Posttraumatic Stress Disorder Symptoms, Psychobiology, and Coexisting Disorders in Police Officers

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Over the last 20 years information describing post-traumatic stress disorder (PTSD) has accumulated at an ever increasing pace as have scientific and anecdotal reports evaluating the efficacy of frequently used treatments. In-depth descriptions of this work can be found in a number of excellent books and research articles (Bisson & Andrew, 2009; Cloitre et al., 2012; Friedman, Resick, Bryant, & Brewin, 2011; Ipser & Stein, 2012; Lanius, Bluhm, & Frewen, 2011; Levine, 2010; Ogden, Pain, & Fisher, 2006; Pitman et al., 2012; Porges, 2011; Schiraldi, 2000; Sharpless & Barber, 2011; Stein, Friedman, & Blanco, 2011; van der Kolk, McFarlane, & Weisaeth, 1996; Violanti, Paton, & Dunning, 2000). This chapter will briefly review the current state of knowledge about PTSD, and includes information on diagnosis, epidemiology, and neurobiology. We also discuss negative physical and mental health conditions that have been found to be associated with PTSD. Any review is necessarily eclectic and it is impossible to include all research contributions. Therefore, the papers discussed in this chapter are representative, but not exhaustive, of the PTSD literature.

PTSD Diagnosis

The World Health Organization (WHO) International Classification of Diseases (ICD-10) states that PTSD is “a response to a stressful event or situation of an exceptionally threatening or catastrophic nature, which is likely to cause pervasive distress in almost anyone (World Health Organtization [WHO], 1992). Prior to 1980 when PTSD was established as a formal diagnostic entity, it was called by many different names such as “hysteria,” “shell-shock,” “soldier’s heart,” and “delayed stress” (Pitman et al., 2012; van der Kolk et al., 1996). As our knowledge and understanding of PTSD has changed, so too has the way we think about and diagnose PTSD (Friedman et al., 2011; van der Kolk et al., 1996). Historically, PTSD was thought to be a “normal response to an abnormal event.” We now know that trauma initiates a complex psychological and biological process, and not all individuals exposed to the same traumatic event will develop PTSD (Keane, Marshall, & Taft, 2006). Despite much research concerning PTSD we have not been able to identify biomarkers, such as specific genes or blood markers

that will help us to predict who will develop the disorder, or biomarkers that are specific to the disorder itself and do not overlap with other psychiatric disorders. Currently, a diagnosis of PTSD is given when an individual meets a certain set of criteria defined in The American Psychiatric Associations (APA) fourth -revised Diagnostic and Statistical Manual of Mental Disorders (DSM-IV-TR) (American Psychiatric Association [APA], 2007).

These criteria include seven main components: 1) experiencing, witnessing, or being confronted with an event or events that involved actual or threatened death or serious injury, or a threat to the physical integrity of self or others, 2) the response to the event involved intense fear, helplessness, or horror, 3) re-experiencing the traumatic event, 4) avoidance/numbing symptoms, 5) symptoms of hyperarousal, 6) symptoms must persist for a defined period of time and, 7) symptoms must result in significant distress or functional impairment (APA, 2007; Schnurr, Friedman, & Bernardy, 2002; Spitzer et al., 2009; Yehuda, 2002).

Re-experiencing the event often comes in the form of intrusive memories, flashbacks, nightmares, or somatic sensations (APA, 2007; Schnurr et al., 2002; Spitzer et al., 2009; Yehuda, 2002). Even though the trauma occurred in the past, when triggered, individuals with PTSD feel as though they are re-experiencing the traumatic event, and report feeling that they will have a foreshortened future. In an attempt to avoid being triggered, individuals with PTSD avoid people, places, conversations, or activities that remind them of the event. They may avoid thoughts and feelings about the event and may even be unable to remember important aspects of the event. Avoidance can also take the form of emotional numbing, feeling estranged and disconnected from self and others. Arousal may be experienced as difficulty falling or staying asleep, difficulty controlling emotions such as anger and irritability, difficulty concentrating, and a state of constant hypervigilance (APA, 2007). Along with these symptoms, to meet the DSM IV-TR diagnostic criteria for PTSD, the duration of the symptoms must have lasted for more than one month and interfere with some important aspect of daily functioning,

such as social or occupational. However, the definition of a traumatic event has broadened over time (Rosen & Lilienfeld, 2008). Previously, a traumatic event was defined as life-threatening and causing intense fear and included events such as battle, car accidents, sexual assault, etc. It is now recognized that events like divorce, unemployment, and a diagnosis of diseases like cancer may result in symptoms of PTSD and that some individuals develop PTSD without having experienced "fear, helplessness, or horror" (APA, 2007; Calhoun et al., 2012; Friedman et al., 2011). These, and other changes in our understanding of PTSD symptomology will be reflected by modifications in the revised PTSD diagnostic criteria to be published as part of the APA's Diagnostic and Statistical Manual of Mental Disorders fifth version (DSM-5) (Friedman et al., 2011).

Epidemiology

Epidemiologic research helps us to understand individual risk factors of PTSD, while biologic and neurologic research helps us understand how trauma affects our bodies and our minds. Much of the existing research on PTSD has evaluated people who already have PTSD; this limits our ability to establish causation. Pre-trauma prospective studies are needed to make progress in establishing causation, and represent a crucial step in further developing effective treatments (Pitman et al., 2012). However, even in light of current limitations, studies continue to help us better understand PTSD and how to treat it more effectively.

Research shows us that PTSD is more likely to occur in occupational groups that are frequently exposed to traumatic events (Berger et al., 2012). These special populations include military personnel and first responders such as rescue workers, ambulance drivers, firefighters, and police officers (Marmar et al., 2006). Police officers are repeatedly exposed to traumatic situations including motor vehicle accidents, armed conflicts, and witnessing violent death across their working lives (Marmar et al., 2006). Approximately 7%-19% of police officers qualify for a diagnosis of PTSD, and approximately 34% experience a number of PTSD symptoms, but do not meet a full PTSD diagnosis (Carlier, Lamberts, &

Gersons, 1997; Gersons, 1989; Robinson, Sigman, & Wilson, 1997). Following a traumatic incident, a number of different factors have been found to affect whether an individual will develop PTSD. These factors include, but are not limited to, having a prior history of trauma, coping styles, irregular work hours, rotating shifts, and lack of social support both inside and outside work (Carlier et al., 1997; Marmar et al., 2006). Differences by gender and ethnic group have also been observed, although this is not consistent across all studies (Bowler et al., 2010; Bowler et al., 2012; Lilly, Pole, Best, Metzler, & Marmar, 2009; Marmar et al., 2006). These studies indicate that developing PTSD is not a function of any one thing, but how an individual's brain processes and store traumatic events.

Trauma and Memory Consolidation

Memory research indicates that individuals without PTSD rarely recall sensory details of an event such as smells, sensations, and movements of events; rather the brain reassembles and constructs stories around events, adding meaning, and a narrative, to create the "story" (Shapiro, 2002). In contrast, individuals with PTSD are able to report very specific, yet fragmented sights, sounds, movements, and sensations that occurred during the traumatic event (van der Kolk et al., 1996). Specific neurohormones such as norepinephrine released at the time of the trauma may play a role in memory over-consolidation while vasopressin and endogenous opioids may result in the amnesia for the traumatic event individuals with PTSD often display (van der Kolk et al., 1996). It has been suggested that due to these neurohormones, traumatized persons do not process the traumatic event into a meaningful narrative; therefore, trauma reminders result in the individual re-experiencing the trauma as if it were occurring in the present rather than as an event of the past. This, in turn, triggers a physiological response, rereleasing the neurohormones, strengthening the memory neuropathways, and perpetuating symptomology.

Psychobiology

Human beings are wired to manage, adapt, and learn from stressful events. The brain continually monitors the environment for danger (Levine, 2010; Siegel, 2010). In a process referred to as

"neuroception," certain neural circuits involving the prefrontal, limbic and brain stem areas determine which individuals and circumstances are safe or dangerous by detecting and evaluating body and facial movement as well as facial expression (Siegel, 2010). In the face of an actual or perceived threat, chemicals known as neurotransmitters (e.g. catecholamines) are released, resulting in the activation of either the sympathetic nervous system (SNS) or the parasympathetic nervous system (PSNS), the two branches of the autonomic nervous system (ANS) (Levine, 2010). Their activation produces automatic primitive neurobiological defense reactions, "fight-flight-freeze" responses (Levine, 2010). The fight or flight response is well understood and controlled by the SNS whereas the defense strategy of freezing/immobilization, thought to be a much older response from an evolutionary perspective, is less understood but is known to be controlled by the dorsal vagal branch of the PSNS (Porges, 2011). Its activation causes our blood pressure to drop and slows down our breathing and heart rate through its effects on nerve conduction and blood vessels (Porges, 2011; Siegel, 2010; Wilson & Keane, 2004).

Detection or perception of a threat or stressor also activates the hypothalamic-pituitary-adrenocortical (HPA) axis resulting in the release of a species-specific glucocorticoid. For humans it is cortisol that is released into the general circulation (O'Connor, O'Halloran, & Shanahan, 2000; van der Kolk et al., 1996). Cortisol increases our metabolism so sufficient energy is available, enabling our muscles and nervous system to quickly and efficiently deal with the threat. Cortisol acting in a classic negative feedback loop, shuts off the initiating steps of HPA activation by binding to glucocorticoid receptors in the brain, bringing the individual back to a state of homeostasis or "normal" function, ready to deal with the next threat. However, in individuals with PTSD the brain and body systems dealing with danger or threat are over-reactive or react in an abnormal way in comparison to individuals without PTSD (Kim et al., 2011).

The neurocircuitry of PTSD has received much attention and brain imaging studies, including various techniques that directly or indirectly image the function and structure of the brain, are helping us

understand the neurological basis for some of the symptoms reported by individuals with PTSD (Hayes, Hayes, & Mikedis, 2012; Lanius et al., 2011; Patel, Spreng, Shin, & Girard, 2012; Pitman et al., 2012; Shin, Rauch, & Pitman, 2006). Because it tracks brain metabolism, functional imaging allows the direct visualization of the centers of the brain involved in processing information. Many functional brain imaging studies show differential activation in some regions of the brain when individuals with PTSD are compared to individuals without PTSD (Hayes et al., 2012; Patel et al., 2012; Pitman et al., 2012). A meta-analysis of multiple functional imaging studies of those individuals diagnosed with PTSD found the mid-anterior cingulate cortex (mACC), the dorsal anterior cingulate cortex (dACC) and the bilateral amygdala are the most hyperactivated regions, while the most hypoactivated include the ventro-medial prefrontal cortex (vmPFC), the inferior frontal gyrus and the insula. These differences in activity of certain brain circuits lead to the hypothesis that PTSD is due to a failure of the vmPFC to inhibit the amygdala.

One job of the vmPFC is to help us predict the occurrence of threatening events based on our past experiences; this is referred to as fear conditioning. Eventually, however if a threatening or painful event repeatedly does not occur following the fear conditioning stimulus, an extinction of the fear response occurs (Wessa & Flor, 2007). It has been suggested by behavioral psychologists that individuals with PTSD exhibit enhanced acquisition of the fear response, as well as a delay in its extinction. The initial traumatic event is the stimulus that triggers a fear response. Through additional conditioning, trauma related reminders or cues appear to maintain the fear response and hinder fear extinction. Decreased activity in the vmPFC may play a role in maintaining the fear response and inhibiting fear extinction, possibly due to the inability of this brain area to reduce amygdala hyperactivity (Pitman et al., 2012).

Besides fear conditioning and extinction, the vmPFC is important in self-referential processing (Lanius et al., 2011). Lower activity in the vmPFC may result in disturbances in self-referential

processing, which undermines an individual's sense of self and life purpose. It is associated with symptoms of shame, identity disturbance, and dissociation. It is also associated with an individual's ability to tune into and attend to the wants and desires of others (Lanius et al., 2011). Another job of the vmPFC is to communicate with the limbic system. Through this communication we are able to monitor and modify our own emotions. Therefore, reduced activity in this region of the brain may be one of the reasons why individuals with PTSD, once triggered, have difficulty identifying and controlling their emotional responses. Similarly, reduced activation of the insula has also been shown to be associated with the inability to label and identify emotions.

Other than the vmPFC, the insula is also associated with our ability to identify and label emotions. It is involved in interoceptive awareness of the body and the regulation of the sympathetic and parasympathetic systems, including the sensation of pain (Hayes et al., 2012; Lanius et al., 2011). Under-activation in this region of the brain may also be one of the reasons why individuals with PTSD become emotionally overwhelmed in the face of traumatic reminders.

Regions of the brain that appear to be more active in individuals with PTSD compared to those without PTSD are the dorsal anterior cingulate cortex (dACC), the amygdala, and the hippocampus. The dACC plays a critical role in fear conditioning and extinction, and is involved in pain perception, as well as appraisal and evaluation of the environment. Increased activity in this region of the brain may be the reason why individuals with PTSD are hypervigilant and report feelings of hyperarousal (Hayes et al., 2012).

A large number of functional imaging studies indicate individuals with PTSD have increased brain activity in the amygdala compared to individuals without PTSD (Hayes et al., 2012; Lanius et al., 2011; Pitman et al., 2012; van der Kolk et al., 1996). The amygdala is important in fear conditioning, modulation of memory consolidation, and emotional and social processing (Lanius et al., 2011). It is responsible for assigning emotional meaning to incoming stimuli that are further processed into a

personal narrative in the neocortex via the hippocampus, hypothalamus, and basal forebrain (van der Kolk et al., 1996). Increased amygdala activity has been shown to have an inverse relationship with activity in the vmPFC (Hayes et al., 2012). It has been suggested that increased amygdala activation is associated with exposure to trauma in general and not PTSD specifically, as activity in this area did not differ when comparisons were made between those exposed to a trauma with and without PTSD (Patel et al., 2012). This finding also suggests it is important to include trauma-exposed and non-trauma-exposed controls in studies of PTSD.

The hippocampus lies next to the amygdala and plays a critical role in processing emotionally laden memories. It is important in short-term memory and is involved in the formation of autobiographical memories, and interpreting environmental cues (Pitman et al., 2012; van der Kolk et al., 1996). Over activation of this region along with the amygdala may contribute to intrusive thoughts frequently experienced by individuals with PTSD (Patel et al., 2012). Structural MRI studies initially indicated that the hippocampal volume was smaller in individuals with PTSD compared to those without, but the idea that a decrease in hippocampal size is linked to trauma exposure is controversial (Pitman et al., 2012). Subsequent research using identical twins found no difference in their hippocampal volume although the twin that experienced combat developed PTSD. Furthermore, the hippocampal size for both twins was smaller than twins discordant for combat where PTSD did not occur. This data suggests that hippocampal volume may be genetically determined and not specifically associated with trauma exposure (Pitman et al., 2012; van der Kolk et al., 1996). Further complicating the issue is the observation that hippocampal volume can be increased by pharmacological treatment (e.g., serotonin reuptake inhibitors) suggesting volume is malleable (Vermetten, Vythilingam, Southwick, Charney, & Bremner, 2003). The size of other brain structures important in PTSD can be impacted by exogenous treatments; for example chronic corticosteroid therapy reduces amygdala as well as hippocampus volume (Brown et al, 2008). Finally, a recent meta-analysis found that trauma exposed

individuals without PTSD have smaller hippocampal volumes when compared to individuals with no trauma exposure and no PTSD suggesting trauma itself may cause a loss of volume (Woon, Sood, & Hedges, 2010). Thus, there is still active debate as to the origins and role of hippocampal volume in PTSD and trauma exposure.

Physical and Psychological Consequences of PTSD

Ongoing research has contributed to our understanding of the biological and neurological link between PTSD and coexisting physical and psychological disorders (Gupta, 2013; Hayes et al., 2012; Kim et al., 2011; Lanius et al., 2011; O'Connor et al., 2000; Pitman et al., 2012). Structural and functional neuroimaging studies indicate that in individuals with PTSD, specific regions of the brain associated with memory consolidation, fear conditioning and fear extinction, as well as emotion regulation, activate responses in the ANS (fight-flight-freeze response) and HPA axis (Hayes et al., 2012; Kim et al., 2011; Lanius et al., 2011; Pitman et al., 2012; Porges; 2011). Both the ANS, through the activation of catecholamines, and the HPA axis, through cortisol, act on multiple biological systems within the body (Gupta, 2013; Lukaschek et al., 2013). In addition, activation of the freeze response is mediated by the dorsal vagal complex, which carries nerve signals from the hypothalamus to the heart, digestive system, and lungs as well as receives nerve signals from these systems (Porges; 2011). Dysregulation of the ANS and HPA axis as seen in individuals with PTSD may be associated with coexisting psychological and biological disorders (Gupta, 2013; Lukaschek et al., 2013).

There are a number of studies that have focused on the general population and on police officers specifically, which indicate that individuals with PTSD are at an increased risk of CVD, coronary heart disease, hypertension, and possibly stroke (Coughlin, 2011; Sareen et al., 2007; Violanti, Andrew, et al., 2006; Violanti, Fekedulegn, et al., 2006). PTSD symptoms have also been found to be associated with metabolic syndrome and a reduction in brachial artery flow-mediated dilation (Violanti, Andrew, et al., 2006; Violanti, Fekedulegn, et al., 2006). In a cross-sectional study of 2,970 general population

participants, those with PTSD were found to be three times more likely to have type 2 diabetes compared to individuals without a traumatic event (Lukaschek et al., 2013). Gupta (2013) conducted a review of the literature and found that PTSD has been shown to be associated with a wide range of chronic diseases other than CVD and diabetes (Gupta, 2013). These include diseases such as chronic fatigue syndrome, fibromyalgia, gastrointestinal disorders, autoimmune disorders, and chronic pain syndromes such as migraine headaches (Gupta, 2013). While most of the studies included in the review were cross-sectional in nature, and therefore cause-effect relations could not be determined, and independent research studies are needed to confirm and expand many of these observed relationships, this review indicates that PTSD is associated with a large number of negative health outcome (Gupta, 2013; Sareen et al., 2007). Finally, it is important to note that the frequency of experienced traumatic events has been found to correlate with symptom severity (Suliman et al., 2009; Uddin et al., 2010). For this reason, officers who are exposed to multiple traumatic incidents may experience more severe symptoms compared to individuals who have had relatively few traumatic events.

Other than an increased risk of physical health conditions, individuals with PTSD are also more likely to experience a number of comorbid psychological conditions, suicidal ideation, and to report reduced quality of life (Maia et al., 2007; Martin, Marchand, & Boyer, 2009; Sareen et al., 2007). Sareen et al. (2007) found that PTSD was associated with major depression, mania, panic attacks, agoraphobia, social phobia, and substance abuse (Sareen et al., 2007). They also reported that PTSD was associated with high distress, high suicidal ideation, and poor psychological well-being (Sareen et al., 2007). In studies that have evaluated PTSD with indicators of poor health in officers, officers with PTSD were more likely to report more frequent medical appointments, more use of sick leave, and more hospital admissions compared to officers without PTSD (Maia et al., 2007; Martin et al., 2009). They were also more likely to report poorer health than individuals without PTSD (Maia et al., 2007; Martin et al., 2009).

Summary

PTSD is frequently diagnosed following a single traumatic event; however epidemiologic research indicates that an individual's risk of PTSD increases with the number of traumatic experiences (Berger et al., 2012). In individuals with PTSD, repeated or chronic activation of the stress response systems (e.g. the SNS and HPA axis), may eventually result in their inefficient operation (Kim et al., 2011; O'Connor et al., 2000). This inefficient operation may result in the release of too much or too little of the physiologically active substances such as catecholamines and cortisol that play a role in dealing with threat (Kim et al., 2011; O'Connor et al., 2000). Abnormal levels of these substances are thought to be associated with the development of inflammatory, endocrine, cardiovascular, and psychiatric disorders in individuals with PTSD (Coughlin, 2011; Gupta, 2013; Lukaschek et al., 2013; Sareen et al., 2007). Because police officers are frequently exposed to traumatic incidents over their working lives, they are at an increased risk of PTSD. This in turn increases their risk of a number of other coexisting biological and psychological disorders such as CVD, diabetes, and depression (Gupta, 2013; Lukaschek et al., 2013; Marmar et al., 2006; Stearns, n.d.; Violanti, Andrew, et al., 2006; Violanti, Fekedulegn, et al., 2006). Because of the profound impact PTSD has on police officers, we should strive to prevent, diagnose and treat this disorder in this population.

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