Prevention of PTSD

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Exposure to extreme stressors is unfortunately very common. Lifetime exposure to potentially traumatic events (PTEs) ranges from approximately 50–90% in samples of adults in stable, economically developed Western nations (e.g., Breslau, Kessler, Chilcoat, et al., 1998; Creamer, Burgess, & McFarlane, 2001; Kessler et al., 1995), and approaches 100% in samples from war-torn regions (e.g., Goldstein, Wampler, & Wise, 1995; Mollica, Poole, & Tor, 1998). Across samples, men appear to be at generally higher risk than women for exposure to physical violence, injuries, disasters, and combat, whereas women appear to be at generally higher risk for exposure to sexual violence. Although most people do not develop mental disorders or problems functioning in the wake of exposure to PTEs (Brewin, Andrews, & Valentine, 2000; Nash et al., 2015), a sizeable minority will experience some degree of posttraumatic stress symptoms (e.g., Norris, 1992), and about 10–20% develop posttraumatic stress disorder (PTSD) (Breslau et al., 1998; Kessler et al., 1995). PTSD is associated with markedly impaired social and occupational functioning (e.g., Kulka, Schlenger, & Fairbank, 1990; Savoca & Rosenheck, 2000) and reduced quality of life (e.g., Malik, Connor, & Sutherland, 1999). Once acquired, the disorder often runs a chronic course (e.g., Prigerson, Maciejewski, & Rosenheck, 2001), and remission, when it does occur, may take many years or decades (Chapman et al., 2012).

PTSD thus imposes a substantial burden on individuals, families, communities, and society. Most people do not seek treatment, and if they do, they do not receive evidence-based care. In addition, the effectiveness of first-line treatments for PTSD is limited, particularly in populations exposed to malicious violence or other complex interpersonal and repeated traumas, such as combat (e.g., Steenkamp & Litz, 2013). These realities underscore the importance of

preventing PTSD. PTSD and other mental and behavioral health problems following from exposure to PTEs could be prevented by reducing the risk for exposure to motor vehicle accidents, child abuse, rape, and war, and so forth. Although allied mental and physical health professionals may be able to help effect such large-scale social changes by working in alliance with legislators, law enforcement, and other key collaborators (e.g., bystander interventions to prevent college sexual assaults), we do not focus on such efforts in this chapter. Instead, we review the literature on factors associated with increased risk for the development of PTSD and preventive interventions designed to promote resilience given the reality of PTE exposure.

We begin with an overview of research on moderators of risk for PTSD, as this information has the potential to help researchers and clinicians to identify who needs prevention or in which contexts prevention should be considered. We then introduce a conceptual framework for organizing and understanding different types of preventive interventions. Next, we review examples of various types of interventions and critically appraise the available evidence of their effectiveness. Finally, we highlight limitations of the existing research and opportunities for advancing the field's understanding of how best to prevent PTSD.

Risk Factors

Risk factors are characteristics of a person, the person's environment, or a PTE that serve as harbingers of or pathways to an exacerbated or prolonged negative response to the PTE. Research on these factors has the potential to identify those individuals who are at greatest risk of developing PTSD in response to PTEs, and may also guide the design of preventive interventions targeting these individuals (King et al, 2012). We organize our review of moderators of risk for PTSD according to whether they originate in the periods before (pre-trauma), during (peri-trauma), or after (post-trauma) the PTE.

Pre-Trauma Factors

Demographic factors. Brewin and colleagues (2000) conducted a meta-analysis of 77 studies of adults to examine risk factors for the development of PTSD. No gender difference emerged for the risk of developing PTSD in military samples, but women in the civilian samples were at greater risk for PTSD than men to a minor degree (weighted effect size difference of r = .13). Surprisingly, lower SES was only slightly associated with risk for PTSD in civilian samples (r = .14), as was younger age in military samples (r = .06). A few studies have investigated whether gender directly confers a heightened risk for PTSD or whether it is a proxy variable for risk (e.g., civilian women's greater PTSD risk may be due to a greater likelihood of exposure to sexual assault when compared to civilian men). These studies found that PTE exposure only partly explained the effect of gender on risk for PTSD. Specifically, women were still more likely than men to develop PTSD even after controlling for trauma type and number of PTE exposures (Olff et al., 2007; Stein et al., 2000).

Cognitive and personality factors. Numerous studies have documented associations between lower cognitive functioning and PTSD (Bomyea et al., 2012). For example, in a longitudinal study of firefighters, difficulty retrieving specific autobiographical memories in response to positive cues (at the time of training) was associated with greater PTSD symptoms four years later (Bryant et al., 2007). In a sample of Vietnam combat veterans, those with lower intelligence (measured pre-deployment) showed a higher rate of PTSD post-service than veterans with higher pre-deployment intelligence (Macklin et al., 1998). Although lower intelligence was confounded with greater combat exposure in this sample, these findings were replicated in a longitudinal study of a New Zealand cohort followed from birth to adulthood (Koenen et al., 2007). Individuals who had a lower level of intelligence measured at age five, as well as a

difficult temperament assessed at ages three and five, were more likely to report PTSD symptoms at age 26, given exposure to at least one PTE in the interim. In a large meta-analysis, lower intelligence and level of education measured prospectively as well as retrospectively were associated with increased risk of PTSD symptoms (r = .18 and.10, respectively; Brewin et al., 2000).

An association between personality and PTSD has also been documented in numerous studies. For instance, in a large study of veterans who served on a peacekeeping mission, negative, dissatisfied, and hostile attitudes toward others and life in general, measured prior to deployment, were modestly correlated with PTSD symptoms post-deployment (Bramsen et al., 2000). Orcutt et al. (2014) found that people who reported habitually avoiding uncomfortable emotions, thoughts, and bodily sensations prior to a shooting rampage on a college campus were more likely to develop chronic PTSD following this event.

Personal history factors. A large body of research has demonstrated that prior exposure to PTEs is a risk factor for developing PTSD in response to new-onset PTEs (Brewin et al., 2000; King et al., 2012; Ozer et al., 2003). A meta-analysis of 23 studies found an average effect size of r = 0.17 for history of prior PTE exposure, with greater risk conferred by a history of noncombat interpersonal violence such as sexual assault (r = .27) compared to a history of combat-related (r = .18) or accident-related (r = .12) PTEs (Ozer et al., 2003). The total number of prior PTEs has also been found to be positively associated with severity of PTSD symptoms in Vietnam combat veterans, sexual assault survivors, and motor vehicle accident victims, suggesting a dose–response relationship between PTE exposure history and PTSD (King et al., 2012). History of childhood PTEs and history of adult PTEs have been found to pose equivalent risk for the development of PTSD in response to further PTE exposure (Ozer et al., 2003).

A history of family psychiatric problems and dysfunction has also been reported to be associated with PTSD. Ozer et al. (2003) found that individuals with a family history of psychopathology reported greater PTSD symptoms and had more PTSD diagnoses than those without such a history (r = .17). The strength of this factor's association with PTSD varied by trauma type; a family history of psychopathology was more strongly related to PTSD in individuals whose principal PTE exposure was noncombat interpersonal violence as opposed to combat or an accident-related event. Similarly, direct indicators of dysfunction in the family of origin, such as history of child abuse and family instability, have also been found to confer risk for PTSD (Brewin et al., 2000).

Psychopathology prior to PTE exposure has been identified as a consistent risk factor for PTSD in numerous studies. In a systematic qualitative review of pre-trauma risk factors for PTSD, prior psychopathology was a significant predictor of PTSD in 19 out of 23 studies (DiGangi et al, 2013). For example, in an analysis utilizing data from the National Comorbidity Survey, after adjusting for trauma type, only pre-exposure affective disorders in women and pre-exposure anxiety disorders in men were significant risk factors for developing PTSD, suggesting that a personal history of psychopathology may be an especially important pre-trauma predictor of adjustment following exposure to a PTE (Bromet et al., 1998).

Many studies of the correlation between personal history factors and PTSD have been retrospective. Therefore, it is possible that effect size estimates have been distorted by biased retrospective recall of prior exposure, family history of psychiatric problems, and prior psychopathology. However, in Brewin et al.'s (2000) meta-analysis, the effect sizes for prior PTE exposure and family psychiatric history were equivalent for prospective and retrospective studies.

Peri-Trauma Factors

Characteristics of the PTE itself, as well as psychological factors surrounding the experience of the event, are among the strongest predictors of developing PTSD (Ozer et al., 2003). Many studies have shown an association between perceived life threat and greater PTSD symptoms, with a stronger association found in military samples than civilian (r = .26 versus .18; Brewin et al., 2000). In addition, individuals who reported experiencing more negative emotional responses, such as fear, helplessness, horror, guilt, and shame, during or immediately after the PTE have been shown to develop significantly greater symptoms and rates of PTSD (r = .26; Ozer et al., 2003). Moreover, dissociative symptoms (e.g., feeling as if you were outside of yourself, watching the experience unfold) during or immediately after the PTE have been associated with greater PTSD symptoms and rates (r = .35; Ozer et al.). For example, among individuals who had been admitted to a hospital due to physical injuries from a PTE, those who reported higher levels of peritraumatic dissociation at one week after the PTE had higher levels of PTSD symptoms at six months post-trauma (Shalev et al., 1996).

In a qualitative review of the literature, individuals' styles of coping with difficulties were associated with risk for developing PTSD after a PTE (DiGangi et al., 2013). For example, in two separate longitudinal studies of Gulf War veterans assessed at two different post-deployment time points, those individuals who reported greater use of avoidant coping while in combat reported more severe PTSD symptoms (Benotsch et al., 2000; Sharkansky et al., 2000). More recently, in a study examining trajectories of PTSD symptoms among highly combat-exposed Marines, Nash et al. (2015) found that avoidant coping and peritraumatic dissociation were the strongest predictors of membership in the trajectories of new-onset PTSD and preexisting PTSD relative to a low-symptom/stable trajectory.

Post-Trauma Factors

Lack of social support is the strongest risk factor for PTSD. Supports can provide empathy, can help reestablish hope, meaning, and purpose, can unburden thoughts and feelings about the PTE, and can affect appraisals of the meaning and implication of the event. Social isolation and poor social support enhance risk considerably (Tremblay et al., 1999). In one meta-analysis, lack of social support showed a stronger association with PTSD (average weighted effect size r = .40) than all other risk factors and was an especially strong predictor of PTSD development in military samples (Brewin et al., 2000). Similarly, Ozer et al. (2003) found that perceived social support following a PTE was significantly negatively correlated with PTSD (r = -.28) and that this relationship was especially strong in cases of combat compared to noncombat violence.

Experiencing additional life stressors after exposure to a PTE has also been found to increase risk for PTSD in studies utilizing retrospective and prospective designs (r = .32; Brewin et al., 2000). For example, in a sample of Vietnam veterans, PTSD symptoms were more severe among those who reported greater post-war life stressors, such as legal or financial difficulties, death or illness of a relative, motor vehicle accidents, physical assaults, and marital disruptions (King et al., 1998). However, this relationship between additional life stressors and PTSD was mediated by social support. Veterans who reported a greater number of post-war life stressors also reported less emotional and instrument support, and those veterans reporting less emotional and instrument support reported a higher level of PTSD symptom severity.

Implications for PTSD Prevention

In sum, the associations between PTSD and pre-trauma risk factors such as gender, cognitive functioning, negative global attitudes, intolerance of emotional discomfort, family

history of psychopathology, and prior PTE exposure have typically been reported as statistically significant but small. In contrast, medium effect sizes have consistently been reported for the correlation between PTSD and the peri-traumatic factors of perceived life threat, intensity of emotional response to the event, dissociation, and avoidant coping style, as well as the post-traumatic factor of low social support.

It would appear that prevention efforts should be considered for individuals who report particularly intense PTEs that evoke extreme emotional responses, who possess an avoidant coping style, and who have little social support. These findings from risk factor research also suggest that preventive interventions should help people manage additional stressor burdens after exposure to PTEs and promote the development of social support resources and an active approach to coping.

Prevention versus Treatment of PTSD

In a comprehensive report aimed at facilitating the development and dissemination of interventions to combat mental disorders, the Institute of Medicine (1994) delineated three major categories of mental health interventions, falling along a *continuum of care*. These included prevention, treatment, and maintenance interventions (see Figure 1). While all three intervention strategies share the same ultimate goal of reducing distress and functional impairment resulting from mental disorders, preventive interventions are distinguished by the fact that they do not specifically target individuals who meet or have met the diagnostic threshold for a given disorder (such as PTSD). Treatment and maintenance interventions are aimed only at people who have or have had a particular recognizable mental disorder, even if these interventions could reasonably be construed as serving some "preventive" functions—for example, PTSD treatment may help to prevent the development of further co-occurring disorders or disability (e.g., substance use

disorders), and maintenance may help to prevent long-term relapse and recurrence in individuals who have successfully completed the acute phase of treatment.

Preventive interventions, in contrast to treatment and maintenance, have as their primary goal reducing the risk of developing a new-onset case of a disorder. The Institute of Medicine (1994) report adapted a conceptual framework from Gordon (1983), developed for general health and disease, to divide preventive mental health interventions into three baskets. We organize our review of preventive interventions for PTSD according to this scheme.

First, *universal* preventive interventions target the general public or some other large, general population that is not thought to be at particularly elevated risk for the disorder that the intervention is designed to help prevent. Immunizations given routinely to all children are a good example of a universal preventive intervention. Because universal interventions typically target a very large number of people, they need to be inexpensive and impose negligible risks to achieve a favorable cost-benefit profile.

Selective preventive interventions target groups or individuals who, by virtue of possessing certain risk factors (e.g., social, environmental, or biological), are thought to be more likely than average to develop a disorder in the immediate or longer-term future. As an example of a selective preventive intervention, the Institute of Medicine (1994) report cites an intensive preschool program targeting only children from low-income neighborhoods.

Finally, *indicated* preventive interventions target individuals who show specific symptoms or signs suggesting that they are at risk of developing the full disorder that the intervention is aimed at preventing. For example, an intervention targeting individuals who report high levels of alcohol consumption at a primary care check-up would be considered indicated prevention. Because the number of individuals targeted by selective, and especially

indicated, preventive interventions is more limited than in universal interventions, these types of intervention may be more resource-intensive while still maintaining a favorable ratio of costs to benefits.

PTSD Preventive Interventions

Universal Prevention

One large-scale universal prevention program has been implemented and its results published. The U.S. Army's Comprehensive Soldier Fitness Program (CSFP) was developed by psychologists to promote wellness and psychological health and to prevent PTSD in soldiers. The CSFP intervention includes computer-administered elements of cognitive therapy borrowed from positive psychology, additional didactic materials, and in-person trainings for leaders so that they can reinforce the information with their troops at a later date. Drawing on analyses suggesting that exposure to the CSFP intervention was associated with lower levels of depression, negative affect, and loneliness, Lester and colleagues (2011) concluded that the program improves the resilience and health of soldiers. However, because these findings were not peer-reviewed and the reported effect sizes were very small, no strong conclusions about the effectiveness of CSFP are warranted (Steenkamp et al., 2013).

Selective Prevention

The impact of various early interventions for individuals who have been exposed to PTEs has been assessed. These interventions can be broadly categorized into debriefing interventions, pharmacological interventions, brief interventions based on principles of cognitive-behavioral therapy (CBT), and Psychological First Aid.

Debriefing. Critical Incident Stress Debriefing (CISD; Mitchell, 1983) is a well-known intervention designed to be administered shortly after exposure to a PTE. A trained CISD

facilitator guides a single-session intensive discussion on mobilizing resources, and individuals are encouraged to express their thoughts and feelings about the PTE. CISD does not include standardized exposure or cognitive restructuring techniques. Although this intervention has been provided widely to individuals and groups who have experienced a broad array of PTEs, numerous studies and reviews have concluded that there is little to no empirical support for its efficacy (Adler et al., 2008; Gray & Litz, 2005; McNally et al., 2003; Forneris et al., 2013).

Adler and colleagues (2009) assessed the impact of debriefing interventions on the adaptation of nearly 2,300 American soldiers recently returned from Iraq. Platoons were randomly assigned to receive psychoeducation, Battlemind debriefing, or Battlemind training at the end of their deployments. Battlemind *debriefing* shares some similarities with CISD but includes less discussion of the PTE, educates service members about common challenges in the transition to civilian life, and promotes the seeking of peer support. Battlemind *training* teaches individuals to understand their experiences in a new way—for instance, perpetual readiness that is adaptive in combat may interfere with functioning in a safe civilian setting—and it encourages soldiers to adapt their existing occupational skills to fit their home environments. Among soldiers who reported high levels of combat exposure, those who received Battlemind debriefing and Battlemind training reported lower levels of PTSD symptoms at four-month follow-up compared to those who received only psychoeducation. In contrast, in a study of the impact of a modified version of Battlemind debriefing administered to United Kingdom military personnel immediately after completing a deployment to Afghanistan, Mulligan et al. (2012) found that individuals receiving the Battlemind intervention showed a lower level of binge drinking four to six months later, but no reduction in PTSD symptoms, compared to individuals receiving a standard post-deployment debriefing.

Psychopharmacology. Other researchers have investigated prevention through psychopharmacological interventions. Pitman and colleagues (2002) and Vaiva and colleagues (2003) tested the hypothesis that dampening or blocking elevated physiological responses in the aftermath of a PTE would reduce the intensity of emotional arousal associated with the PTE and, consequently, would prevent the development of PTSD. Pitman et al. (2002) studied 41 individuals presenting to an emergency department following PTEs. These individuals were randomly assigned to a 10 day course of a placebo or propranolol, a β-adrenergic-blocker. Participants receiving the active medication reported fewer symptoms of PTSD at one month follow-up. These results were replicated in a naturalistic series of 19 individuals who presented to an emergency room, 11 of whom received treatment with propranolol and 8 of whom agreed to participate but declined the medication (Vaiva et al., 2003). Extending these findings, Stein and colleagues (2007) compared the preventive effects of propranolol and gabapentin in 48 men and women who presented to a surgical trauma center. Within two days of their injuries, the individuals were randomly assigned to begin a 14 day course of propranolol, gabapentin, or placebo. Individuals were assessed at one, four, and eight months post-injury; reports of PTSD symptoms declined for participants in all three groups. There were no significant differences between groups. Although two of these three studies provide preliminary evidence for the usefulness of propranolol as an early intervention to prevent PTSD in PTE-exposed individuals, the very small sample sizes limit the conclusions that can be drawn until larger trials are completed.

Holbrook and colleagues (2010) and Bryant and colleagues (2009) conducted two separate naturalistic studies of the impact of morphine use following injury. Holbrook et al. found that among 453 U.S. military personnel who had served with the Navy or Marine Corps

and who had received treatment for serious physical injuries, those who were given morphine during early resuscitation and trauma care were less likely to develop PTSD, even when controlling for injury severity. Bryant et al. found similar results in 155 civilians admitted to a hospital following a range of PTEs. However, these and most other studies on pharmacological preventive interventions for PTSD (e.g., Gelpin et al., 1996) have been fraught with methodological limitations, including lack of randomization to treatment and inadequate sample sizes. More rigorous research is needed.

Brief CBT. Rothbaum and colleagues (2012) tested an early intervention designed to facilitate healthy processing of the memory of a PTE. They enrolled 137 adults presenting to the emergency department for a variety of civilian PTEs within the first day following the exposure. Participants were randomly assigned to three 60-minute weekly sessions of prolonged exposure—an abbreviated dose of an empirically supported treatment for PTSD—or to an assessment-only control group. The intervention included in vivo and imaginal exposure exercises and post-exposure processing of the trauma memory with a therapist. At 4 weeks and at 12 weeks post-injury, participants who received the intervention reported significantly lower levels of PTSD symptoms, as well as fewer symptoms of depression, than those in the assessment-only group. The authors reported a medium effect size for the decrease in PTSD symptoms for the intervention group in comparison to the assessment-only group. Subsequent analyses found that good treatment response was predicted by trauma type (i.e., injuries that were not sustained as a result of a sexual assault) and lower levels of dissociation at the start of the intervention (Price et al., 2014).

In contrast to most selective prevention efforts, which have involved interventions delivered by therapists or other trained professionals, Mouthaan and colleagues (2013) tested a

self-guided, Internet-based early intervention intended to prevent PTSD. The intervention included psychoeducation on PTSD, stress management and relaxation skills, and information on self-guided in vivo exposure. Three hundred adults who visited a trauma center and consented to participate in the study were randomized to receive the Internet intervention or a no-intervention control group; neither group was discouraged from seeking assistance outside of the study. Participants in both the intervention and control groups reported a significant decrease in distress over time, and no differences were found between the two conditions. The intervention's effectiveness may have been limited by the low dose utilized by most participants; on average, participants viewed the intervention website fewer than two times, suggesting that the intervention may not have been sufficiently compelling or tolerable.

Other selective preventive interventions have been tested in children, rather than adults, who were exposed to PTEs. Wolmer and colleagues (2011) reported on a large-scale intervention implemented in Israel's public educational system in a sample of approximately 1,500 fourth and fifth grade children. Five months after the end of the second Lebanon war, during which Israel experienced a series of high intensity rocket attacks, teachers provided a 14-session preventive intervention in a classroom setting. The intervention was based on stress inoculation models and included didactic information on emotions, relaxation techniques, and coping. The children enrolled in the study were assessed shortly before the intervention was rolled out and then 9 and 12 months after the attacks. The children who received the preventive intervention reported lower mean levels of PTSD symptoms at follow-up.

Berkowitz and colleagues (2011) conducted a pilot study of a four-session caregiver-child intervention in 176 children presenting to the emergency room for a variety of injuries sustained as a result of a PTE. Participants (and their caregivers) were randomized to receive

either the Child and Family Traumatic Stress Intervention (CFTSI) or a four-session control intervention within 30 days of exposure. CFTSI included psychoeducation for the caregivers and children as well as cognitive and behavioral techniques developed to facilitate adjustment. At 3 months post-treatment, the children who received the CFTSI intervention reported fewer and less intense symptoms of PTSD.

Aside from these relatively large studies of CBT-based selective preventive interventions, there have also been a few noteworthy smaller pilot studies. In a small randomized controlled trial, Resnick and colleagues targeted anxiety experienced during a gynecological forensic exam conducted shortly after sexual victimization (Resnick et al., 1999). The intervention consisted of a video featuring a model patient demonstrating a relaxed response to the examination as well as information about exposure, avoidance, and behavioral activation. The control treatment consisted of a brief conversation with a rape crisis counselor. At six-week follow-up there was no difference in rate of PTSD between the control and intervention groups, but those who received the treatment reported fewer symptoms of anxiety associated with the exam. In a sample of 17 traffic accident survivors, Gidron et al. (2001) tested a two-session phone-based intervention aimed at integrating verbal and non-verbal aspects of memories for PTEs. The participants were assessed within 24 hours of their motor vehicle accident and, in the intervention condition, were then contacted within the next 24 hours and were asked to recount their experience in great detail. Participants who received the second phone call were less likely to develop PTSD three to four months post-accident. Larger studies will be required, of course, before any strong conclusions can be drawn.

Psychological First Aid (PFA) and Combat and Operational Stress First Aid (COSFA). PFA and COSFA are widely disseminated intervention frameworks that were

developed to assist providers operating as part of an organized disaster response team (Nash & Watson, 2012; Ruzek et al., 2007; Schultz & Forbes, 2014). Although there are currently no data to speak to these programs' efficacy, several of their key components address known risk factors for PTSD. The overarching goal of PFA and COSFA is to promote resilience in the early post-trauma period and to connect individuals with resources that will facilitate recovery. Both PFA and COSFA place an emphasis on assessing and assisting with the practical needs and concerns of trauma survivors in order to help establish safety and reduce the incidence and magnitude of further stressors in the early post-trauma period. They also encourage individuals to connect to pre-existing or naturally occurring sources of social support and utilize active coping skills. Furthermore, they seek to destigmatize mental health treatment for individuals requiring professional help.

Indicated Prevention

Indicated prevention is predicated on the ability to identify individuals with pre-disease levels of suffering and impairment and on the assumption that offering early intervention to these individuals is a particularly effective and efficient way to head off chronic, severe, and debilitating distress and dysfunction. The existing literature includes a variety of operational definitions of pre-disease suffering and impairment. Most often screening instruments have been used to measure PTSD symptoms or the related symptoms of acute stress disorder (ASD), a diagnosis that was introduced chiefly to identify individuals whose significant early (first month) traumatic stress symptomatology in the wake of a PTE may be a precursor of PTSD. Approximately 50-75% of individuals who meet ASD criteria go on to develop PTSD, making individuals with the diagnosis an appropriate target for PTSD prevention efforts (Bryant, 2011).

In addition to targeting individuals with ASD or subsyndromal levels of PTSD symptoms, some studies noted below have also included individuals who have been diagnosed with PTSD or who might meet diagnostic criteria if they were formally evaluated. The distinction between indicated prevention and early treatment is blurry in PTSD, and we have thus been liberal in our inclusion criteria to ensure broad coverage of early interventions for posttraumatic symptoms. That said, none of the studies described below included participants who would be considered to have chronic PTSD.

Brewin and colleagues (2008) reported on the impact of a preventive intervention delivered to 82 individuals in the wake of a bombing in London; each of these individuals reported significantly elevated distress on the Trauma Screening Questionnaire, a 10-item measure of distress that has been demonstrated to have high levels of sensitivity and specificity in the prediction of PTSD. The intervention included trauma-focused cognitive–behavioral therapy and eye movement desensitization and reprocessing; the modal number of sessions was nine. By the end of the intervention, there was a large drop in the average level of PTSD symptoms reported by participants. However, in the absence of a control group, it is unclear how much, if any, of this reduction in symptoms was due to the intervention rather than to natural recovery.

Ehlers and colleagues (2003) conducted a randomized controlled trial comparing the impact of a self-help book versus a cognitive therapy intervention (vs. a repeated-assessments control condition) for 85 individuals who had been in a motor vehicle accident within the last three months, and who reported significant levels of PTSD symptoms during a three-week daily symptom monitoring period at the start of the study. Reports of PTSD, depression, and anxiety

symptoms declined in all conditions over the nine months of the study, but participants receiving the cognitive therapy reported the greatest treatment gains.

O'Donnell and colleagues (2012) designed and tested an early intervention aimed at preventing PTSD in high-risk individuals. A sample of 683 consecutive individuals who were hospitalized following traumatic injuries were administered the Posttraumatic Adjustment Screen, a 10-item measure intended to identify individuals at high risk of developing PTSD. A total of 366 individuals judged to be at high risk of PTSD based on their initial levels of distress were contacted again at four weeks post-injury and were re-assessed for symptoms of anxiety and depression. The 46 individuals who continued to show elevated levels of distress at this time were randomly assigned to receive 4–10 sessions of cognitive–behavioral therapy or usual care. Relative to usual care, patients receiving the early intervention had significantly better mental health at 12 month follow-up. Large treatment effects favoring the CBT intervention were found for PTSD, depression, and anxiety symptoms. However, the cell sizes were small, and the flexible treatment manual and variable doses of therapy utilized in this study make it difficult to compare its results to those of other studies in which the intervention procedure and dose were more rigidly defined.

Foa and colleagues (1995) pilot-tested the efficacy of four weekly two-hour CBT sessions in a sample of female assault victims with ASD. The 10 women who received the intervention showed earlier drops in PTSD and depression symptoms than 10 matched individuals who received only repeated assessments. However, at the final assessment, 5.5 months post-assault, the two groups of individuals did not differ significantly on measures of PTSD. In a larger sample (N = 90), Foa and colleagues (2006) found similar results. Specifically, in a randomized controlled trial comparing this brief CBT intervention to an assessment-only

condition and to a supportive counseling condition, final outcomes were similar across conditions.

More encouraging results came from a series of studies in which Bryant and colleagues (1998, 2003) compared the efficacy of five sessions of brief CBT versus supportive counseling. The therapies were begun within two weeks of a motor vehicle accident, industrial accident, or assault for individuals meeting ASD criteria. The CBT intervention included a combination of prolonged exposure, cognitive therapy, and anxiety management skill-building. The supportive counseling incorporated education on trauma, problem-solving skills, and supportive listening. At six months and at four years after the accident or assault, individuals who received the CBT intervention reported less severe PTSD symptoms than did those who received supportive counseling. However, there was a trend for greater dropout in the CBT intervention, suggesting that CBT may be less tolerable than supportive counseling for recently traumatized individuals. In addition, the lack of an assessment-only condition makes it unclear how the impact of either treatment would compare to the natural recovery process.

In a rigorous, randomized controlled trial that included treatment adherence monitoring, Shalev and colleagues (2012) studied 242 individuals who had visited a hospital following a PTE (e.g., terrorist attack, motor vehicle accident). The participants were enrolled within 30 days of the PTE exposure and met all criteria for PTSD other than the one-month duration requirement. They were assigned to prolonged exposure therapy, cognitive therapy, antidepressant medication, placebo, or a waitlist condition. Shalev and colleagues reported a large effect size for prolonged exposure and cognitive therapy on PTSD symptoms at five and nine months after exposure to the PTE; these interventions were more likely to prevent chronic PTSD than the wait list, placebo, and antidepressant condition.

Synthesis and Discussion

We presented preventive interventions within the Institute of Medicine scheme, which consists of three divisions: universal, selective, and indicated. Only one universal prevention program has been tested on a broad scale, and although it did not appear to cause harm, any benefits from it were very small and of questionable cost-effectiveness. The published results of selective prevention efforts are more varied. There is minimal evidence for the efficacy of debriefing-based approaches. Psychopharmacological interventions in the immediate aftermath of PTEs hold more promise, particularly for propranolol, but larger scale studies are needed to establish their risks and benefits more clearly. Evidence has been mixed for brief CBT interventions delivered to samples of individuals exposed to PTEs. However, it is possible that the reports of the mean change in distress in studies obscures the benefits of selective (as well as universal) interventions for higher-risk individuals or masks the potential benefits for some and the potential harm for others.

Greater attention is needed to identify how and for whom these broadly applied interventions are helpful (or harmful). The population that appears most clearly to have the potential to benefit from preventive interventions is individuals who show high levels of posttraumatic stress symptoms in the first month following a PTE, such as those with full or partial ASD. Indicated prevention interventions aimed at this population have largely consisted of adaptions of empirically supported PTSD psychotherapeutic treatments. Whether these interventions produce greater long-term results when delivered early to all individuals showing high levels of acute distress following PTEs, rather than later only to those individuals who develop chronic PTSD in the absence of a preventive intervention, is currently unknown.

It is noteworthy that although social support, subsequent stressor exposure (and presumed poor coping), and avoidant coping style are robust predictors of chronic PTSD, these factors have yet to be specifically targeted in preventive interventions other than Psychological First Aid. Rather, the assumption is typically that individuals with ASD or sub-syndromal levels of PTSD require some kind of intrapsychic processing of their experience (or sharing it with a therapist) to heal and recover from exposure to PTEs. This supposition is unfortunate because interventions designed to help people garner social support, reduce the impact of inevitable stressors, and change coping repertoires may be more ecologically valid and palatable than intrapsychic processing of the PTE.

Conclusion

Most people will be exposed to PTEs during their lifetime, and these events can have a significant impact on well-being and functioning. Although the impact is usually transient, in some cases individuals develop chronic and disabling PTSD. Psychologists have therefore worked to identify who is at highest risk for PTSD following PTE exposure, and have developed and tested a variety of early interventions aimed at preventing new cases of PTSD.

Little evidence supports the efficacy of preventive interventions aimed at broad populations of individuals who have experienced PTEs or who are at risk of doing so. In contrast, a growing body of evidence suggests that indicated psychotherapeutic interventions specifically targeting those individuals who show significant PTSD or ASD symptoms in the wake of PTEs may alleviate distress beyond what would be expected from the passage of time alone. However, key limitations of the existing research prohibit strong conclusions about the comparative effectiveness of such preventive interventions and their ideal timing, intensity, and dose, as well as the selection of individuals who should receive these interventions. For example,

studies have varied tremendously in participant eligibility criteria (e.g., the level of pre-disease suffering or impairment required to qualify) and the time elapsed between PTE exposure and the initiation of intervention, drop-out rates have often been high, and some studies have not included appropriate control conditions. Moreover, most preventive interventions have included several diverse therapeutic components; in the absence of dismantling studies, it is impossible to know which aspects of the interventions are beneficial and which aspects may instead be superfluous or possibly even detrimental. In addition to the dismantling of treatment components, future research on the prevention of PTSD should examine outcomes that extend beyond PTSD symptoms, such as suicidality and functional impairment, and should investigate how intervention outcomes are moderated by trauma type.

The literature on preventive interventions for PTSD has developed in parallel with the literature on risk factors for PTSD. The latter area of research has yielded results suggesting that screening for posttraumatic symptoms might most effectively be directed toward individuals who have experienced especially severe PTEs and who have a history of prior psychological difficulties or a limited repertoire of coping skills (e.g., an avoidant coping style). Furthermore, research on PTSD risk factors suggests that preventive interventions should include a stronger emphasis on helping individuals to utilize and build upon their social support networks, as post-trauma social support has consistently shown a strong buffering effect against the development of PTSD. Existing preventive interventions have typically focused almost exclusively on intrapsychic factors and would benefit from greater attention to interpersonal factors that facilitate recovery from early posttraumatic stress symptoms. Research on PTSD risk factors, in turn, would benefit from a greater focus on identifying *malleable* risk and protective factors

(such as post-trauma social support) and the interplay among these factors, so that this research can yield greater practical implications for the development of preventive interventions.

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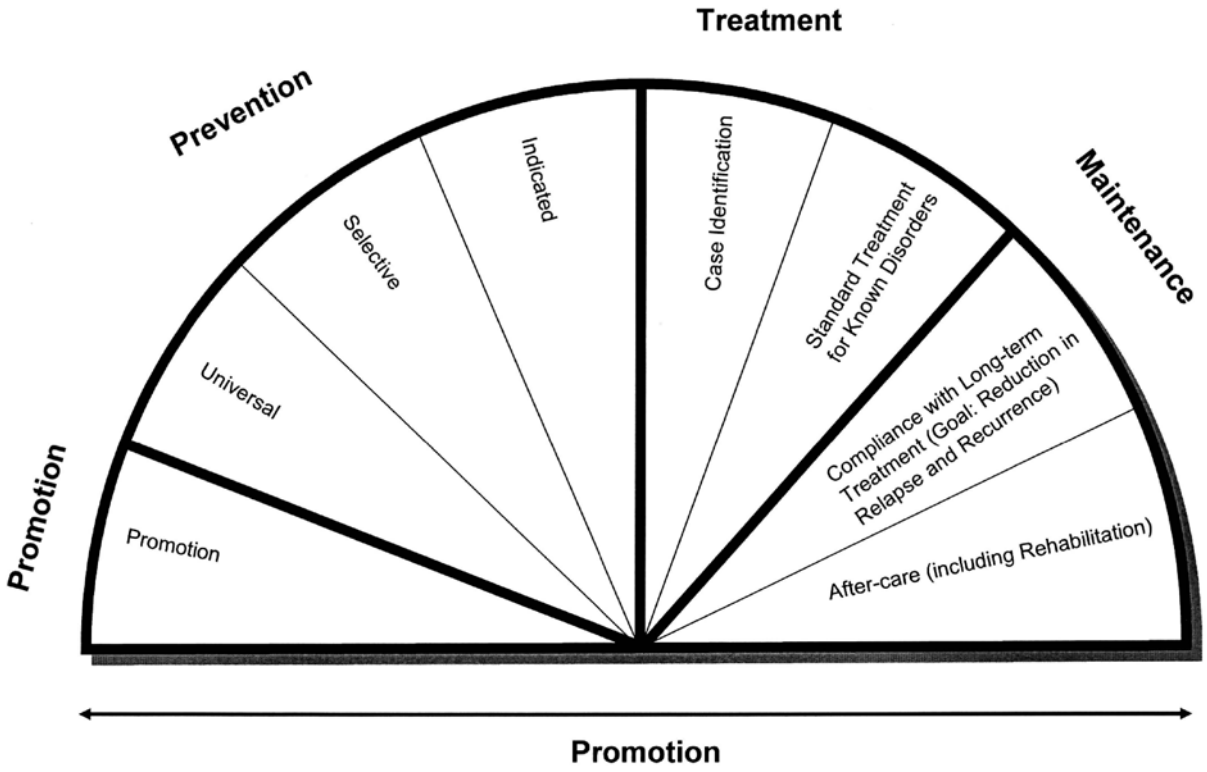

Figure 1. The Institute of Medicine (1994) continuum of care model.