or overt sexual violence—such as systemic marginalization, street harassment, and sexual harassment (Avina & O'Donohue, 2002; Burstow, 2005; Wasco, 2003)—have traumatic potential, although a meta-analytic review suggests that the impact of such stressors is relatively smaller than criterion A traumas (Larsen & Pacella, 2016). Moreover, feminist scholars argue that the diagnostic criteria focus too narrowly on a single, discrete event and ignore threats to survivors' wellbeing that persist after the assault has ended (e.g., victim blaming; Burstow, 2005).

How Does PTSD Develop After Sexual Assault?

Although there are multiple theories that explain the etiology of PTSD following SA and other traumas, among the most prominent are emotional processing theory (Foa & Kozak,

1986) and social-cognitive theory (Resick & Schnicke, 1992).

Emotional Processing Theory

Foa and Kozak's (1986) emotional processing theory is the theoretical foundation of Prolonged Exposure (Foa & Rothbaum, 1998), an evidence-based PTSD treatment (e.g., Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010) reviewed later in this chapter. This theory builds upon Lang's (1977, 1979) concept of "fear structures," which conceptualize fear as a memory network of information about (1) a feared situation (e.g., the SA); (2) verbal, bodily, and behavioral responses to the situation (e.g., heart rate increases, running away); and (3) the interpreted meaning of the situation and one's responses (e.g., "I am in danger"). Adaptive fear structures accurately reflect reality and act as a "program" to identify and respond to dangerous situations (e.g., escaping an assault).

The fear structures underlying PTSD are thought to be pathological because they are (1) activated by nonthreatening situations and stimuli; (2) elicit verbal, bodily, and behavioral fear responses that are disproportionate to the reality of the situation; and (3) include distorted or inaccurate meanings and interpretations of the situation and one's response to it. For example, after SA, a pathological fear structure may be activated by smells or sounds that remind a survivor of a perpetrator, even though the perpetrator is not there, which result in fear-network responses (e.g., heart rate increases, urges to escape a situation) although the survivor is not in danger. The fear structure may also involve distorted interpretations about the situation (e.g., "it is never safe to be in a bar"), or responses (e.g., "I cannot cope with being reminded of my assault") (Ehlers & Clark, 2000). Pathological fear structures are resistant to change because they lead individuals to avoid trauma-related thoughts and reminders. This avoidance prevents survivors from incorporating new information into the fear structure that could correct the inaccuracies (e.g., learning that one will not be sexually assaulted every time they are in a bar). This theory suggests that SA-related PTSD treatment should involve activating fear structures and integrating corrective and realistic information into it by approaching feared stimuli, as in Prolonged Exposure treatment for PTSD (Foa & Kozak, 1986).

Social-Cognitive Theory

Social-cognitive theory of PTSD emphasizes the content of trauma-related beliefs and their associated emotions (Janoff-Bulman & Frieze, 1983; Resick, Monson, & Chard, 2016). This theory underpins Cognitive Processing Therapy (Resick & Schnicke, 1992), another leading evidence-based treatment for PTSD reviewed later.

Social-cognitive theory emphasizes schemas, which are mental frameworks that allow individuals to organize information into categories to interpret their world and predict their future (Janoff-Bulman, 1989). Experiences like SA can result in information that does not "fit" with existing positive schemas and confirms negative schemas (Resick et al., 2016). For example, a common schema prior to trauma, called the "just world belief," reflects the idea that good things happen to good people, and bad things happen to bad people. These positive schemas are incompatible with new information from a SA (e.g., "a bad thing happened to me, even though I thought I was a good person"). Therefore, survivors may change their understanding of the trauma in unhelpful and inaccurate ways to fit it into their previously held schema (e.g., their belief may change to, "this bad thing happened to me because I am a bad person"). For individuals who held negative schemas, such as "the world is dangerous," the traumatic event may confirm these schemas, holding the prior unhelpful schemas intact (Resick et al., 2016). Individuals also may modify schemas with new information as a result of the assault. This modification, if it accurately incorporates the new learning from the assault without being extreme, facilitates recovery (e.g., their belief may change to, "sometimes bad things happen even to good people"). Other individuals may modify schemas in ways that become

too rigid, inaccurate, and extreme. For example, rather than updating the schema of "bad things happen to bad people" to "bad things sometimes happen even to good people," some trauma survivors may instead believe "I am never safe from bad things even if I do the right thing all the time."

Also embedded in social-cognitive theory is the transaction between trauma-related beliefs and trauma-related emotions. The theory distinguishes between *natural emotions* that occur as a direct consequence of the assault, like fear or anger from an immediate life threat, and *manufactured emotions*, which come from inaccurate, unhelpful, or extreme beliefs (e.g., shame or guilt in response to beliefs such as "I am a bad person"). Natural emotions will decrease over time on their own if experienced and expressed, but manufactured emotions change only when beliefs change. Avoidance of trauma memories, emotions, and reminders prevents opportunities to correct the problematic beliefs and experience natural emotions, so treatment requires stopping avoidance and correcting problematic beliefs (Resick et al., 2016).

How Common and Severe is PTSD After Sexual Assault?

SA is associated with high rates of PTSD relative to other traumatic events (Brewin, Andrews, & Valentine, 2000; Dworkin, Menon, et al., 2017). Recent epidemiological research found that between a quarter and a third of women who have been raped meet full criteria for a current PTSD diagnosis (Kilpatrick, Resnick, Ruggiero, Conoscenti, & McCauley, 2007). Similarly, a recent meta-analysis identified that 26% of survivors of SA met diagnostic criteria for past-year PTSD as compared to 10% of people who had not (Dworkin, in press). SA is associated with more severe PTSD as well. In a comparison of symptom severity, a randomly selected person who had experienced SA had a 69% chance of having more severe PTSD symptoms than a randomly selected person without a SA history (Dworkin, Menon, et al., 2017).

Several prospective studies show that most survivors of SA have PTSD symptoms after SA, although overall symptoms decrease over time. When removing the criterion that symptoms endure for at least 1 month, 67–94% of SA survivors evidenced sufficient symptoms to warrant a PTSD diagnosis within the first month following the rape (Frazier, 2000; Frazier, Conlon, & Glaser, 2001; Gutner, Rizvi, Monson, & Resick, 2006; Rothbaum et al., 1992; Steenkamp et al., 2012). At 3 months post-SA, 42–78% met criteria (Darves-Bornoz, Degiovanni, & Gaillard, 1998; Frazier, 2000; Gutner et al., 2006; Rothbaum et al., 1992; Steenkamp et al., 2012; Ulirsch et al., 2013), and 48–58% met criteria for PTSD by 1 year (Darves-Bornoz et al., 1998; Frazier, 2000; Frazier et al., 2001).

What Factors Increase the Likelihood of Developing PTSD After Sexual Assault?

Substantial research has examined characteristics of assaults, SA survivors, and the contexts in which they recover to understand who is most likely to develop PTSD.

Assault Characteristics

A variety of assault characteristics have been associated with the likelihood of developing PTSD and other disorders, including whether assaults involve physical injury (Bownes, O'Gorman, & Sayers, 1991; Dworkin, Menon, et al., 2017; Möller, Bäckström, Söndergaard, & Helström, 2014), force or physical violence (Brown, Testa, & Messman-Moore, 2009; Peter-Hagene & Ullman, 2015; Ullman, Townsend, Filipas, & Starzynski, 2007; Zinzow et al., 2010), a perpetrator who is a stranger (Bownes et al., 1991; Ullman, Townsend, Starzynski, & Long, 2006) or a partner (Temple, Weston, Rodriguez, & Marshall, 2007; Ullman et al., 2006), more than one perpetrator (Möller et al., 2014), distress or perceived life threat (Elklit & Christiansen, 2013; Kaysen, Rosen, Bowman, & Resick, 2010;

Ullman, Filipas, Townsend, & Starzynski, 2007), having a "freeze response" (Bovin, Jager-Hyman, Gold, Marx, & Sloan, 2008; Rizvi, Kaysen, Gutner, Griffin, & Resick, 2008; Rocha-Rego et al., 2009), and weapon use (Bownes et al., 1991; Dworkin, Menon, et al., 2017). SAs involving different types of tactics, like force, coercion, and incapacitation, do not appear to differ substantially in terms of their risk for PTSD. Varying the definition of SA to include coerced and incapacitated SA as well as completed forced SA did not significantly alter the observed relationship with psychopathology when comparing sexually assaulted to unassaulted samples in a meta-analysis (Dworkin, Menon, et al., 2017). Including attempted SAs along with completed SAs decreased the effect size of the relationship with psychopathology in this meta-analysis, but it remained statistically significant, suggesting that noncompleted SA also can lead to psychopathology. As the authors noted, conclusions that can be drawn are somewhat limited as the research is based solely on study definitions of SA. Still, these findings are compelling in suggesting that SA is highly predictive of PTSD and other forms of psychopathology, regardless of the tactics used. Of note, individual studies that have looked at severity of assault as a predictor of PTSD generally show that higher severity SAs are related to increased PTSD (Pegram & Abbey, 2016; Peter-Hagene & Ullman, 2015; Resnick et al., 1993; Ullman & Filipas, 2001). However, one study demonstrated that alcohol-involved assaults evidenced as strong a relationship with PTSD as forcible assaults over a longer-term follow-up (Peter-Hagene & Ullman, 2015), which is consistent with an epidemiological study that found PTSD associated with both forcible and incapacitated SA (Zinzow et al., 2010). Thus, the relationship between assault severity and PTSD is likely complicated by varied definitions of SA and associated individual and situational factors.

Characteristics of Survivors

Demographic factors have been explored in relation to risk of developing PTSD after SA. Across all trauma types, women are more likely to develop PTSD than men, but there do not appear to be gender differences after SA specifically. Similarly, although racial differences have been found for PTSD prevalence (Roberts, Gilman, Breslau, Breslau, & Koenen, 2011), reviews have not found racial differences in PTSD following SA (Campbell, Dworkin, & Cabral, 2009; Dworkin, Menon, et al., 2017). It is possible that the higher likelihood of developing PTSD after SA versus other forms of trauma reduces observed racial differences. With regard to sexual orientation, higher rates of PTSD have been observed among sexual minority individuals (Balsam, Rothblum, & Beauchaine, 2005; Rothman, Exner, & Baughman, 2011), but no studies to our knowledge have looked at sexual orientation as a predictor of PTSD after SA specifically. Finally, younger age predicts increased PTSD severity in assault victims, as does a history of childhood sexual abuse (CSA; Ullman, Filipas, et al., 2007; Ullman, Najdowski, & Filipas, 2009). One large study of 1769 women in the US showed increased PTSD prevalence in women assaulted before age 18 (35.3%) compared to those assaulted after 18 (30.2%; Masho & Ahmed, 2007). Experiencing multiple SAs over the lifespan also predicts PTSD severity (Ullman, 2016). Consistent with these findings, in two large meta-analyses, experiencing trauma at a younger age was a significant predictor of increased likelihood of developing PTSD (Brewin et al., 2000; Ozer, Best, Lipsey, & Weiss, 2003).

Genetic and biological factors have been proposed as determinants for post-SA adjustment including PTSD, although to our knowledge, no studies have looked specifically at genetic determinants of PTSD following SA. Results of twin and large-scale genome-wide association studies do suggest that heritability may play a significant role in the development of PTSD following trauma, particularly for women (see Yehuda et al., 2015 for a review). The hypothalamic pituitary adrenal axis and sympathetic nervous system are implicated in the stress response that occurs following trauma, and thus stress hormones (e.g., cortisol) may be affected by SA and

influence the development of PTSD (Zoladz & Diamond, 2013). Studies on cortisol levels following SA show mixed findings in relationship to predicting PTSD, with some studies finding elevated cortisol levels to be related to PTSD (Resnick, Yehuda, & Acierno, 1997) and other studies finding cortisol levels to be unrelated to PTSD in those exposed to SA (Resnick, Yehuda, Pitman, & Foy, 1995). A recent review proposed the posttrauma administration of pharmacological interventions that target systems related to cortisol (e.g., glucocorticoid receptor agonists, oxytocin) to prevent PTSD (van Zuiden, Kavelaars, Geuze, Olff, & Heijnen, 2013). Others point out that ovarian hormones may influence emotional memory processing and, potentially, partially account for gender differences in PTSD (Zoladz & Diamond, 2013).

Psychophysiological indicators have also been explored as possible indicators of who is likely to develop PTSD following trauma. No psychophysiological studies to our knowledge have focused specifically on participants exposed to SA, but two studies of survivors of primarily physical assaults show that this may be an important area of exploration. Individuals with higher resting heart rate 2 weeks after assault were more likely to develop PTSD at 6 months post assault, although the observed effect size was small (Kleim, Ehlers, & Glucksman, 2007). Women with increased heart rate reactivity to assault imagery at 2 weeks post-assault were significantly more likely to develop PTSD compared to men and to women without high heart rate reactivity (Kleim, Wilhelm, Glucksman, & Ehlers, 2010). This indicates that physiological reactivity to trauma reminders—which is a symptom of PTSD if it endures for more than 1 month—may be an early indicator of the likelihood that trauma symptoms will naturally remit. A large meta-analysis on psychophysiology and PTSD showed that presence of PTSD following trauma exposure more broadly was associated with diverse psychophysiological reactions, including elevated heart rate and skin conductance at rest and in response to startle probe tasks and trauma cues (Pole, 2007).

The types of coping strategies used by survivors appear to alter the risk of PTSD following SA. Maladaptive coping strategies, typically defined as those that involve avoidance instead of approach strategies (e.g., substance use, isolation, avoidance of thoughts or feelings related to the SA, avoidance of reminders of the SA, withdrawal from social support) predict higher PTSD symptoms in SA survivors (Frazier, Mortensen, & Steward, 2005; Gutner et al., 2006; Read et al., 2013; Ullman, Relyea, Peter-Hagene, & Vasquez, 2013; Ullman, Townsend, et al., 2007). In contrast, adaptive coping strategies, such as problem-focused coping and seeking social support, have been associated with lower PTSD symptoms in survivors of SA (Frazier et al., 2005; Gutner et al., 2006; Valentiner, Foa, Riggs, & Gershuny, 1996). The context in which a coping strategy is employed may be important in determining its relationship with PTSD. For example, among college women who experienced SA, the positive effects of social support on PTSD symptoms were lower for women who were using substances to cope (Dworkin, Ojalehto, Bedard-Gilligan, Cadigan, & Kaysen, 2018). This suggests that coping strategies should not be considered in isolation.

Contextual Factors

SA occurs within a multilevel social context that influences recovery (Campbell et al., 2009). On a societal level, it is clear that there is significant stigma associated with SA, as evidenced by the pervasiveness of rape myths (e.g., the beliefs that victims are to blame for the assault and that victims lie about being sexually assaulted; Deitz, Williams, Rife, & Cantrell, 2015; Edwards, Turchik, Dardis, Reynolds, & Gidycz, 2011) and victim blaming in response to SA disclosure (Ullman, 2010). These social-context-related factors may increase self-blame and ultimately affect survivors' post-assault mental health, including PTSD (Kennedy & Prock, 2018). Indeed, most SA survivors (80% or more) choose to disclose to at least one person (Ahrens, Campbell, Ternier-Thames, Wasco, & Sefl, 2007)

and often receive negative social reactions (e.g., victim blaming, taking control of survivors' decision-making, disbelief), which are associated with psychopathology (Dworkin, Brill, & Ullman, 2019; Littleton, 2010; Ullman & Peter-Hagene, 2014; Ullman, Starzynski, Long, Mason, & Long, 2008; Ullman, Townsend, et al., 2007). Preliminary evidence also suggests that SA survivors in more stigmatizing contexts, regardless of whether they disclose, have worse mental health (Dworkin, Sessarego, Pittenger, Edwards, & Banyard, 2017). The detrimental effects of stigma on survivors' current distress and trajectories of recovery are robust and warrant further research into effective interventions following SA.

What Are the Best Clinical Practices for Addressing Sexual Assault-Related PTSD?

In the past 30 years, substantial research has focused on developing effective strategies to assess, treat, and prevent the development of PTSD. We review this work next.

Assessment of Trauma Exposure and Psychopathology

It is important to assess trauma exposure broadly, rather than SA history alone, in order to create a comprehensive picture of the client's trauma history. One challenge is selecting between longer, more thorough measures of SA that use behaviorally specific definitions of traumatic events, such as the Sexual Experiences Survey (Koss et al., 2007), which are thought to more accurately capture SA exposure as compared to briefer measures which use only terms such as "rape" (Cook, Gidycz, Koss, & Murphy, 2011). There are several commonly used brief measures of broader trauma exposure, including the trauma history portion of the Posttraumatic Diagnostic Scale (Foa, Cashman, Jaycox, & Perry, 1997) and the Life Events Checklist (Weathers et al., 2013).

It is also important to systematically assess PTSD through the use of validated psychological assessments (Briere & Jordan, 2004). This can include clinical interviews like the Clinician-Administered PTSD Scale (Blake et al., 1995), or self-report measures like the Primary Care-PTSD Screen (Prins et al., 2003) or the Posttraumatic Checklist for DSM-5 (Weathers et al., 2013). These PTSD measures must be asked in reference to a specific trauma. It is important to not solely assess PTSD, though, as SA can be associated with other psychological symptoms such as depression and anxiety (Dworkin, Menon, et al., 2017).

Treating PTSD Related to Sexual Assault

The treatment of PTSD has been significantly influenced by the adoption and implementation of evidence-based practices, defined by the American Psychological Association (APA) as, "the integration of the best available research with clinical expertise in the context of patient characteristics, culture, and preferences" (APA Presidential Task Force on Evidence-Based Practice, 2006, p. 273). Based on substantial research, only cognitive behavioral therapies (CBTs) with an explicit trauma focus received strong recommendations as evidence-based practices for PTSD by the APA (APA, 2017), meaning that the treatments have been rigorously tested by independent investigators with a preponderance of studies agreeing that the treatment is efficacious (Chambless & Hollon, 1998). Broadly speaking, CBTs include cognitive components like *cognitive restructuring* (i.e., identifying and changing rigid or inaccurate beliefs) and/or behavioral components like *exposure* (i.e., systematically confronting feared and/or avoided stimuli until fear is extinguished). All major clinical practice guidelines currently recommend trauma-focused CBTs as a first-line treatment for PTSD (see Forbes et al., 2010 for a review of guidelines). A report by the Institute of Medicine (IOM), which is considered the most rigorous of the current published treatment guidelines for PTSD (Forbes et al., 2010), concluded that only exposure therapies and Cognitive Processing

Therapy specifically met the designation of "efficacious," and that other psychotherapies (e.g., eye movement desensitization and reprocessing) and pharmacotherapies (e.g., SSRIs, benzodiazapines) had insufficient evidence to determine efficacy (IOM, 2007). Meta-analyses of randomized controlled trials assessing PTSD treatments found large effect sizes for improvements in PTSD symptoms in CBTs relative to control groups, and medium effects for pharmacotherapy (Bradley, Greene, Russ, Dutra, & Westen, 2005; Watts et al., 2013). Of individuals receiving exposure therapy, 90% improved more than individuals on a waitlist, and 79% improved more than individuals receiving supportive counseling. Of those receiving combined exposure and cognitive restructuring, 93% improved more than individuals receiving no treatment, and 84% improved more than those receiving supportive counseling. This indicates that it is especially important to ensure that SA survivors with PTSD have access to trauma-focused CBTs as treatment options. Next, we describe two commonly used trauma-focused CBT treatments for PTSD—Cognitive Processing Therapy and Prolonged Exposure—both of which were developed for use with female rape victims and have an extensive evidence base for their efficacy in treating PTSD. Then, we review other treatment options.

Prolonged Exposure

Prolonged Exposure is a well-validated treatment for PTSD (Foa, Rothbaum, Riggs, & Murdock, 1991). It utilizes two forms of exposure—in vivo exposure and imaginal exposure—over 8 to 15 sessions. Through *in vivo exposure*, clients construct a list of feared or avoided, but objectively safe, stimuli that are gradually approached throughout the course of treatment. For example, a SA survivor who was assaulted at a party may avoid all parties thereafter due to the expectation that parties are dangerous or reminders of the original SA cued by the party; a natural in vivo exposure for such a client would be to attend a safe party. By approaching these situations, clients learn that the feared consequence is unlikely to happen and that they can tolerate the distress associated with the situation. Clients usually stay in exposure situations until distress decreases by at least half, or for an agreed-upon amount of time (e.g., 30 min), to allow time for new learning (i.e., that the feared situation is not actually dangerous) to occur. Through *imaginal exposure*, clients repeatedly recount the trauma memory aloud in session to the therapist for 30–45 min. Following imaginal exposure, therapists guide clients in processing their emotions and cognitions related to the traumatic event in order to promote new learning, ability to tolerate distress, and extinction of emotional responses. In a meta-analysis, Prolonged Exposure was highly effective in reducing PTSD; 86% of those receiving Prolonged Exposure experienced greater reductions in PTSD symptoms than those in a control condition (Powers et al., 2010). Prolonged Exposure was equally effective in treating SA-related PTSD as compared to PTSD due to other traumas, and treatment effects remained in follow-up assessments up to 10 years later (Resick, Williams, Suvak, Monson, & Gradus, 2012).

Cognitive Processing Therapy

Cognitive Processing Therapy is a well-validated treatment for PTSD following many kinds of trauma, including SA (Resick et al., 2016; Resick & Schnicke, 1992). It is typically delivered in 12 sessions, and involves cognitive restructuring (i.e., identifying and modifying maladaptive thoughts to develop more balanced, flexible, and adaptive beliefs) and emotional processing of trauma-related content. Initially, sessions focus on cognitive restructuring of beliefs about the traumatic event, such as self-blame or hindsight bias (e.g., "It was all my fault," "I should have prevented the assault"). Cognitive restructuring then targets beliefs about the self and others that are the result of a traumatic event in five key areas: safety (e.g., "the world is dangerous"), trust (e.g., "I can never trust anyone"), power (e.g., "I am helpless"), esteem (e.g., "all people are evil"), and intimacy (e.g., "I can't get close to

other people"). Randomized clinical trials conducted with different trauma-exposed populations, including survivors of SA, show that Cognitive Processing Therapy is effective in treating PTSD and depression (Bass et al., 2013; Chard, 2005; Resick, Nishith, Weaver, Astin, & Feuer, 2002; Surís, Link-Malcolm, Chard, Ahn, & North, 2013). In these trials, effects largely endured 5 to 10 years later (Resick, Williams, et al., 2012).

Other Psychological Treatments

Several other treatments have some evidence for their effectiveness in treating PTSD. First, eye-movement desensitization and reprocessing therapy (EMDR) is a common treatment for PTSD that shows similar effects to trauma-focused CBT in research studies (Watts et al., 2013), although the quality of evidence is somewhat less strong than for CBT (IOM, 2007). EMDR involves in-session recall of the traumatic memory (as in imaginal exposure) while focusing on an external stimulus (commonly, therapist-directed lateral eye movements). The focus on an external stimulus that defines EMDR is considered by its creators to be an active ingredient of the treatment, but the absence of theoretical or empirical support for the additive benefit of this portion of the treatment (Seidler & Wagner, 2006) has led to criticisms that EMDR is a variant of exposure therapy (Rosen & Davison, 2003). Moreover, concerns have been raised about treatment fidelity in practice (Rosen, 1999), given the emphasis placed on eye movements as the active ingredient rather than exposure. As a result, the APA gave EMDR a second-strength rating, and the Australian clinical practice guidelines recommended EMDR only when in vivo exposure is additionally included (Ironson, Freund, Strauss, & Williams, 2002). Second, Narrative Exposure Therapy (NET) is an exposure-based treatment adapted from Prolonged Exposure therapy and testimony therapy that has a growing evidence base supporting its efficacy (see Robjant & Fazel, 2010 for a review). In NET, the client constructs a narrative of their life from childhood through the present moment, including traumatic events. The therapist then goes back and processes the details of those traumatic events as exposure. NET is proposed to work through habituation and through creating an integrated autobiographic memory. The APA has given NET a second strength rating, whereas other practice guidelines, like the Australian practice guidelines, group NET in with other trauma-focused CBTs. There are currently no trials of NET specifically focusing on SA survivors. Other treatments, such as Acceptance and Commitment Therapy (ACT) and mindfulness-based therapies, show initial promise in reducing PTSD in case studies and small, pilot trials (Kearney, McDermott, Malte, Martinez, & Simpson, 2013; Orsillo & Batten, 2005) but have not yet been tested in large-scale randomized controlled trials.

Preventing PTSD Related to Sexual Assault

Increasingly, efforts have been made to intervene in the aftermath of trauma exposure to prevent the development of PTSD. One type of early intervention, critical incident stress debriefing (Mitchell, 1983), involves a single psychological debriefing session for a group of trauma survivors and is typically provided 1–10 days after trauma exposure. Despite its widespread use, several meta-analyses have found that critical incident stress debriefing and other forms of psychological debriefing are ineffective in preventing PTSD and may even increase the risk of developing PTSD, perhaps by interfering with a survivor's natural recovery process (Rose, Bisson, Churchill, & Wessely, 2002; van Emmerik, Kamphuis, Hulsbosch, & Emmelkamp, 2002). Similarly, the positive-psychology-based Comprehensive Soldier Fitness program (Cornum, Matthews, & Seligman, 2011), which provides soldiers with resiliency training to buffer against the negative effects of trauma exposure, does not have empirical evidence for its efficacy (Eidelson & Soldz, 2012). However, other interventions have had more promising effects, including with SA survivors. In a review,

a brief form of CBT reduced PTSD severity among individuals with acute stress disorder (Forneris et al., 2013). Similarly, a small randomized controlled trial of a video intervention that taught CBT-based strategies to SA survivors receiving a forensic medical exam was associated with significantly lower PTSD symptoms compared to treatment as usual among women who had a prior SA history (Resnick et al., 2007). A qualitative review on reducing PTSD after SA via early intervention found that perceptions of the helpfulness of early contact with responders was associated with lower PTSD severity (Dworkin & Schumacher, 2018).

Future Directions

There are several gaps in extant literature regarding the impact of SA on PTSD and how PTSD can be most effectively treated in this population. First, there is a need for longitudinal research with at-risk populations (e.g., military recruits) to understand the effects of risk factors measured before SA (e.g., personality traits, mental disorders, other traumatic life experiences) on the likelihood of developing PTSD and its trajectory. Such research could improve the ability to predict risk for PTSD. Second, more focus is needed on under-researched populations. There is a tremendous need for research on the experiences of male survivors and for members of historically marginalized groups (e.g., sexual minority populations, people of color). This work is critical to understand how the unique processes that affect these groups (e.g., stigma) interact with PTSD risk and recovery, and to inform tailored treatments for them. Third, to reduce the degree to which survivors of SA develop PTSD, early interventions delivered soon after SA should be tested and refined. Finally, given that characteristics of survivors' social contexts (e.g., social reactions to disclosure) may affect PTSD symptoms, it is important to continue to search for contextual variables that could impact symptoms.

There are also several important priorities for intervention efforts and policy to support these efforts. Given that cognitive behavioral therapies with a focus on the traumatic event have strong research evidence regarding their ability to treat PTSD and are able to do so in a short-term (e.g., 12-week) manner, it is important that these interventions be made broadly available to survivors as a first-line option. However, the vast majority of therapists and rape crisis centers do not offer these treatments, and instead commonly offer treatments of unknown efficacy (Foa, Gillihan, & Bryant, 2013), which limits survivor choice and autonomy. To address this gap between research and practice, it is critical that service providers be trained in delivering these treatments. Additionally, service provider misconceptions about these treatments that get in the way of offering such treatments should be addressed (Cook, Schnurr, & Foa, 2004; Shafran et al., 2009). From a policy perspective, increasing funding for dissemination and implementation of these treatments, including in non-US settings, is important. It is also apparent that survivors face many barriers in accessing effective treatments, including lack of access to therapists, financial barriers, and lack of knowledge about effective treatment options (Gunter & Whittal, 2010). Making treatments available via technology (e.g., apps) and remote delivery (e.g., telehealth), and addressing policy issues such as reimbursement for telepsychology and increasing health literacy, could increase access to care. Healthcare reform is also an important part of removing financial barriers to treatment access. Further, interventions that prevent PTSD are needed. PTSD prevention efforts should focus on reducing changeable risk factors, such as maladaptive coping strategies, and building protective factors. Immutable risk factors (e.g., survivor characteristics) could be used to identify individuals in need of early intervention. Efforts should also be made to improve the degree to which contexts promote recovery. This could include efforts to improve social responses to SA, by, for example, training likely disclosure recipients (e.g., college students, police officers) in avoiding negative social reactions. More broadly, social movements that aim to reduce societal stigma are a critical component of improving the climate for SA survivors.

Conclusion

It is clear that PTSD is common among SA survivors. Although there are many known risk factors for PTSD, many have yet to be identified, and intervention efforts have largely focused on treating PTSD after it has already developed rather than reducing risk factors directly. Further, although effective treatments exist, they are not widely available. As a result, there is much work left to be done to reduce the degree to which survivors of SA suffer from PTSD.

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