


# Physical Health Problems After Single Trauma Exposure: When Stress Takes Root in the Body

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## Abstract

Research has established that chronic stress, including traumatic events, leads to adverse health outcomes. The literature has primarily used two approaches: examining the effect of acute stress in a laboratory setting and examining the link between chronic stress and negative health outcomes. However, the potential health impact of a single or acute traumatic event is less clear. The goal of this literature review is to extend the literature linking both chronic trauma exposure and posttraumatic stress disorder to adverse health outcomes by examining current literature suggesting that a single trauma may also have negative consequences for physical health. The authors review studies on health, including cardiovascular, immune, gastrointestinal, neurohormonal, and musculoskeletal outcomes; describe potential pathways through which single, acute trauma exposure could adversely affect health; and consider research and clinical implications.

## Keywords

posttraumatic stress trauma, health, health behavior, comorbidity

The question of how trauma affects health is highly salient, given the prevalence of trauma in our society. Estimates vary, but the most comprehensive and nationally representative prevalence study to date indicates that at least 60% of men and 51% of women in the general population report experiencing at least one traumatic event in their lives (Kessler, Sonnega, Bromet, Hughes, & Nelson, 1995). In Kessler et al.'s study, the three most commonly experienced types of trauma were witnessing someone being badly injured or killed; being involved in a fire, flood, or natural disaster; and being involved in a life-threatening accident. In addition to single-incident or acute trauma exposure, chronic or complex trauma exposure is prevalent in our culture. Exposure to early life adversity, such as physical abuse, sexual abuse, and neglect are common: Approximately one third of children are estimated to experience physical abuse (Anda et al., 1999); approximately one in four girls and one in five boys experience sexual victimization during childhood (Finkelhor, Hotaling, Lewis, & Smith, 1990). Other forms of nonabuse trauma, such as exposure to community violence, are also common (Bell & Jenkins, 1993). Such forms of trauma are notable because they occur repeatedly over key developmental periods, thereby increasing the likelihood of negative outcomes via chronic activation of stress response systems.

The association between health outcome and chronic traumatization have been researched extensively (Irish, Kobayashi, & Delahanty, 2010; Neumann, Houskamp, Pollock, & Briere, 1996), and concepts such as allostatic load have been elaborated to help study the connection between chronic stress exposure and health burden (for review, see Friedman & McEwen, 2004). What remain less clear are whether and how exposure to more circumscribed trauma could bear similar weight and the processes leading to illness.

Our premise is this: *Trauma sets the stage for ongoing psychological and physical distress, which can mutually affect one another, possibly for the duration of the survivor's*

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*life span*. The purpose of this review is to examine the empirically derived basis for whether and how acute trauma exposure could adversely affect physical health. First, we will examine how this could occur using the following chain of reasoning: (a) that in early development the impact of trauma exposure is seen in dose–response relationships to the leading causes of death, (2) that it causes posttraumatic psychopathology that can be chronic and accrue comorbidities, (3) that trauma exposure can have a unique, independent association with health outcomes, but (4) that trauma’s psychological impact, mediated via impacts on the neuropsychiatric system, may relate to long-term health outcomes through two pathways: (a) the “cues” or triggered reminders that sustain the association of the trauma exposure to distress and, in turn, to adverse health outcomes and (b) the demands that may be attributed to emotional management techniques such as suppression and dissociation. We hypothesize that these processes turn an acute event into a chronic one.

### **Definitions and Caveats**

One area of confusion and ambiguity within this literature is related to thresholds distinguishing several concepts: “acute” and “chronic,” “exposure” and “impact,” and “trauma” and “stress.” Some of the confusion arises because the reality is that creating thresholds between continuous constructs creates a false dichotomy. Nonetheless, for our purposes, a clarification of how terms will be used in this article may leave the reader with a more precise understanding of what research has definitively stated, and what remains ambiguous. “Trauma” will refer to events, rather than their potential consequences, and will be used consistent with the *Diagnostic and Statistical Manual of Mental Disorders*, fourth edition, text revision (*DSM-IV-TR*; American Psychiatric Association, 2000) definition: a discrete event or events with threat to life or bodily integrity that result in fear, helplessness, or horror. In contrast, stressors are mild to extreme demands. Stress is a state of burdened response to stressors, resulting in deterioration and dysfunction (Selye, 1956). Traumatic events or trauma exposure refers to the actual events themselves: car accidents, assaults, abuse, natural disasters. Posttraumatic stress, posttraumatic symptoms, and posttraumatic stress disorder (PTSD; as defined by the *DSM-IV-TR*) refer to the client-reported distress attributed to traumatic events (e.g., feeling upset in response to reminders, wanting to avoid places related to the trauma). So, too, does psychiatric distress refer to the client’s self-reported emotional distress, rather than possible underlying or unconscious pathology or other psychological processes. Traumatization refers to the process by which a traumatic event incurs posttraumatic stress. Both stressors and traumatic events, as well as their impact,

may be acute (lasting moments or hours) or chronic (lasting months or years). Physiological arousal is measured as both tonic (e.g., baseline or trait) and phasic (e.g., change from baseline in response to an experimental stimulus). As we will argue here, an acute event may nonetheless have a chronic impact. Finally, the term *somatization*, arising from the psychoanalytic literature, refers to the process of expressing psychological distress in physical terms, as a defense for keeping psychological distress and conflicts out of awareness (Lipowski, 1988). Generally, somatization refers to physiological distress in the absence of any physical indicators of disease.

Of course these terms and their suggested precision are somewhat misleading. A traumatic event may not register as such for an individual whose level of daily stress is extremely elevated. Conversely, for someone already burdened and depleted by extreme stress, an event that might be otherwise manageable might be processed as a traumatic event. Similarly, a traumatic event may not be “over” when it is over; in the eyes of the PTSD sufferer, the event may be reexperienced as intensely as when the event first occurred. The result is that the lines between acute and chronic and between event and outcome become unclear, creating ambiguity in how we interpret research findings. This ambiguity is reflected in the research: Many studies attempting to capture the impact of acute traumatic events have not thoroughly included measures of premorbid trauma exposure or level of life stress. The findings presented herein may be characterized by some of these flaws. We will provide as much precision as possible.

### **Chronicity of Exposure Is Related to Health Outcomes**

Multiple exposures to traumatic event can greatly affect the intensity of not only the psychological symptoms but also the physical symptoms. The Adverse Childhood Experiences Study (Anda et al., 2006; Felitti et al., 1998) is one of the most powerful studies to document such findings. Felitti et al. surveyed more than 16,000 adults with health insurance. Of the risk factors investigated (e.g., trauma exposure in childhood and adulthood, health behaviors, and potential genetic factors), childhood trauma exposure accounted for negative health outcomes across multiple diagnoses, from psychiatric (depression, anxiety, substance abuse) to physical (diabetes, heart disease, and cancer). Risk for disease increased linearly with exposure to multiple forms of traumatization. Similarly, using data from the National Comorbidity Survey Replication, Sledjeski, Speisman, and Dierker’s (2008) recent study supported the Adverse Childhood Experiences Study findings, demonstrating that exposure to multiple,

cumulative traumatic events across the life span was linked to negative health outcomes and that the health outcomes appear to be independent of PTSD. Although this review highlights acute trauma exposure for argument purposes, the data make clear that exposure to chronic traumatization, particularly early in life, confers exceptional risk for health burdens. Anda et al. (2006) conclude that childhood trauma is the biggest public health problem in the United States. Furthermore, chronic exposure is the rule, rather than the exception (Cook et al., 2005).

## Trauma Causes Psychopathology

Although the psychological effects of trauma are well established, we will briefly review them here for the purpose of orienting the reader to the interplay of psychiatric and physical illness. In particular, we would like to emphasize the multiple psychiatric presentations of distress following trauma exposure in order to help alert practitioners to the possibility that trauma may play a role in a given patient's symptoms.

PTSD is characterized by three symptom clusters: intrusions (e.g., flashbacks, nightmares, emotional or physiological reactivity to reminders), avoidance/numbing (e.g., avoiding reminders associated with the event, loss of affect), and hyperarousal (exaggerated startle, hypervigilance, difficulty sleeping). To meet criteria for PTSD, one must have experienced a "Criterion A" traumatic event, characterized by an event in which the life or physical integrity of the person or a loved one is harmed or threatened with harm and that results in the emotional reactions of fear, helplessness, or horror. Though PTSD is among the most commonly recognized outcomes of trauma, trauma most often results in a broad array of co-occurring psychiatric diagnoses. Almost all people with PTSD have at least one other diagnosis (Kessler et al., 1997). Trauma results in forms of mental illness including (but not limited to) major depressive disorder, generalized anxiety disorder, panic disorder, and substance abuse disorders (Kessler et al., 1997). At least one third of people with PTSD do not respond to short-term intervention (Bradley, Greene, Russ, Dutra, & Westen, 2005).

### *Trauma Results in Multiple Co-Occurring Forms of Psychopathology*

Available research suggests that trauma rarely has a discrete impact; rather, its psychological impact appears to be broad, affecting mood and anxiety. Kessler et al. (1997) interviewed 8,000 civilians and found that of those with PTSD, 88% had at least one other co-occurring psychiatric illness. Creamer, Burgess, and McFarlane (2001) used a gold standard structured psychiatric interview to determine co-occurring mental illness in a sample

of 10,000 Australians. Compared with men without PTSD, men with PTSD were 27 times more likely to develop major depressive disorder, 38 times more likely to develop generalized anxiety disorder, and 28 times more likely to develop panic disorder. Compared with women without PTSD, women with PTSD were 23 times more likely to develop depression, 10 times more likely to develop generalized anxiety disorder, and 10 times more likely to develop panic disorder. Zimmerman, McGlinchey, Chelminski, and Young (2008) interviewed 2,300 psychiatric outpatients: People with PTSD had an average of two other co-occurring psychiatric diagnoses.

### *The Effects of Trauma on Mental Health Are Long Lasting and Treatment Resistant*

Breslau (2001) found that one third of a sample of 1,000 people have significant symptoms of PTSD at least 10 years following a traumatic event. Barth, Kopfmann, Nyberg, Angenendt, and Frommberger (2005) found that among traffic accident victims interviewed 5 years after their trauma, 24% still had clinically significant PTSD. They also had significant ongoing occupational and relational impairment. Similarly, Koren, Arnon, and Klein (2001) found that among traffic accident survivors, half who had symptoms of PTSD had significant symptoms even 4 years later. Recently, a meta-analysis on the treatment of depression (Nanni, Uher, & Danese, 2011) has documented that treatment resistance in mood disorders is significantly higher in people with early-life trauma histories. Similarly, in a meta-analysis of gold standard treatments for PTSD (Bradley et al., 2005), the authors found that least one third of people with PTSD do not respond to currently established interventions, suggesting that PTSD is resistant to short-term treatment.

### **Trauma Exposure Leads to Overall Negative Physical Health Consequences**

The diagnosis of PTSD contains within it physiological symptoms (e.g., difficulty sleeping, physiological reactivity to reminders), as do other psychological disorders, such as anxiety and depression. That physical symptoms are embedded in psychiatric diagnoses suggests recognition that psychological and physical processes are intertwined, and this relationship can be seen clearly in trauma.

In terms of self-reported health and trauma exposure, a study of 1,500 individuals in New Zealand showed a strong association between exposure to criminal victimization and accidents and reduced health status, which included more current and chronic physical symptoms and an interference with daily functioning (Flett, Kazantzis,

Long, MacDonald, & Millar, 2002). Findings held when controlling for sex, age, and ethnicity. An additional study by Ullman and Siegel (1996) of a community sample of 2,634 participants found that when potentially confounding variables, such as demographics, psychiatric diagnoses, and stressful nontraumatic daily life events, were controlled, the association between trauma exposure (both acute and chronic) and poor health perception and health limitations on daily life remained.

Other research has focused on physician-documented medical impairment. In a 3-year longitudinal national study by Holman et al. (2008), 2,729 participants were studied before and after the terrorist attacks on 9/11. This study showed that physician-diagnosed cardiovascular ailments increased from the years preceding the attacks. Increases were also evident in respiratory, gastrointestinal, genitourinary, and musculoskeletal conditions, all adjusted for age. It is of note that the majority of participants in their study did not have direct exposure to the attacks (e.g., a loved one who was killed, in proximity of Ground Zero during the attacks). This note is important for three reasons: First, it suggests that the participants were likely not the most severely affected, as their exposure was indirect. Second, it suggests that physical ailment was not caused by characteristics of the attack, such as exposure to toxins. Finally, it is important to note that the level of traumatic exposure experienced by participants in this study might not be considered a Criterion A traumatic event as defined by the *DSM-IV-TR* (American Psychiatric Association, 2000), which would formally exclude participants from a diagnosis of PTSD. Furthermore, authors assessed somatization (a tendency to report physical distress in the absence of physiological indicators of illness) and found that it did not explain the increase in physical health incidents after the attacks.

A diagnosis of PTSD may additively increase the impact of trauma exposure on health. A PTSD diagnosis is associated with poor self-report health and physical symptoms (Kulka et al., 1990), poor disease course (Fifer et al., 1994), chronic physical health conditions (Schnurr, Ford, et al., 2000), more physician-diagnosed medical conditions (Beckham et al., 1998), and more health-related functional impairment (Stein, McQuaid, Pedrelli, Lenox, & McCahill, 2000). Even when the trauma is in the distant past, persistent PTSD diagnostic status is a risk factor for the later development of a medical illness (Friedman & McEwen, 2004; Schnurr & Jankowski, 1999). According to Boscarino's (2004) study of 2,490 Vietnam veterans, PTSD is linked with arthritis, insulin-dependent diabetes, and thyroid disease, up to 30 years after the initial trauma exposure.

Although it is possible that psychiatric symptoms could lead to poor health behavior, it is important to note that the health burden of PTSD exists even when health behaviors

are held constant. Boscarino (1997) found that severity of trauma exposure was related to significant increases in a variety of diseases, including musculoskeletal, digestive, endocrine, and circulatory disorders, even when controlling for health behavior. PTSD diagnosis additively increased health burden. Other studies have documented the same effect. Schnurr and Spiro (1999) found that among 921 male veterans, PTSD had a direct effect on health status, more than the effect of smoking and alcohol use. Zatzick et al. (2002) found that among acute injury survivors, physical health symptoms persisted and were more severe among people with higher levels of PTSD, independent of initial injury severity. Lauterbach, Vora, and Rakow (2005) found that in a sample of 5,877 participants, people with PTSD had a higher frequency of physical health diagnoses even after controlling for sex, health perception, daily stress, health-related behaviors, trauma exposure, co-occurring psychiatric diagnoses, and neuroticism. Lawler, Ouimette, and Dahlstedt (2005) found that in 138 students, PTSD symptoms were associated with poorer physical health status independent of age, health behavior, and co-occurring psychiatric diagnoses. Norris, Slone, Baker, and Murphy (2006) found that PTSD mediated between trauma exposure and physical health in a sample of 666 flood and disaster survivors. Taken together, these studies persuasively argue that having PTSD is bad for one's health, even if one avoids negative health behaviors.

### Potential Mechanisms for How Trauma Affects Health

As has been demonstrated above, traumatized individuals are clearly at risk for increased physical illnesses. It appears that the physical consequences of trauma occur in two scenarios. In one, individuals manifest clear psychiatric distress, most frequently reported as PTSD but also as depression and anxiety. In this scenario, the experience of trauma creates sensitivity to reminders that continually reactivates stress, creating multiple recurring "acute" stressors that string together. This scenario parallels the chronic stress literature that elaborates models of allostatic load (McEwen & Wingfeld, 2003). In the other scenario, no distress is reported or documented. We hypothesize that this scenario represents one of two conditions: (1) physical illness is generated by cued reactivity, but that reactivity (and any associated distress) is below the individual's awareness threshold; and/or (2) emotional coping styles that serve to reduce stress are physical taxing. That both people with and without PTSD are at risk for physical illness following trauma exposure suggests that there is more than one pathway between trauma exposure and physical illness. Here we will explore two potential (possibly overlapping) pathways: triggered associations and emotion inhibition.



### **Trauma Creates Associations to a Triggered Reminder That Continually Reactivates Symptoms**

The psychological and physical impacts of a traumatic event frequently do not cease after the end of the immediate traumatic event. Exposure to a traumatic event can lead to continued physiological, behavioral, and cognitive responding to previously neutral, nonthreatening stimuli (Resnick, Acierno, & Kilpatrick, 1997). These cues involve a wide range of stimuli, not just the specific event and environment (i.e., time of day, sounds, smells, etc.). Research suggests that the stimuli that may provoke this psychophysiological arousal may not necessarily be directly related to the original trauma (e.g., Pitman, Orr, & Shalev, 1993). The triggers may be generalized and precipitate equally intense, extreme reactions. Reminders of the event may lead to an acute autonomic nervous system arousal that may be as strong as when enduring the initial trauma (Pitman et al., 1993). In other words, a person who has experienced a traumatic event is prone to a resurgence of the physical complaint when reexposed to people, places, or things that evoke recollections of the event. This triggered reminder is not time limited and can be prevalent years after the initial trauma exposure.

Physiological reactivity to cued reminders is thoroughly documented in autonomic nervous system research. Pole's (2007) meta-analysis examined 58 studies and more than 1,700 participants. His summed findings supported the idea that individuals have physical reactions to internal cues (e.g., thoughts, feelings) and external cues (e.g., sounds, sights). Participants with PTSD had elevated heart rate and skin conductance (an indicator of sympathetic activity) when their resting activity was measured; when exposed to personalized or standardized reminders, heart rate was elevated; and, when exposed to startling sounds (a measure of reflexive arousal), heart rate and skin conductance were elevated. These findings suggest that PTSD is related to exaggerated sympathetic nervous system activity both when resting and in response to proximal or distal trauma cues. However, it is important to note that elevated heart rate may be a result of increased sympathetic activity as well as decreased parasympathetic activity (Berntson, Cacioppo, & Quigley, 1993).

One important point to note: Someone may be physiologically reactive to trauma cues without self-reports of emotional distress. As a proof of principle, Lazarus and McCleary (1951) presented neutral experimental stimuli outside the threshold for conscious perception and paired the stimuli with aversive shocks. On retesting, days later, the previously neutral stimuli elicited significant autonomic stress responses—even when the stimuli were presented below the threshold for conscious perception. This effect has been replicated in studies of brain activity as well (e.g.,

Wong, Bernat, Bunce, & Shevrin, 1997; Wong, Shevrin, & Williams, 1994). Research such as this demonstrates that (a) reactivity to cues is persistent and (b) reactivity to trauma cues may occur outside of conscious awareness.

While reactivity to trauma-related stimuli is one pathway to chronic physical stress, this matter is compounded by the fact that individuals with PTSD develop neurocognitive deficits that impair capacity to interpret and regulate responses to both emotional and neutral stimuli. For example, permanent neuronal changes within the central nervous system can be seen with the acoustic startle response, which occurs when an individual misperceives harmless stimuli as threatening (Shalev & Rogel-Fuchs, 1992). Neuro-psychologically aberrant responses are particularly evident with respect to attention, memory, planning, organization, inhibition of impulses and fear, and abilities to filter out irrelevant information (for a thorough review, see Buckley, Blanchard, & O'Neill, 2000). Brewin, Kleiner, Vasterling, and Field (2007) meta-analyzed 27 studies involving 1,400 participants and found that PTSD was consistently associated with memory impairment, particularly verbal memory, even for nontraumatic stimuli. Deficits were found for both immediate and delayed recall cognitive tasks and were not explained by a history of head injuries. Importantly, participants with trauma exposure but not PTSD did not differ from participants with PTSD. These data suggest that neurocognitive deficits may create scenarios in which life becomes difficult to navigate and interpret. Ambiguous or diffuse memory and cognition may turn day-to-day events into triggers of distress, perpetuating the course of illness and enhancing stress reactivity. These deficits may be particularly relevant to trauma-exposed patients who are otherwise nonsymptomatic: They may report such symptoms in the absence of other distress.

With the advent of technologies that allow the direct assessment of central nervous system activity, research has begun to document how the brain responds when presented with trauma reminders. Because they have been reviewed systematically elsewhere (see Karl et al., 2006; Smith, 2005), we will only briefly summarize. Karl et al. (2006) meta-analyzed 50 studies examining structural brain abnormalities with trauma exposure, both with and without PTSD. People with PTSD had significantly smaller amygdala, an area of the brain responsible for coordinating responses to fear. People with PTSD also had significantly smaller anterior cingulate cortices, an area of the brain that is responsible for inhibiting fear and compulsivity. People with PTSD also exhibited smaller frontal lobe problems, which may result in overall difficulties with thought, coordination of behavior, planning, and judgment. Remarkably, people with trauma exposure, regardless of PTSD symptomatology, had significantly smaller hippocampi, a structure that is responsible for memory. The finding for hippocampal atrophy was further

supported in Smith's (2005) meta-analysis. The mean hippocampal reduction was 6.9% left and 6.6% right smaller hippocampal volume for PTSD patients compared with control participants. Given that the amygdala and hippocampus work together to generate selective learning of fear cues as well as extinction of fear (Phelps, Delgado, Nearing, & LeDoux, 2004), it is possible that the deficits associated with these brain regions set the stage for pathological reactivity to emotional stimuli of traumatic and nontraumatic nature.

Physical reactions to cued trauma reminders are also evident in the neuroendocrine domain. One of the body's hormonal stress response systems, the hypothalamic–pituitary–adrenocortical axis (HPA) may become dysregulated following exposure to violence and trauma (Bevans, Cerbone, & Overstreet, 2005; Miller, Chen, & Zhou, 2007). This system is responsible for regulating the production and release of cortisol. Miller et al.'s (2007) meta-analysis of cortisol responses in PTSD examined 107 studies with a total of 8,521 participants. The authors found cortisol levels for trauma exposed individuals were related to timing (i.e., cortisol suppression in the morning, higher levels of cortisol in the evening, and an overall greater daily cortisol output), features of the stressful event, and features of the individual facing the event. Furthermore, the authors found that people with PTSD have blunted cortisol stress responses but higher overall daily cortisol output. The study also found that the HPA axis system can be either activated or downregulated depending on moderating factors such as timing, nature of stress, controllability, and individual psychiatric response. This finding suggests that trauma is related to both too much and too little cortisol, and therefore health outcomes related to both too much and too little cortisol could be traced back to trauma exposure. Most notably, several studies have documented that mere exposure to trauma, in the absence of diagnosable psychopathology, is related to abnormal cortisol activity (de Kloet et al., 2007; Heim et al., 2000). Higher levels of cortisol suppression were found in both trauma-exposed and PTSD individuals when compared with healthy controls (de Kloet et al., 2007). This study was significant in that it demonstrated evidence that trauma exposure (not solely PTSD diagnosis) influences cortisol suppression.

### *Physical Health Impairment in the Absence of Emotional Distress*

Though preliminary, research suggests that trauma exposure may lead to health impairment in *nondistressed* or psychiatrically symptomatic persons. For example, Buckley and Kaloupek's (2001) meta-analysis suggests that trauma-exposed people without PTSD still have elevated cardiovascular arousal. PTSD patients displayed higher blood

pressure and resting heart rates than trauma-exposed and nonexposed controls. However, the difference was greater between the PTSD and nonexposed group than between the PTSD group and the trauma-exposed group without PTSD, suggesting cardiovascular risk in the absence of PTSD diagnosis. Similarly, cortisol studies from Heim et al. (2000, 2001) and de Kloet et al. (2007) found that the trauma-exposed people without PTSD nonetheless had aberrant cortisol responses. Similarly, Brewin et al.'s (2007) meta-analysis finds that trauma-exposed non-PTSD people still show information processing deficits, relative to their nonexposed peers, whereas Karl et al.'s (2006) meta-analysis shows hippocampal atrophy in trauma-exposed people with and without PTSD. These studies have documented that nonpsychiatric trauma-exposed groups have responses between those of nonexposed persons and those with PTSD, suggesting that some factor other than the stress of active PTSD symptoms links trauma exposure to health outcome. Furthermore, this group is not simply a somatization group; whether or not they self-report physiological distress, they manifest with concrete physical markers of disease risk.

Of course, it is possible that this nondistressed group is accounted for by a failure of researchers to examine trauma psychopathology beyond PTSD. Contemporary psychiatry has yet to fully incorporate the full spectrum of trauma reactions into its lexicon, a shortcoming that affects what is measured and therefore what is discovered in research. PTSD's common comorbidities (Kessler et al., 1997) may also be related to physical illness but are not frequently investigated as links in the trauma–psychopathology–health chain. Similarly, PTSD's less investigated comorbidities of dissociation and somatization are forms of psychiatric distress that may affect physical health while disguising psychiatric distress.

But what if we take seriously the idea that there is a group of people with trauma exposure, without psychiatric distress or psychiatric impairment but with concrete physical dysfunction—not just hypersensitivity to interoceptive cues or somatic idioms of distress or hypochondria? In addition to the potential impact of cued reactivity, whether conscious or unconscious, inhibitory emotion regulation styles, such as dissociation (shutting down or cordoning off of distressing emotional and cognitive material), alexithymia (difficulty knowing and describing internal states; Taylor, 1984), suppression (intentional attempts to reduce emotional reactions), and repression, (unintentionally reduced emotional reactions) may generate physical distress. If this group (trauma exposed, psychiatrically asymptomatic, physically at risk) truly does exist, then the development of models to describe them is critical. Fortunately, the groundwork for such models has been laid by experimental research paradigms that examine the physiological consequences of emotion suppression. These ideas might suggest that the

emotional coping techniques used to reduce emotional pain *in and of themselves* create physical stress.

### **Physically Taxing Emotion Regulation Styles**

Numerous studies have documented that emotional suppression is physically taxing. For example, Gross and Levenson (1993; for review, see Gross, 2002) have used paradigms in which participants are instructed to either reappraise or suppress reactions to an emotionally evocative film clip. In the reappraise condition, participants might cognitively reevaluate their reaction (e.g., think to themselves “it’s just a movie, it’s not real”); in the suppress condition, participants are asked to make their feelings “go away” so that an observer would not be able to discern a response. Across multiple replications, participants in the “suppress” condition show increased heart rate. Over time, habitual use of suppression may be physically taxing, leading to deteriorating health.

Although amenable to experimental manipulation, some individuals are “natural” suppressors (Gross & John, 2003). Emotional suppression may be used more readily when cognitive reappraisal or other regulatory strategies fail, as in the case of overwhelming traumatic events. Although suppression generates elevated heart rate, which may increase physical stress, it may also increase stress in other important ways. Emotional suppression is related to memory loss for the suppressed event (Richards & Gross, 2000), increasing the likelihood that important details of salient stressful events remain inaccessible to consciousness and therefore more difficult to address through reflection or psychosocial intervention. Suppression also seems to disrupt emotional relationships: Emotional suppressors are perceived as less likable (Butler et al., 2003); therefore, suppressors may be more socially isolated and alienated from the protective buffer of social support. Finally, suppression leads to an exaggerated startle response (Hagemann, Levenson, & Gross, 2006); in the case of PTSD, suppression and hyperarousal may combine to create a perpetual state of physiological arousal with low reported affect.

Emotional suppression paradigms may help explain how somatization may progress into documentable physical illness. “Somatization,” a coping style dependent on repression, may start out as client-reported physical symptoms without physical markers or emotional distress, but the very process of somatization (i.e., the process of converting something emotional to something physical, in service of keeping psychological distress out of awareness) may be physiologically taxing. In such a scenario, we would expect someone to report no distress, but their body would tell the story of the trauma not immediately but over time. Research on people who

somatize as a defensive style documents that repressive coping leads to sustained sympathetic arousal but low reported affect (Newton & Contrada, 1994).

Along a similar vein, people with alexithymia have well-documented elevated disease risk (e.g., Jorgensen, Johnson, Kolodziej, & Schreer, 1996; Jula, Salminen, & Saarijarvi, 1999). Whether alexithymia is a skill deficit or a defense (e.g., an attempt to avoid or disavow painful affect) remains unclear, though research suggests that both deficit and defense characterize alexithymia (Helmes, McNeill, Holden, & Jackosn, 2008). Thus, people who struggle with reporting emotions are likely to be prone to emotion suppression, further exacerbating their physiological dysregulation while, in interactions with health providers, reporting low or diffuse distress. This emotional style means that alexithymics may be less likely to describe stressors to providers or may be less likely to attribute distress to critical life events—a situation that sets the stage for ongoing physical tolls. Indeed, people with alexithymic traits have greater blood pressure reactions to stress, as well as more body fat (Waldstein, Kauhanen, Neumann & Katzel, 2002), both of which may generate disease. Paradoxically, trait alexithymia predicts the development of PTSD symptoms (McCaslin et al., 2006). Perhaps one explanation for how alexithymia affects health is that trauma exposure creates a triggered reminder that alexithymic people are unable to articulate, though trauma exposure as a moderator between alexithymia and negative health outcomes has not yet been explored. Other trauma-related emotional styles, such as dissociation, may function similarly.

Fortunately, some researchers have found that emotional events can be processed in ways that reduce the physical burden of trauma. Most notably, in one of the most widely replicated intervention paradigms in existence, Pennebaker and Beall (1986; for meta-analysis, see Frisina, Borod, & Lepore, 2004), following the hypothesis that emotion inhibition was physiologically costly, encouraged study participants to write daily about their thoughts, feelings, and reactions to traumatic or stressful events. Even over a short time span, participants showed significant health improvements, varying from decreased doctors’ visits to decreased heart rate to increased T cell production (for review, see Pennebaker, 1997). These findings suggest that people prone to emotional suppression may find relief in structured emotional expression.

### **Review of Affected Systems: Trauma Causes Multiple Types of Acute and Chronic Physical Illness.**

Research has documented specific impacts of trauma exposure and PTSD on numerous body systems: cardiovascular,

immunological, neuroendocrine, reproductive, and gastrointestinal. We will review affected systems here.

**Cardiovascular illness.** In addition to research that examines how acute and long-term stress affects heart rate, there is extensive support linking acute trauma to impairments in other indices of cardiovascular functioning. Witnessing and experiencing a traumatic event leads to long-term changes in systolic blood pressure (Wilson et al., 1998), atrioventricular defects (Boscarino & Chang, 1999), and increased risk for coronary events (Brydon, Magid, & Steptoe, 2006). Stress is linked to sustained high blood pressure levels, which in turn leads to atherosclerotic plaque (McEwen, 2005). In a longitudinal study by Uchino, Holt-Lunstad, Bloor, and Campo (2005), trauma-exposed adults were found to have an increase in systolic blood pressure reactivity and parasympathetic withdrawal in response to acute stress. Acute stress has been found to induce cardiac mast cell activation and elevate serum levels of histamine and IL-6 (interleukin-6), which may indicate higher risk levels for future coronary events (Huang, Pang, Karalis, & Theoharides, 2003; Theoharides & Cochran, 2004). Similarly, changes in posttrauma cardiac functioning have been demonstrated within animal studies. In Geerse, van Gorp, Wiegant, and Stam's (2006) animal research, exposure to a stressful event leads to long-term cardiovascular hyperresponsivity.

Chronic PTSD (when controlling for other factors, e.g., age, cigarette smoking, body mass index, and substance use) has been closely linked with cardiovascular problems, including electrocardiogram abnormalities, atrioventricular defects, and infarctions (Boscarino & Chang, 1999). Altered blood coagulation (hypercoagulability) was linked with PTSD, which may partly explain how PTSD affects the development of cardiovascular disease (von Kanel et al., 2006).

**Immune functioning.** Trauma exposure and stress have a direct consequence on an individual's immune system functioning. McEwen (2005) has suggested that acute stress may immediately enhance the immune system, whereas chronic stress is linked with immunosuppression. Initially, the immediate response to stress activates and elevates the immune system. However, with continued exposure to stress there is evidence of dramatically reduced functioning of the immune system, which leads to vulnerability to disease; similarly, autoimmune disturbance leads to the deterioration of vital organ systems.

Research has examined the mechanisms by which stress affects the immune system. Stress increases the peripheral lymphocytes (natural killer [NK] cells and cytotoxic T cells) during exposure to the stressor; then those immune cells decrease to below baseline levels following the stressor (Breznitz et al., 1998; Delahanty, Wang, Maravich, Forlenza, & Baum, 2000; Dougall & Baum, 2004). Thus, with chronic stress (similar to what is

seen in PTSD), the immune system has a blunted response to any new acute stressors (Baum, Cohen, & Hall, 1993; Dougall & Baum, 2004). For example, T cells do not immediately proliferate following an immediate stressor when exposed to chronic stress. Increased plasma levels of IL-6, IL-1 $\beta$ , and C-reactive protein are found following exposure to acute stress, as demonstrated by a meta-analysis of 30 studies with a total of 1,749 participants (Steptoe, Hammer, & Chida, 2007). Taken together, these results suggest a blunted response to chronic, low-level stress but an exaggerated response to an immediate, acute stressor. These results indicate strong potential risk factors in the development of disease when stress influences inflammatory markers. Elevated C-reactive protein and IL-6 predicts hypertension, coronary heart disease, and cardiac mortality (Albert, Ma, Rifai, Stampfer, & Ridker, 2002; Sesso et al., 2003; Steptoe et al., 2007). Mast cells (immune cells) can be activated by acute stress and play a role in neuroinflammatory diseases, which include migraines, arthritis, cardiovascular disease, interstitial cystitis of the urinary bladder, and irritable bowel syndrome (IBS; Theoharides & Cochran, 2004), suggesting that immune dysfunction may play a role in the development of other posttrauma illnesses. In a study by Kawamura, Kim, and Asukai (2001), the immune system of individuals with PTSD was found to be compromised and linked to long-term health complications. The number of lymphocytes, T cells, NK cell activity, and IL-4 were significantly lower than in non-PTSD individuals.

**Gastrointestinal conditions.** Acute stressful events and psychological stress have been found to be a precipitant to gastrointestinal disorders that affect the entire gastrointestinal tract, including IBS (Farthing, 1995; Fleisher, 1997). Both psychological and physical stressors are associated with altered contractile responses of the colon (Horwitz & Fisher, 2001). Murray et al. (2004) examined the effects of stress on the autonomic outflow to the gut in IBS. In both healthy participants and individuals diagnosed with IBS, acute physical and psychological stress changed their gut-specific autonomic tone and increased visceral sensitivity in IBS participants, suggesting stress as a precipitant of acute IBS attacks. Acute stress has also been found to induce exaggerated arousal and dysrhythmic gastric activity (Muth, Koch, Stern, & Thayer, 1999). A study by Leserman et al. (1996) found that women with assault histories were more at risk for IBS and severe organic gastrointestinal disorders. Furthermore, psychological stress also influences the motor function in the small bowel in patients with and without IBS (Horwitz & Fisher, 2001; Snape, Carlson, Matarazzo, & Cohen, 1977). According to Horwitz and Fisher (2001), there is a higher risk for comorbid disorders, including fibromyalgia and interstitial cystitis, if a patient is diagnosed with IBS, leading to a cumulative stress burden.



PTSD diagnosis following trauma exposure is also a predictor for the development of gastrointestinal disorders, including ulcers and IBS (Grahn, Kalman, Brennan, Watkins, & Maier, 1995; Stam, 2007; Weisberg et al., 2002). In a study by Irwin et al. (1996), a diagnosis of PTSD frequently preceded the development of IBS for individuals seeking medical services, which shows a potential association between development of PTSD and gastrointestinal complications. Seng, Clark, McCarthy, and Ronis (2006) found that in a sample of more than 16,000 women, the risk for developing IBS was 4.7 times greater when an individual is diagnosed with PTSD without a co-occurring psychiatric diagnosis and higher when psychiatric comorbidities were present.

**Reproductive disorders.** Although less investigated than other associations between trauma and physical health, the evidence on the association between trauma and reproductive disorders is consistent. Trauma has been linked with chronic pelvic pain, sexual problems, infertility and miscarriage, preterm delivery, and low birth weight of one's children. Walker, Gelfand, Gelfand, and Koss (1995) found that histories of trauma were common in women with chronic pelvic pain. Farley and Patsalides (2001) interviewed 600 adult women and found an increased incidence of reproductive symptoms in women with a history of trauma. Spinhoven et al. (2004) found that physical assaults were related to chronic pelvic pain. Matsubayashi, Hosaka, and Makino (2006) examined the role of distress in infertility. They found that women experiencing significant psychological distress had a higher level of a type of immune cells called "natural killer" (NK) cells. Elevated NK cell activity may expel an embryo from the uterus. Elevated NK cell counts are associated with both traumatization and recurrent pregnancy loss.

In a large sample of women, Seng et al. (2006) found that PTSD diagnosis was associated with increased menstrual pain, endometriosis, dyspareunia, and cervical dysplasia. Increased incidence was above and beyond exposure to victimization and appeared to be linked particularly to psychiatric diagnosis. Rogal et al. (2007) analyzed hospital records of 1,100 women. Preterm delivery was 2.8 times more likely in women with PTSD than women without, whereas low birth weight was 1.82 times more likely in women with PTSD than without.

**Musculoskeletal and pain disorders.** Trauma increases the risk for fibromyalgia (Haviland, Morton, Oda, & Fraser, 2010) and musculoskeletal disease (McFarlane, Atchison, Rafalowics, & Papay, 1994). Dorn, Yzermans, Spreeuwenberg, Schilder, and van der Zee (2008) found that in a sample of accident survivors, musculoskeletal problems were elevated relative to community controls. Schnurr, Spiro, and Paris (2000) found that among 605 veterans, PTSD symptoms were related to increased musculoskeletal disorders even when controlling for other

health indicators such as age, smoking, alcohol use, and body weight. Farley and Patsalides (2001) surveyed 600 women and found increased musculoskeletal symptoms in those with a trauma history. Seng et al. (2006) found that PTSD without co-occurring psychiatric diagnoses had an odds ratio for fibromyalgia of 1.9 compared with people with no trauma, though victimization alone was associated with an odds ratio of 2.2. Roy-Byrne, Smith, Goldberg, Afari, and Buchwald (2004) examined 571 fibromyalgia patients and found that pain severity was associated with PTSD symptom severity, particularly when patients had co-occurring depression. Amital et al. (2006) found that in a study of 124 men, nearly half of PTSD patients had fibromyalgia, whereas no participants without psychiatric diagnoses had fibromyalgia.

In an examination of the potential coheritability of PTSD and fibromyalgia, Arguelles et al. (2006) examined PTSD symptoms and fibromyalgia in 1,800 twin pairs. They found that PTSD and fibromyalgia were strongly related but that the relationship between PTSD symptoms and fibromyalgia was not stronger in identical versus non-identical twins. Therefore, though fibromyalgia is strongly linked to PTSD, the association is not through a common genetic vulnerability but possibly through the effects of chronic stress on physical and mental health.

**Cortisol abnormalities as a mediating factor.** As we have already reviewed, the HPA axis becomes dysregulated after trauma. It is possible that this system plays a key role in disrupting other organ systems. HPA system dysfunction is theorized to be a primary biological mechanism in the pathway from stress to adverse health outcomes (De Bellis, 2001; Friedman & McEwen, 2004); in particular, elevated cortisol (released by the HPA axis) appears to be a cause of hippocampal degeneration (Armanini, Hutchins, Stein, & Sapolsky, 1990). The HPA axis mediates the hormone release of glucocorticoids (necessary for glucose metabolism), vasopressin (necessary for water reabsorption by the kidneys), and oxytocin (necessary for anxiety reduction and important in social relationships). Repeated elevation of levels hormones from the HPA axis predisposes an individual to disease (McEwen, 2005; McEwen & Wingfield, 2003). An increase in glucocorticoids results may result in insulin resistance and a higher risk for obesity, cardiovascular disease, and stroke (McEwen, 2005), suggesting that neurohormonal adaptations to trauma may point toward mediators that maintain the trauma burden.

## Conclusions

### Summary

In conclusion, this review of literature supports the assertion that acute trauma exposure leads to negative physical

health consequences. These consequences increase with chronicity of environmental exposure as well as with psychiatric distress. The majority of the body's systems are adversely affected by trauma. Specifically, there is a significant disruption to gastrointestinal functioning, the cardiovascular system, immunological functioning, the reproductive system, the musculoskeletal system, neuroendocrine functioning, and finally brain structure and functioning. Not only are these systems broadly affected by symptoms, but the risk factors for development of future medical disease are exponentially greater following exposure to trauma.

To add to the physical impact of trauma, the psychological consequences are equally devastating. It is noted throughout the literature that individuals exposed to trauma frequently experience comorbid disorders, such as anxiety, depression, and PTSD. The psychological impacts of trauma are long term and treatment resistant. Furthermore, the psychological impact of trauma imparts further risk and exacerbation of physical illness. Functioning is significantly impaired because of the increased physiological arousal with external and internal cues or triggers. Thus, it is frequently difficult for an individual to return to baseline activities (including social and occupational functioning). Taken together, the extant literature demonstrates that trauma leads to chronic impairment in one's psychological and physical functioning.

The data reviewed here suggest several pathways to health problems following exposure to trauma: (1) Conscious or nonconscious reactivity to triggered reminders chronically elevates the body's stress response and (2) suppressive emotion management techniques generate a physical strain that chronically elevates the body's stress response.

### **Limitations and Future Research Directions**

*Limitations of the current review.* Although this review was written with the spirit of thoroughness, it is not comprehensive or complete. The goal of the current review is to describe a chain of reasoning supported in the literature that links trauma and physical illness; however, it does not give an estimate of the magnitude of such a relationship, as one might find in a meta-analysis. Rather, it is written with two aspirations in mind: first, that it will be useful in generating further, more specific research adding to an extant literature that has significant merits; and second, that it will be a useful guide to practicing clinicians. Nonetheless, it is an appropriate critique of this review to note that it does not accomplish the same goal as a systematic investigation such as a meta-analysis.

*Limitations of the literature.* One weakness in the current literature is that it has inadequately addressed the impact of developmental factors. Much of the research on adult

health outcomes is limited to (1) adults in middle age or younger and (2) adults who self-report both trauma and physical distress. Little is known about the impact of trauma in geriatric samples. Similarly, it is possible that adults who perceive life events as traumatic are likely to exaggerate health problems, though research has attempted to address this concern (e.g., Boscarino, 1997; Schnurr et al., 1999; Schnurr & Spiro, 2000). Conversely, it is possible that adults who are not experiencing psychiatric distress due to a traumatic event may fail to report early life traumas, thereby diminishing the magnitude of impact that trauma may have on physical health. Therefore, prospective research that uses substantiated reports of traumatic stressors would lend strength to this body of literature.

A second weakness of the current literature is that causal mechanisms between acute traumatic stressors and health outcomes would benefit from further elaboration. Specifically, research that examines the details of which organ systems are likely to be affected for a given individual would add predictive power as well as clinical significance to this literature. As it stands, most research on mechanisms occurs within a "silo" of research expertise: neuroanatomists, endocrinologists, and immunologists work independent of one another. Given that a link between trauma and multiple physical illnesses is well-established, collaboration across subspecialties may enhance clarity in the field. Similarly, given that research has solidly established a link between exposure to traumatic stress and physical illness *in the absence of a PTSD diagnosis*, pan-diagnostic research that examines mechanism instead of DSM diagnosis could enhance understanding. Such investigations are in line with current efforts to examine research domain criteria (Insel et al., 2010).

*Implications for health professionals.* How might this information be helpful for health professionals, such as psychiatric nurses, social workers, and psychotherapists? First, we believe that raising awareness of the health impact of acute trauma will help focus health professionals' attention on the antecedent event: overwhelming stressful experiences. When a patient has multiple diagnoses, or unexplained diagnoses, either acute or chronic trauma may have set the disease wheel in motion. Therefore, working to overcome the emotional impact of the trauma—either through trauma-focused interventions or through stress reduction—may be a "first line of defense" in chronic illness. This knowledge may empower clinicians to use "minimally invasive" treatments such as mindfulness and yoga as their recommendations. Interventions that offset the impact of emotional inhibition, such as expressive writing, may be particularly helpful. Communicating the impact of stress on the body to patients in a detailed way may help patients place more value on stress reduction in their lives.

Taking a different vein, these data may help psychiatric nurses appreciate the multiple health burdens associated with trauma and facilitate a shift in perception of patients with multiple health problems. Such patients may use tremendous health resources and be at risk for being dismissed as histrionic. However, their histrionic expression may cover significant and long-ignored distress. An awareness on the behalf of a clinician that a client with multiple health problems may be, at the root, a trauma patient may lead to a shift in how clinicians interact with their clients. Even the most patient clinician may become frustrated with someone whose problems seem insurmountable. However, evidence from other fields suggests that treating underlying trauma can lead to amelioration of symptoms that had not been previously recognized as trauma symptoms (e.g., Morrissey et al., 2005; Pavuluri et al., 2006). Recognizing trauma and increasing one's facility in working with traumatized clients may lead to increased efficacy, reduced work burden, and increased job satisfaction. Above all, however, becoming a trauma-informed clinician who works at the intersection of physical and mental health is providing the best care for the numerous patients with trauma histories who present in a variety of physical and mental health settings.

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### Author Roles

Dr. Spinazzola: Overview of trauma  
 Drs. D'Andrea and Sharma: Review of Affected Systems  
 Dr. Zelechowski: Implications  
 Dr. D'Andrea: All other sections

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