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An Enduring Somatic Threat Model of Posttraumatic Stress Disorder Due to Acute Life-Threatening Medical Events

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Abstract

Posttraumatic stress disorder (PTSD) occurs in 12-25% of survivors of acute life-threatening medical events such as heart attack, stroke, and cancer, and is associated with recurrence of cardiac events and mortality in heart attack survivors. This article reviews the current state of knowledge about PTSD after such events, and proposes an Enduring Somatic Threat (EST) model of PTSD due to acute life-threatening medical events to address underappreciated differences between PTSD due to past, discrete/external traumatic events (such as combat) and PTSD due to acute manifestations of chronic disease that are enduring/internal in nature (such as heart attack and stroke). The differences include the external versus internal/somatic source of the threat, the past versus present/future temporal focus of threatening cognitions, the different types and consequences of avoidance behavior, and the different character and consequences of hyperarousal. Although important differences between the two types of PTSD exist, the EST model proposes that the underlying fear of mortality maintains PTSD symptoms due to both discrete/external and ongoing/somatic events. Finally, this article offers a research agenda for testing the EST model, with a particular focus on areas that may improve cardiovascular prognosis and health behaviors in survivors of heart attack and stroke.

I have never been the same since. I suffer from extreme anxiety, hyper vigilance and a host of other psychological symptoms related to my cardiac history. I am hyper aware of every extra or missed heart beat which brings me right back to the original arrhythmia. I struggle on a daily basis to do the most basic things.

Sophia, NYC, Comment in response to “Heart Attack Survivors May Develop PTSD,” Tara Parker-Pope, *New York Times*, June 20, 2012.

Posttraumatic stress disorder (PTSD) is an anxiety disorder initiated by an exposure to a discrete traumatic event that has generally occurred in the past, such as combat, disaster, or sexual assault, and is characterized by symptoms such as re-experiencing (e.g., intrusive thoughts, nightmares, flashbacks), cognitive or behavioral avoidance of reminders of the event (e.g., avoiding thinking about the event, avoiding places or people associated with the event), and physiological hyperarousal (e.g., difficulty sleeping, heightened startle reflex). It is associated with abnormal amygdala, prefrontal cortex, and hippocampal function (Shin,

Rauch, & Pitman, 2006) as well as abnormal neuroendocrinologic characteristics (Yehuda, 2006). In the Diagnostic and Statistical Manual of Mental Disorders (Fourth Edition; *DSM-IV*) (American Psychiatric Association, 1994), life-threatening illness was recognized as an event that can elicit PTSD, however, important differences between PTSD due to discrete past events and PTSD due to ongoing medical illness have not been fully appreciated.

The potential for PTSD due to life-threatening illness has been studied most in cancer, acute coronary syndrome (ACS; myocardial infarction or unstable angina), and increasingly, stroke patients. While individual studies offer prevalence estimates for medically-induced PTSD that vary widely, from 0% to more than 30%, large observational studies and meta-analytic estimates within each literature have returned fairly consistent estimates of approximately 12-25% (Edmondson & Cohen, 2013; Edmondson et al., 2012; Edmondson, Richardson, et al., 2013). The implications of that estimate have not yet been appreciated by patients, clinicians, or the health care system, and the popular conception of what PTSD is -- and who is at risk -- has lagged even farther behind.

Today in the U.S., there are 21 million veterans and about 1.5 million active duty service people (United States Department of Defense, 2013). Only a proportion of those have seen combat and are thereby vulnerable to combat-induced PTSD, though the nature of "combat" exposure in the current conflicts is difficult to quantify. By comparison, according to the American Heart Association, there are currently more than 17 million survivors of acute manifestations of cardiovascular disease in the U.S. alone, with 1.2 million more added each year, all of whom have survived a life-threatening acute medical event (Roger et al., 2012).

The health consequences of PTSD due to acute life threatening illness are substantial. In survivors of cancer or stroke, the development of PTSD is associated with poorer physical functioning, and mental and physical quality of life (Goldfinger et al., 2013; Shelby, Golden-Kreutz, & Andersen, 2008). In acute coronary syndrome (i.e., myocardial infarction or unstable angina) patients who develop PTSD due to the event, they are at double the risk of ACS recurrence and mortality in the subsequent three years relative to their counterparts who do not develop PTSD (Edmondson et al., 2012). A number of mechanisms likely explain these associations, including sleep disruption (Shaffer, Kronish, Burg, Clemow, & Edmondson, 2013), poor medication adherence (Kronish, Edmondson, Goldfinger, Fei, & Horowitz, 2012; Shemesh et al., 2001), autonomic imbalance (Gander & von Kanel, 2006), and increased systemic inflammation (von Kanel et al., 2010).

In all of the studies of PTSD due to a medical event or diagnosis, PTSD has been measured using traditional PTSD screening instruments or clinical interviews keyed to a single event in the past (i.e., the diagnosis and treatment of cancer or the experience of myocardial infarction or stroke). However, a number of researchers have questioned whether PTSD due to a medical event may have features that diverge conceptually and experientially from traditional models of PTSD. For example, the re-experiencing symptoms of PTSD due to a medical illness are often focused on enduring threats of recurrence and functional decline rather than on a discrete event in the past whose danger has passed, and the avoidance, negative alterations in cognition or mood, and hyperarousal symptoms may present in a different manner and with different psychological, behavioral, and health consequences

(Green et al., 1998). However, while the phenomenon currently (and perhaps rightly) labeled PTSD due to acute medical illness does not fit neatly into the traditional PTSD framework, it is also substantially distinct from other somatic fear-based phenomena such as hypochondriasis or somatic anxiety—neither of which are rooted in an identifiable traumatic trigger or have present a substantiated threat of mortality.

This article reviews the current state of knowledge about PTSD after such events, and proposes an Enduring Somatic Threat (EST) model of PTSD due to acute life-threatening medical events to address underappreciated differences between PTSD due to past, discrete/external traumatic events (such as combat) and PTSD due to acute manifestations of chronic disease that are chronic/somatic in nature (such as heart attack and stroke). The differences include the external versus somatic source of the threat, the past versus present/future temporal focus of threatening cognitions, the different types and consequences of avoidance behavior, and the different character and consequences of hyperarousal. Although important differences between the two types of PTSD exist, the EST model proposes that the underlying fear of mortality maintains PTSD symptoms due to both discrete/external and ongoing/somatic events. As can be seen in Table 1, a number of diagnostic criteria must be met to be diagnosed with PTSD.

Scope

EST was developed to explain the maladaptive psychological and behavioral sequelae that often follow life-threatening medical illnesses that are currently classified as symptoms or correlates of PTSD. The most relevant phenomena are those psychological sequelae whose features diverge from classical conceptions of PTSD symptoms or correlates, and those behavioral sequelae with unique features or consequences in survivors of life-threatening illness. Finally, EST proposes an underlying commonality that may give rise to both classical presentations of PTSD and the unique presentation of PTSD-like sequelae of life-threatening medical events.

Propositions

The following three propositions draw on quantitative and qualitative investigations of medically-induced PTSD, and on existential social psychological and behavioral medicine theories of PTSD and health behavior, including cognitive worldview models of PTSD (Dalglish, 2004), the Terror Management Health Model (Goldenberg & Arndt, 2008), and Anxiety Buffer Disruption Theory (Edmondson, Chaudoir, et al., 2011; Pyszczynski & Kesebir, 2011).

Proposition 1: Event Dimensions. There exist at least two distinctions between discrete/external traumatic events and medical traumas. First, discrete/external traumas are represented psychologically as having occurred in the past while medical traumas are perceived as ongoing events of somatic origin that are recalled whenever attention is directed toward the body. Second, discrete/external traumas are directed at the individual from the outside while medical traumas reside within.

Proposition 2: Character of Symptoms. PTSD symptoms due to medical events differ from those due to discrete/external traumatic events due to their divergent temporal focus, the nature and consequences of avoidance behaviors, and the relative necessity of vigilance to signals of threat.

Proposition 3: Source of Distress. While the dimensions of the triggering event and the character of PTSD symptoms differ between discrete/external trauma events and ongoing medical traumas, the underlying source of distress for both is the terrifying awareness of mortality that is made conscious by traumatic events of either type.

Proposition 1: The uniqueness of medical trauma as a triggering event

Life-threatening medical illness possesses all of the hallmarks of a potentially PTSD-inducing event, but differs in important ways from discrete/external past traumatic events (Smith, Redd, Peyser, & Vogl, 1999). The term *life-threatening* implies satisfaction of Criterion A1, and though Criterion A2 (see Table) has been abandoned in DSM-V, research has shown that many survivors report intense fear, helplessness, or horror (Palmer, Kagee, Coyne, & DeMichele, 2004). Unlike the prototypical traumatic events such as combat or sexual assault that have long been known to induce PTSD symptoms, however, medical traumas are rarely discrete events with a defined endpoint. Once a discrete/external event has concluded, the physical threat has usually subsided. Although a combat veteran who experiences traumatic violence may thereafter develop a heightened awareness of the *possibility* of non-combat violence in the world, she recognizes that the event that triggered her PTSD symptoms is over, and that safe places exist wherein the threat of future violence is exceedingly unlikely. In contrast, traumatic life-threatening illness is often the acute manifestation of a permanently disrupted physiological system whose consequences may last for years and place an ongoing threat squarely in the body of the survivor, from whence no safe haven exists (Fox et al., 2006; Goldberg et al., 2004; Tang, Wong, & Herbison, 2007).

For example, the cancer experience often begins with suspicious test results, and continues through cancer detection and diagnosis, and treatment that may be long-lasting, invasive, and painful. Following the completion of active treatment and recovery, many patients must continue to undergo regular oncologic check-ups for many years. Although cancer recurrence rates vary, they are substantial for many forms (Baker, Denniston, Smith, & West, 2005; Smith et al., 1999). Thus, the traumatic character of the cancer experience does not reflect the horror of a discrete event, but rather arises from an often years-long experience of vulnerability, pain, and fear. Further, the nature of the disease is such that continued vigilance to threat is both statistically rational and prescribed by medical authorities.

Similarly, acute coronary syndromes and strokes are acute indicators of chronic disease that recur with great frequency (Ay et al., 2010; Fox et al., 2006; Goldberg et al., 2004; Jørgensen, Nakayama, Reith, Raaschou, & Olsen, 1997; Pedersen, van Domburg, & Larsen, 2004; Sacco, Wolf, Kannel, & McNamara, 1982; Tang et al., 2007), and require chronic disease management and continued daily vigilance to physiological markers of

cardiovascular functioning and life-long adherence to medication regimens (Redfern, Briffa, Ellis, & Freedman, 2009; Smith Jr et al., 2011; Vedin et al., 2012).

ACS patients often report include intense fear (Bennett, Owen, Koutsakis, & Bisson, 2002), perceived life threat, lack of control, (Doerfler, Paraskos, & Piniarski, 2005) helplessness and chest pain during the ACS event (Wiedemar et al., 2008), and those factors are associated with increased risk for PTSD, which is present in 1 out of every 8 ACS survivors (Edmondson et al., 2012). These types of peri-traumatic factors (fear, perceived control, pain) are also highly predictive of subsequent PTSD symptoms due to discrete/external events. Although very few studies of medically-induced PTSD have assessed less traditional perceptions of the medical event as an ongoing somatic threat, those that have done so find that perceptions of a heart attack as central to the patient's identity, that the event is more chronic than acute and has major consequences for the future, as well as negative emotional representations of the illness are strongly predictive of subsequent PTSD symptoms (Sheldrick, Tarrier, Berry, & Kincey, 2006). Such results suggest an important role for the perception of an enduring somatic threat in medically-induced PTSD. Currently, little is known about the long-term course or severity of PTSD due to cardiovascular events. Wikman and colleagues (Wikman, Bhattacharyya, Perkins-Porras, & Steptoe, 2008) found that 12% of 213 participants screened positive for ACS-induced PTSD at 12 months post-ACS, and that 12.8% of 179 of those participants screened positive for ACS-induced PTSD at 36 months. However, while PTSD symptoms were relatively stable, there were participants who screened positive at 12 months who did not at 36 months, and vice versa, as only 63% of the variance in 36 month PTSD symptoms were explained by 12 month PTSD symptoms. In a different study with the longest follow-up to date, Ginzburg & Ein-Dor found that half of those who developed PTSD initially still screened positive 8 years later (Ginzburg & Ein-Dor, 2011). Thus, we require more information about the incidence, prevalence, and course of PTSD symptoms due to acute, life-threatening medical illnesses to understand if these symptoms are similar to, or diverge from the course taken by symptoms due to more traditionally conceived traumas.

Stroke is among the most frightening of acute medical events, as it often results in sudden and often progressive loss of bodily control and cognitive function. In one of the first qualitative investigations of the subjective experience of stroke, stroke survivors interviewed within 72 hours of stroke onset consistently described the terror of being awake and alert as the affected arm and leg became weaker, often ending in complete paralysis of the entire side of the body (Doolittle, 1991). The feelings most often cited in the acute phase were shock, extreme fear, and desperation, and these feelings persisted throughout patients' hospitalization, due to their uncertainty about continued loss of bodily control or recovery of function over the first few days as the stroke evolved. Further, survivors described intense disillusionment in learning that the hospital could not halt or reverse the damage caused by the stroke, as the limits of technology and medical control became obvious. Finally, fear of recurrence, impaired social functioning, and substantial physical disability contributed to patients' profound feelings of loss of control over the self and future. In another study that followed patients over the 12 months following their stroke, patients described the fear of dying that accompanied the stroke, as well as the inescapability of the stroke's bodily effects and the near complete lack of control they experience months after the stroke (Burton,

2000). These qualitative data suggest that the experience of a stroke is initially traumatic, but that the lasting psychological damage is strongly associated with the ongoing consequences of the event. Among stroke survivors screened within the first year after the stroke, a recent meta-analysis suggests that as many as 1 in 4 screen positive for PTSD, and among those screened after the first year, 1 in 8 screen positive (Edmondson, Richardson, et al., 2013).

The desired outcome for most survivors of discrete/external, past, non-medical traumas is to diminish the emotional tenor of the memory of the discrete traumatic event and relegate that event to a distinct moment in the past. In contrast, after diagnosis of cancer or the experience of an ACS or stroke, patients are required to make the occurrence of the event a primary driver of many of their day to day activities. That is, they are asked to actively monitor and combat an ongoing threat with lifestyle changes, medication regimens, and interactions with the medical system—all of which are potent reminders of ongoing vulnerability, and of the original trauma. Such reminders can be highly distressing (Whitaker, Watson, & Brewin, 2009), and have been associated with poor psychological adjustment in both cancer survivors and cardiovascular disease patients (Edmondson, Park, Chaudoir, & Wortmann, 2008; Matsuoka et al., 2002).

Further, for discrete non-medical traumas, the trauma confronts the individual from the outside, so that in its aftermath the individual can find havens of safety away from the threatening context in which to recover and begin reintegrating into former life patterns. However, for survivors of life-threatening illness, the source of the threat is within the body itself, and as such is ever-present.

The temporal distinction between discrete events in the past and ongoing somatic threats has led some researchers to question the conceptual fit of PTSD in medical populations (Anja Mehnert & Koch, 2007; Mundy & Baum, 2004), as PTSD prevalence estimates in studies of cancer patients are substantially lower when present- and future-oriented intrusive thoughts or nightmares are not allowed to fulfill Criterion B (reexperiencing) (Green et al., 1998). However, other research on the factor structure of responses to PTSD symptom inventories has suggested that PTSD symptoms in medical populations tend to conform to DSM-IV symptom clusters (Cordova, Studts, Hann, Jacobsen, & Andrykowski, 2000; DuHamel et al., 2004). Since investigations of PTSD due to medical events began in earnest first in cancer survivors and the traditional conceptualization of PTSD was generally accepted, subsequent research in ACS and stroke patients has tended to ignore temporal distinctions in symptom expression and treat medically-induced PTSD as if the medical trauma were a discrete past event (Edmondson et al., 2012; Edmondson, Richardson, et al., 2013). The EST model proposes a fundamental distinction between traumatic discrete/external events and ongoing/somatic ones that may have important consequences for the identification of PTSD (or PTSD-like disruptions) in survivors of acute life-threatening medical events. That distinction may also have consequences for the character and consequences of PTSD symptoms after acute life-threatening medical events.

Proposition 2: Unique symptom features of medically-induced PTSD

PTSD can be usefully thought of as an overgeneralization of an intense fear response to a conditioned stimulus, and a failure of fear extinction once the actual threat has subsided. This model for understanding PTSD due to discrete/external, past events has proven highly useful for developing successful psychotherapy approaches based on fear extinction paradigms (eg., prolonged exposure). However, the fear extinction paradigm—at least for exposure-based PTSD treatments—is predicated on the notion that the threat itself has passed, and that the diminished fear response to traumatic cues represents a match between the true nature of the threat and the individual's cognitive orientation to it. That is, exposure-based approaches seek to situate the traumatic event within a defined temporal-spatial context that is removed from the individual's present context so that he or she can adopt a more adaptive cognitive stance toward what is understood as a psychologically uncomfortable but physiologically benign *memory of the past*.

In contrast, PTSD due to medical events represents an exaggerated and maladaptive fear response to a valid and ongoing threat to an individual's existence. Whereas successful extinction of the fear response to a past discrete/external event rightly places the threat of mortality in the past so that the individual can resume a normative attitude toward death as something that will occur somewhere in the distant future, modulation of the fear response to an ongoing/somatic threat must acknowledge that the threat of mortality persists. Indeed, the lifelong continued medical maintenance of secondary risk is prefaced on the fact that the acute medical event signaled an ongoing vulnerability within the body itself that must be monitored. This primary distinction between PTSD due to discrete/external, past events and ongoing/somatic ones is apparent in the unique character of PTSD symptoms due to medical events.

Reexperiencing symptoms

Diagnostic criteria for PTSD specify that re-experiencing symptoms are keyed to “memories.” Since the vast majority of research on medically-induced PTSD has been conducted from the traditional discrete traumatic event perspective, little is known about the character of ongoing present and future-oriented “reexperiencing” symptoms such as intrusive thoughts and nightmares. However, studies that have reported qualitative data on present- and future-oriented symptoms suggest that the acute medical event (ie., cancer diagnosis) is not cognitively distinct from ongoing concerns about present and future threats implied by the event, even if assessed (?) years later (Matsuoka et al., 2002). Indeed, fear of cancer progression, physical symptoms, doctors appointments, and other future-oriented concerns have been shown to be strongly correlated with past-oriented intrusion symptoms in breast cancer patients (A. Mehnert, Berg, Henrich, & Herschbach, 2009). In another study, in which anxious cancer patients were asked to identify their most troubling intrusive thoughts, 81% of intrusions reported were future oriented (e.g., I am going to die, the cancer will not go away) (Whitaker et al., 2009). In our research, we have found that a substantial proportion of cancer survivors (mean time since treatment end was 2 years) still think about cancer at least once a day (47%) and many think about it multiple times a day (14%). In that study, participants who thought most about cancer on a daily basis reported almost a full

standard deviation worse mental health quality of life (SF-12 MCS) relative to those who reported rarely thinking about it (Edmondson, Park, Blank, Fenster, & Mills, 2008; Park, Edmondson, Fenster, & Blank, 2008).

Aside from the above distinctions between the character of re-experiencing symptoms due to discrete/external past events versus that of re-experiencing symptoms due to acute life-threatening events, there is some evidence that the consequences of re-experiencing symptoms may themselves be differentially life-threatening for survivors of some types of acute life-threatening medical events. In a study of 247 ACS patients, we found that 11% screened positive for ACS-induced PTSD. Although screening positive for PTSD at 1 month post-ACS was not itself a statistically significant independent risk factor for a recurrent ACS event or mortality, participants who reported high re-experiencing symptoms were at more than 3-fold increased risk for recurrent ACS or mortality in the 42 months after their index ACS event (Edmondson, Rieckmann, et al., 2011).

This finding highlights the strong link between distressing psychological symptoms and the activity of the cardiovascular system—which is driven largely by the autonomic nervous system. Other research has found that intrusions are related to elevated systolic blood pressure, blood markers of endothelial dysfunction (von Känel et al., 2008), and higher circulating levels of both catecholamines (Hawk, Dougall, Ursano, & Baum, 2000) and C-reactive protein (Miller, Sutherland, Hutchison, & Alexander, 2001), all of which are important markers of cardiovascular risk that are more consequential for survivors of acute cardiovascular events, at least in the near term, than for survivors of discrete/external past events without cardiovascular disease.

Avoidance symptoms

The avoidance cluster of PTSD symptoms, like the other clusters, was observed first in survivors of discrete/external traumatic events. In the case of combat veterans, survivors may avoid thinking or talking about their combat experiences, may avoid loud noises or outdoor environments that serve as reminders of the combat zone in which they served, or avoid friends they made in the armed services. Although these strategies may not be successful in keeping distressing thoughts at bay, and often represent a significant detriment to quality of life, their continued use suggests that they provide negative reinforcement. Symptoms of avoidance in patients with PTSD due to acute life-threatening illnesses that indicate an ongoing somatic threat are more difficult to conceptualize. For survivors of heart attack or stroke, how does a person avoid an awareness of an irregular heart beat or slightly elevated blood pressure?

A year before DSM-IV made life-threatening illness a qualifying event for PTSD, Shalev and colleagues described the phenomenon in 4 patients, and noted that PTSD was associated with avoidance of future medical treatment (Shalev, Schreiber, Galai, & Melmed, 1993). Since then, a growing number of studies have shown that PTSD symptoms are associated with nonadherence to secondary prevention medications in both ACS (Shemesh et al., 2004) and stroke survivors. For example, stroke survivors with PTSD symptoms suggestive of likely PTSD diagnosis were almost 3 times as likely to be nonadherent to their prescribed

medications as those without PTSD symptoms years after the index stroke event (Kronish et al., 2012). Another study from that cohort of stroke survivors found that the association of PTSD to nonadherence was mediated by increased concerns about medications (ie., they disrupt my life, fear of side effects), but not their understanding of the necessity of those medications for secondary prevention. That finding suggests that nonadherence to medications may represent a deeper ambivalence created by conscious or unconscious associations between medications and the event itself (Edmondson, Horowitz, Goldfinger, Fei, & Kronish, 2013). EST posits that PTSD due to life-threatening medical events is associated with nonadherence to medical treatment because such treatments serve as potent reminders of the initial event and ongoing somatic threat represented by their chronic disease.

Hyperarousal symptoms

Hypervigilance to threat cues, difficulty sleeping, and irritability are the prototypical hyperarousal symptoms of PTSD, and are relatively common in survivors of discrete/external traumatic events. However, little research has focused on hyperarousal symptoms in medically-induced PTSD. Anecdotal data (see the opening quote of this article) suggest that whereas hyperarousal symptoms due to discrete/external events are distressing physiological responses to powerful memories, hyperarousal symptoms due to medically-induced traumas are both focused on – and contributory to -- somatic cues of threat. PTSD due to ACS or stroke is the best illustration of the unique character of hyperarousal symptoms in medically-induced PTSD, because of the positive feedback loop between anxiety and sympathetic/cardiovascular activity such as increased heart rate and blood pressure. When a heart attack survivor is hypervigilant to cardiovascular indicators of threat, the anxiety caused by any fluctuation in those highly variable indicators itself causes increased sympathetic and thus cardiovascular activity.

Not only are the cardiovascular cues that may signal threat to a heart attack or stroke survivor ubiquitous, but hyperarousal symptoms may actually influence future cardiovascular risk. For example, research by von Kanel and colleagues have shown that PTSD symptoms are associated with increased inflammation – an important risk factor for cardiovascular events-- in survivors of myocardial infarction (von Kanel et al., 2010), likely due to increased autonomic activity. Further, sleep difficulties associated with hyperarousal in PTSD (Shaffer et al., 2013) may themselves be predictors of adverse cardiovascular outcome in survivors of acute cardiovascular events (Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011).

Proposition 3: Fear of death underlies both past and future oriented symptoms

The defining characteristic of traumatic events is that they force a psychological confrontation between an individual and mortality. Some cognitive/worldview models of PTSD and social psychological theories such as terror management theory consider the fear of mortality the primary driver of anxiety (Dalgleish, 2004). Such theories suggest that

humans collectively rely on worldviews to assuage the fear of death, but that those worldviews falter in the face of traumatic events (Janoff-Bulman, 1989).

For example, according to “shattered assumptions” theory (Janoff-Bulman, 1992), individuals develop fundamental, yet unarticulated, assumptions about the world and themselves (i.e., worldviews) that allow for healthy human functioning. The most important assumptions include beliefs in a just, benevolent, predictable world in which the individual possesses competence and worth. The worldview’s primary function is to provide the individual with meaning, self-esteem, and the illusion of invulnerability. Both worldview models of PTSD and terror management theory (TMT) suggest that worldviews provide people with a sense of meaning, structure, purpose, and invulnerability (Solomon et al., 1991). The profound awareness of mortality that accompanies traumatic events has the potential to incite psychological “terror” if individuals are unable to suppress thoughts about their own inescapable mortality through redoubled investment in worldviews that promise literal or symbolic immortality (Pyszczynski & Kesebir, 2011).

Anxiety buffer disruption theory combines two theoretical traditions (experimental investigations of the cognitive and behavioral consequences of mortality awareness and clinical investigations of PTSD), and has quickly accumulated support showing that individuals with clinically significant PTSD symptoms are unable to suppress death-related thoughts (Edmondson, Chaudoir, et al., 2011), and that the degree to which death-related thoughts are cognitively accessible predict chronic PTSD symptoms. For example, Abdollahi and colleagues showed that earthquake survivors with no PTSD symptoms showed evidence of normal worldview defense (i.e., defending culturally important components of worldview -- a marker of a functional, mortality-buffering worldview) after reminders of mortality or reminders of the earthquake, while those with clinically significant PTSD symptoms showed no such worldview defense (Abdollahi, Pyszczynski, Maxfield, & Luszczynska, 2011). Most compelling, however, were the findings that the relationship between initial psychological response to trauma and PTSD symptoms 2 years later was partially mediated by decreased worldview defense (Abdollahi et al., 2011).

For all of these existential, worldview-based theories, traumatic events force survivors to confront, with a newfound immediacy, the fact that their “self” and their body are one and the same. While the destruction of intuitive dualism, and the awareness of mortality that accompanies it, are problematic for many trauma survivors, those who survive acute life-threatening medical events that imply enduring somatic threat are particularly vulnerable. In documenting his death from cancer in the memoir *Mortality*, Christopher Hitchens sums up the conflict.

Always prided myself on my reasoning faculty and my stoic materialism. I don't have a body, I am a body. Yet consciously and regularly acted as if this was not true, or as if an exception would be made in my case.

Particularly for medically-induced PTSD, difficulty suppressing awareness of the ongoing threat of mortality that exists in the body may be heightened by hypervigilance toward somatic threat cues. An important line of research for understanding the association between somatic focus and awareness of mortality has emerged in recent years, and has shown that

reminders of the body's corporeality induce death-related thoughts and profound psychological reactions (Cox, Goldenberg, Pyszczynski, & Weise, 2007). In one of the most striking studies to illuminate the link between a focus on the body and mortality concerns, a means for understanding the ironic association between PTSD and nonadherence to medication in ACS and stroke survivors can be found. Goldenberg and colleagues showed that after mortality was made salient for women participants, reminders of their body's fallible nature reduced intentions to conduct breast self-examinations (BSE). In a second study, they found that conducting BSEs increased death-thought accessibility, and reminders of the body's fallible nature decreased the time women spent conducting BSEs (Goldenberg, Arndt, Hart, & Routledge, 2008). Similarly, for women high on neuroticism reminders of mortality and the fallibility of the body caused increased desire to avoid thinking about mammography, and in a second study, caused increased discomfort during a mammography (Goldenberg, Routledge, & Arndt, 2009). Observations that mortality awareness may have ironic effects on health behaviors led to the development of the terror management health model (TMHM), which lays out the conditions under which psychological confrontations with the physical body can undermine symbolic defenses against mortality and, ironically, serve as barriers to health promotion behaviors (Goldenberg & Arndt, 2008).

Future Directions

EST can inform unique physiological and behavioral challenges associated with psychological and medical management of patients with PTSD due to life-threatening illness. Although there are a number of personality (e.g., neuroticism), psychological (e.g., prior anxiety or mood disorder), demographic, and social or situational factors (family conflict, job or work related issues or losses) that may predispose survivors of acute medical events to develop PTSD, those factors are also predictors of PTSD due to discrete/external traumatic events, and thus do not appear to offer unique insight into medically-induced PTSD. EST predicts that the degree of life-threat subjectively experienced by the individual at the acute onset of the traumatic medical event is the most powerful prognostic factor for later PTSD. Second, the medical prognosis associated with the event will help to determine the severity and duration of PTSD symptoms. Third, more frequent experiences of somatic symptoms that can serve as reminders of impending mortality and will predict the frequency, severity, and duration of PTSD symptoms. Fourth, patients with PTSD will experience increased awareness of -- and physiological reactivity to --somatic indicators of disease, will attempt to avoid reminders of mortality through cognitive or behavioral attempts to disengage from the body (e.g., distraction, substance use), will avoid medical treatments associated with the event, and will be at greater risk for recurrence of acute exacerbations of disease (e.g., recurrent ACS). As such, the previously observed associations between medically-induced PTSD and medication non-adherence can be explained by explicit or implicit aversive associations between secondary prevention medications and the traumatic event, and ironically, with mortality.

A research agenda based on the EST model should first test whether the proposed uniqueness of trauma due to medical events, relative to discrete past traumatic events, is confirmed by patient experience using both qualitative and quantitative research methods. Second, it should further illuminate important differences in PTSD onset, symptom

character, risk and maintaining factors, and chronicity between PTSD caused by ongoing/somatic and discrete/external past events. In particular, the influence of the enduring character and somatic source of the traumatic threat should be investigated. Similarly, research should disentangle the dual influences of the ongoing nature and somatic source on the presentation of PTSD symptoms, perhaps by comparing PTSD symptom reports of medical patients with survivors of motor vehicle accidents, for whom a threat of “recurrence” is also ongoing. Third, a thorough investigation of the role of mortality fears in the initiation, maintenance, and symptom expression should be conducted, with particular emphasis on the role of heightened somatic vigilance in the disruption of normative fear extinction in survivors of life-threatening illness.

Conclusions

Across a number of different acute life-threatening medical events, recent meta-analyses suggest that 12-25% of survivors develop significant PTSD symptoms (Edmondson & Cohen, 2013; Edmondson et al., 2012; Edmondson, Richardson, et al., 2013). Given the frequency with which these events occur, medically-induced PTSD thus represents the lion's share of the PTSD burden in most developed countries. Based on these estimates, as many as 168,000 ACS patients and more than 200,000 stroke survivors will develop clinically significant symptoms of PTSD this year in the U.S. alone. It is important to note that, unlike PTSD due to many discrete/external traumatic events, PTSD due to acute life-threatening medical events develops at least in part while patients are in our hospitals. The way that we structure those hospitals and deliver care should take into account that those factors may contribute to patients' PTSD symptoms in the future (Edmondson, Shimbo, Ye, Wyer, & Davidson, 2013). Aside from the obvious mental health and quality of life consequences of the disorder, cardiovascular disease patients who develop PTSD also experience increased cardiovascular risk. For example, as mentioned above, ACS-induced PTSD has been associated with a doubling of risk of recurrence and mortality (Edmondson et al., 2012), and stroke-induced PTSD has been associated with behavioral risk factors such as nonadherence to medications (Kronish et al., 2012).

To date, PTSD symptoms due to acute medical events have been assessed with measures designed to assess PTSD due to a discrete past event that occurred outside the individual, rather than an enduring somatic threat of mortality. That strategy overlooks important distinctions between the two phenomena, and fails to identify potentially important avenues for intervention and medical management of patients at increased medical and psychological risk. By acknowledging the unique qualities of medically-induced PTSD, researchers and clinicians can open new avenues for understanding and treating this highly prevalent but largely overlooked response to common life-threatening illnesses.

A recent systematic review and meta-analysis (Nenova et al., 2013) suggested that 68% of the 19 randomized controlled trials (RCTs) so far tested for reduction of PTSD symptoms in cancer survivors showed no effect, and only 1 study showed a consistent reduction across all PTSD symptom clusters. To date, only one pilot RCT of cognitive behavioral therapy for PTSD due to cardiovascular disease has been conducted, which was powered only to assess safety of the approach in cardiac patients (Shemesh et al., 2011). If the research agenda laid

out by the EST model uncovers the expected associations between medical symptoms, somatic focus, and enduring mortality fears, clinicians should look for ways to modify existing PTSD treatments in order to acknowledge the actual ongoing somatic threat experienced by survivors, address the present- and future-focus of medically-induced PTSD symptoms, discuss the meaning of and appropriate response to somatic symptoms, and be aware of avoidance strategies that patients might employ to reduce psychological discomfort but that may ironically increase their recurrence and mortality risk.

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References

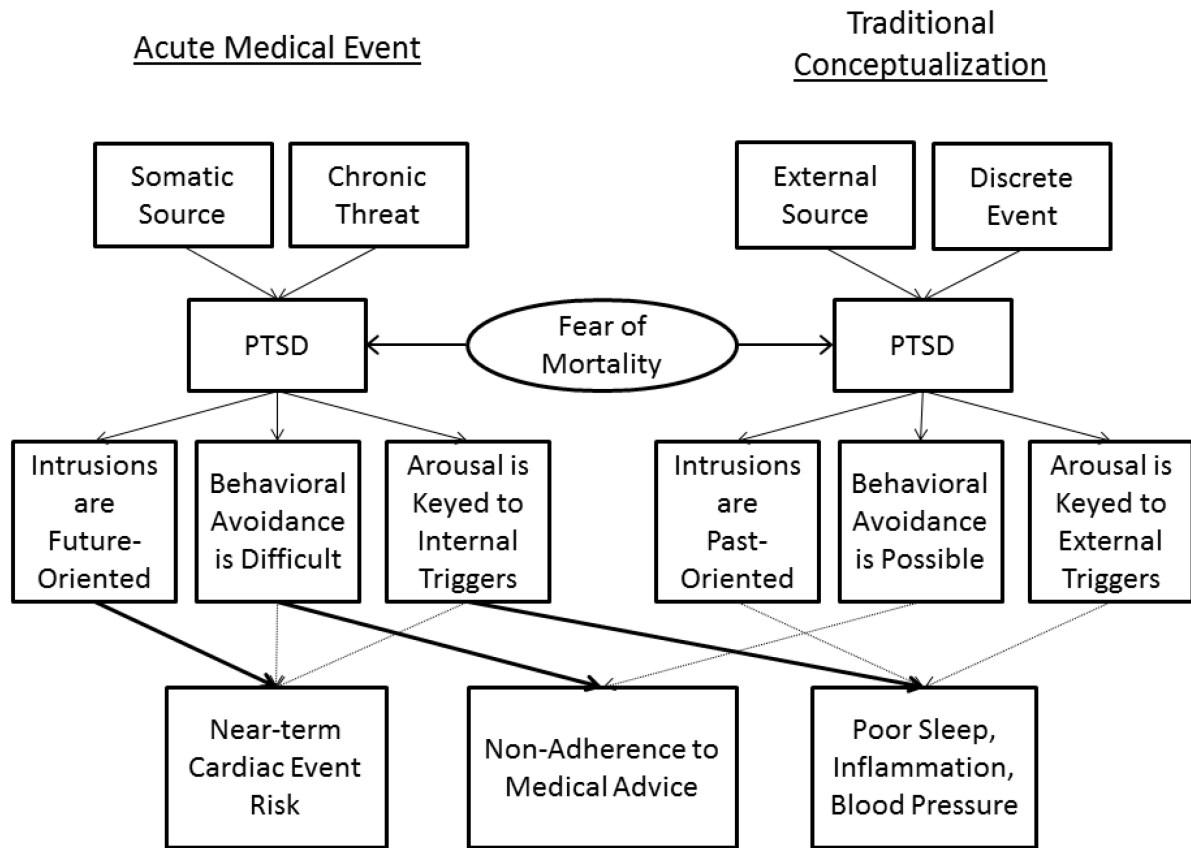
- Abdollahi A, Pyszczynski T, Maxfield M, Luszczynska A. Posttraumatic stress reactions as a disruption in anxiety-buffer functioning: Dissociation and responses to mortality salience as predictors of severity of posttraumatic symptoms. *Psychological Trauma: Theory, Research, Practice, and Policy*. 2011; 3(4):329.
- American Psychiatric Association. *Diagnostic and statistical manual of mental disorders*. 4th ed. American Psychiatric Association; Washington, D.C.: 1994.
- Ay H, Gungor L, Arsava E, Rosand J, Vangel M, Benner T, Sorensen A. A score to predict early risk of recurrence after ischemic stroke. *Neurology*. 2010; 74(2):128–135. [PubMed: 20018608]
- Baker F, Denniston M, Smith T, West MM. Adult cancer survivors: how are they faring? *Cancer*. 2005; 104(S11):2565–2576. [PubMed: 16258929]
- Bennett P, Owen RL, Koutsakis S, Bisson J. Personality, social context and cognitive predictors of post-traumatic stress disorder in myocardial infarction patients. *Psychology & Health*. 2002; 17(4): 489–500. doi: <http://dx.doi.org/10.1080/088704402200004966>.
- Burton CR. Living with stroke: a phenomenological study. *Journal of Advanced Nursing*. 2000; 32(2): 301–309. doi: 10.1046/j.1365-2648.2000.01477.x. [PubMed: 10964176]
- Cappuccio FP, Cooper D, D'Elia L, Strazzullo P, Miller MA. Sleep duration predicts cardiovascular outcomes: a systematic review and meta-analysis of prospective studies. *European heart journal*. 2011; 32(12):1484–1492. [PubMed: 21300732]
- Cordova MJ, Studts JL, Hann DM, Jacobsen PB, Andrykowski MA. Symptom structure of PTSD following breast cancer. *Journal of traumatic stress*. 2000; 13(2):301–319. [PubMed: 10838677]
- Cox CR, Goldenberg JL, Pyszczynski T, Weise D. Disgust, creatureliness and the accessibility of death-related thoughts. *European Journal of Social Psychology*. 2007; 37(3):494–507.
- Dalgleish T. Cognitive approaches to posttraumatic stress disorder: the evolution of multirepresentational theorizing. *Psychological Bulletin*. 2004; 130(2):228. [PubMed: 14979771]
- Doerfler LA, Paraskos JA, Piniarski L. Relationship of quality of life and perceived control with posttraumatic stress disorder symptoms 3 to 6 months after myocardial infarction. *J Cardiopulm Rehabil*. 2005; 25(3):166–172. [PubMed: 15931021]
- Doolittle ND. Clinical ethnography of lacunar stroke: implications for acute care. *Journal of Neuroscience Nursing*. 1991; 23(4):235. [PubMed: 1833483]
- DuHamel KN, Ostrof J, Ashman T, Winkel G, Mundy EA, Keane TM, Burkhalter J. Construct validity of the posttraumatic stress disorder checklist in cancer survivors: analyses based on two samples. *Psychological Assessment*. 2004; 16(3):255. [PubMed: 15456381]

- Edmondson D, Chaudoir S, Mills M, Park C, Holub J, Bartkowiak J. From shattered assumptions to weakened worldviews: Trauma symptoms signal anxiety buffer disruption. *Journal of Loss and Trauma*. 2011; 16:358–385. [PubMed: 24077677]
- Edmondson D, Cohen BE. Posttraumatic stress disorder and cardiovascular disease. *Prog Cardiovasc Dis*. 2013; 55(6):548–556. doi: 10.1016/j.pcad.2013.03.004. [PubMed: 23621964]
- Edmondson D, Horowitz CR, Goldfinger JZ, Fei K, Kronish IM. Concerns about medications mediate the association of posttraumatic stress disorder with adherence to medication in stroke survivors. *British Journal of Health Psychology*. 2013 doi: 10.1111/bjhp.12022.
- Edmondson D, Park CL, Blank TO, Fenster JR, Mills MA. Deconstructing spiritual well-being: existential well-being and HRQOL in cancer survivors. *Psychooncology*. 2008; 17(2):161–169. doi: 10.1002/pon.1197. [PubMed: 17506077]
- Edmondson D, Park CL, Chaudoir SR, Wortmann JH. Death without God: religious struggle, death concerns, and depression in the terminally ill. *Psychol Sci*. 2008; 19(8):754–758. doi: 10.1111/j.1467-9280.2008.02152.x. [PubMed: 18816280]
- Edmondson D, Richardson S, Falzon L, Davidson KW, Mills MA, Neria Y. Posttraumatic stress disorder prevalence and risk of recurrence in acute coronary syndrome patients: a meta-analytic review. *PLoS One*. 2012; 7(6):e38915. doi: 10.1371/journal.pone.0038915. [PubMed: 22745687]
- Edmondson D, Richardson S, Fausett JK, Falzon L, Howard VJ, Kronish IM. Prevalence of PTSD in Survivors of Stroke and Transient Ischemic Attack: A Meta-Analytic Review. *PLoS One*. 2013; 8(6):e66435. [PubMed: 23840467]
- Edmondson D, Rieckmann N, Shaffer JA, Schwartz JE, Burg MM, Davidson KW, Kronish IM. Posttraumatic stress due to an acute coronary syndrome increases risk of 42-month major adverse cardiac events and all-cause mortality. *J Psychiatr Res*. 2011; 45(12):1621–1626. doi: 10.1016/j.jpsychires.2011.07.004. [PubMed: 21807378]
- Edmondson D, Shimbo D, Ye S, Wyer P, Davidson KW. The association of emergency department crowding during treatment for acute coronary syndrome with subsequent posttraumatic stress disorder symptoms. *JAMA Internal Medicine*. 2013; 173(6):472–474. doi: 10.1001/jamainternmed.2013.2536. [PubMed: 23400256]
- Fox K, Dabbous O, Goldberg R, Pieper K, Eagle K, Van de Werf F, Anderson F Jr. Prediction of risk of death and myocardial infarction in the six months after presentation with acute coronary syndrome: prospective multinational observational study (GRACE). *British Medical Journal*. 2006; 333(7578):1091. [PubMed: 17032691]
- Gander M-L, von Kanel R. Myocardial infarction and post-traumatic stress disorder: frequency, outcome, and atherosclerotic mechanisms. *European Journal of Cardiovascular Prevention & Rehabilitation*. 2006; 13(2):165–172. [PubMed: 16575268]
- Ginzburg K, Ein-Dor T. Posttraumatic stress syndromes and health-related quality of life following myocardial infarction: 8-year follow-up. *General Hospital Psychiatry*. 2011; 33(6):565–571. [PubMed: 21958446]
- Goldberg RJ, Currie K, White K, Brieger D, Steg PG, Goodman SG, Gore JM. Six-month outcomes in a multinational registry of patients hospitalized with an acute coronary syndrome (The Global Registry of Acute Coronary Events [GRACE])* 1. *The American journal of cardiology*. 2004; 93(3):288–293. [PubMed: 14759376]
- Goldenberg JL, Arndt J. The implications of death for health: A terror management health model for behavioral health promotion. *Psychological Review*. 2008; 115(4):1032. [PubMed: 18954213]
- Goldenberg JL, Arndt J, Hart J, Routledge C. Uncovering an existential barrier to breast self-exam behavior. *Journal of Experimental Social Psychology*. 2008; 44(2):260–274. [PubMed: 19255593]
- Goldenberg JL, Routledge C, Arndt J. Mammograms and the management of existential discomfort: Threats associated with the physicality of the body and neuroticism. *Psychology & Health*. 2009; 24(5):563–581. doi: 10.1080/08870440701864546. [PubMed: 20205012]
- Goldfinger JZ, Edmondson D, Kronish IM, Fei K, Balakrishnan R, Tuhim S, Horowitz CR. Correlates of Post-traumatic Stress Disorder in Stroke Survivors. *Journal of Stroke and Cerebrovascular Diseases*. 2013

- Green BL, Rowland JH, Krupnick JL, Epstein SA, Stockton P, Stern NM, Steakley C. Prevalence of posttraumatic stress disorder in women with breast cancer. *Psychosomatics*. 1998; 39(2):102–111. [PubMed: 9584535]
- Hawk LW, Dougall AL, Ursano RJ, Baum A. Urinary catecholamines and cortisol in recent-onset posttraumatic stress disorder after motor vehicle accidents. *Psychosomatic Medicine*. 2000; 62(3): 423–434. [PubMed: 10845356]
- Janoff-Bulman R. Assumptive worlds and the stress of traumatic events: Applications of the schema construct. *Social cognition*. 1989; 7(2):113–136.
- Jørgensen H, Nakayama H, Reith J, Raaschou H, Olsen T. Stroke recurrence: predictors, severity, and prognosis. The Copenhagen Stroke Study. *Neurology*. 1997; 48(4):891. [PubMed: 9109873]
- Kronish IM, Edmondson D, Goldfinger J, Fei K, Horowitz C. Posttraumatic stress disorder and adherence to medications in survivors of strokes and transient ischemic attacks. *Stroke*. 2012 in press.
- Matsuoka Y, Nakano T, Inagaki M, Sugawara Y, Akechi T, Imoto S, Uchitomi Y. Cancer-related intrusive thoughts as an indicator of poor psychological adjustment at 3 or more years after breast surgery: a preliminary study. *Breast cancer research and treatment*. 2002; 76(2):117–124. [PubMed: 12452448]
- Mehnert A, Berg P, Henrich G, Herschbach P. Fear of cancer progression and cancer-related intrusive cognitions in breast cancer survivors. *Psycho-Oncology*. 2009; 18(12):1273–1280. [PubMed: 19267364]
- Mehnert A, Koch U. Prevalence of acute and post-traumatic stress disorder and comorbid mental disorders in breast cancer patients during primary cancer care: a prospective study. *Psycho-Oncology*. 2007; 16(3):181–188. doi: 10.1002/pon.1057. [PubMed: 16856147]
- Miller RJ, Sutherland AG, Hutchison JD, Alexander DA. C-reactive protein and interleukin 6 receptor in post-traumatic stress disorder: a pilot study. *Cytokine*. 2001; 13(4):253–255. [PubMed: 11237435]
- Mundy E, Baum A. Medical disorders as a cause of psychological trauma and posttraumatic stress disorder. *Current opinion in psychiatry*. 2004; 17(2):123.
- Nenova MMA, Morris LMA, Paul LMA, Li YP, Applebaum AP, DuHamel KP. Psychosocial Interventions With Cognitive-Behavioral Components for the Treatment of Cancer-Related Traumatic Stress Symptoms: A Review of Randomized Controlled Trials. *Journal of Cognitive Psychotherapy*. 2013; 27(3):258–284.
- Palmer SC, Kagee A, Coyne JC, DeMichele A. Experience of Trauma, Distress, and Posttraumatic Stress Disorder Among Breast Cancer Patients. *Psychosomatic Medicine*. 2004; 66(2):258–264. doi: 10.1097/01.psy.0000116755.71033.10. [PubMed: 15039512]
- Park CL, Edmondson D, Fenster JR, Blank TO. Meaning making and psychological adjustment following cancer: the mediating roles of growth, life meaning, and restored just-world beliefs. *J Consult Clin Psychol*. 2008; 76(5):863–875. doi: 10.1037/a0013348. [PubMed: 18837603]
- Pedersen SS, van Domburg RT, Larsen ML. The effect of low social support on short-term prognosis in patients following a first myocardial infarction. *Scand J Psychol*. 2004; 45(4):313–318. doi: 10.1111/j.1467-9450.2004.00410.x [doi] SJOP410 [pii]. [PubMed: 15281920]
- Pyszczyński T, Kesebir P. Anxiety buffer disruption theory: A terror management account of posttraumatic stress disorder. *Anxiety, Stress, & Coping*. 2011; 24(1):3–26.
- Redfern J, Briffa T, Ellis E, Freedman SB. Choice of secondary prevention improves risk factors after acute coronary syndrome: 1-year follow-up of the CHOICE (Choice of Health Options In prevention of Cardiovascular Events) randomised controlled trial. *Heart*. 2009; 95(6):468–475. [PubMed: 18801781]
- Roger VL, Go AS, Lloyd-Jones DM, Benjamin EJ, Berry JD, Borden WB, Fox CS. Heart Disease and Stroke Statistics—2012 Update A Report From the American Heart Association. *Circulation*. 2012; 125(1):e2–e220. [PubMed: 22179539]
- Sacco R, Wolf P, Kannel W, McNamara P. Survival and recurrence following stroke. The Framingham study. *Stroke*. 1982; 13(3):290–295. doi: 10.1161/01.str.13.3.290. [PubMed: 7080120]

- Shaffer JA, Kronish IM, Burg M, Clemow L, Edmondson D. Association of Acute Coronary Syndrome-Induced Posttraumatic Stress Disorder Symptoms with Self-Reported Sleep. *Ann Behav Med*. 2013 doi: 10.1007/s12160-013-9512-8.
- Shalev AY, Schreiber S, Galai T, Melmed RN. Post-traumatic stress disorder following medical events. *British Journal of Clinical Psychology*. 1993; 32(2):247–253. [PubMed: 8318945]
- Shelby RA, Golden-Kreutz DM, Andersen BL. PTSD diagnoses, subsyndromal symptoms, and comorbidities contribute to impairments for breast cancer survivors. *Journal of traumatic stress*. 2008; 21(2):165–172. doi: 10.1002/jts.20316. [PubMed: 18404636]
- Sheldrick R, Tarrier N, Berry E, Kincey J. Post-traumatic stress disorder and illness perceptions over time following myocardial infarction and subarachnoid haemorrhage. *British Journal of Health Psychology*. 2006; 11(3):387–400. doi: 10.1348/135910705X71434. [PubMed: 16870051]
- Shemesh, E.; Annunziato, RA.; Weatherley, BD.; Cotter, G.; Feaganes, JR.; Santra, M.; Rubinstein, D. A randomized controlled trial of the safety and promise of cognitive-behavioral therapy using imaginal exposure in patients with posttraumatic stress disorder resulting from cardiovascular illness. 2011.
- Shemesh E, Rudnick A, Kaluski E, Milovanov O, Salah A, Alon D, Cotter G. A prospective study of posttraumatic stress symptoms and nonadherence in survivors of a myocardial infarction (MI). *General Hospital Psychiatry*. 2001; 23(4):215–222. [PubMed: 11543848]
- Shemesh E, Yehuda R, Milo O, Dinur I, Rudnick A, Vered Z, Cotter G. Posttraumatic stress, nonadherence, and adverse outcome in survivors of a myocardial infarction. *Psychosomatic Medicine*. 2004; 66(4):521–526. [PubMed: 15272097]
- Shin LM, Rauch SL, Pitman RK. Amygdala, medial prefrontal cortex, and hippocampal function in PTSD. *Ann N Y Acad Sci*. 2006; 1071:67–79. doi: 10.1196/annals.1364.007. [PubMed: 16891563]
- Smith SC Jr, Benjamin EJ, Bonow RO, Braun LT, Creager MA, Franklin BA, Jones DW. *AHA/ACCF Secondary Prevention and Risk Reduction Therapy for Patients With Coronary and Other Atherosclerotic Vascular Disease: 2011 Update*. *Circulation*. 2011; 124(22):2458–2473. [PubMed: 22052934]
- Smith MY, Redd WH, Peyser C, Vogl D. Post-traumatic stress disorder in cancer: a review. *Psycho-Oncology*. 1999; 8(6):521–537. [PubMed: 10607985]
- Tang EW, Wong CK, Herbison P. Global Registry of Acute Coronary Events (GRACE) hospital discharge risk score accurately predicts long-term mortality post acute coronary syndrome. *American Heart Journal*. 2007; 153(1):29–35. [PubMed: 17174633]
- United States Department of Defense. *Armed Forces Strength Figures for January*. 2013; 31:2013.
- Vedin O, Hagström E, Stewart R, Brown R, Krug-Gourley S, Davies R, Held C. Secondary prevention and risk factor target achievement in a global, high-risk population with established coronary heart disease: baseline results from the STABILITY study. *European Journal of Preventive Cardiology*. 2012
- von Kanel R, Begre S, Abbas CC, Saner H, Gander M-L, Schmid J-P. Inflammatory biomarkers in patients with posttraumatic stress disorder caused by myocardial infarction and the role of depressive symptoms. *Neuroimmunomodulation*. 2010; 17(1):39–46. [PubMed: 19816056]
- von Känel R, Hepp U, Traber R, Kraemer B, Mica L, Keel M, Schnyder U. Measures of endothelial dysfunction in plasma of patients with posttraumatic stress disorder. *Psychiatry Research*. 2008; 158(3):363–373. [PubMed: 18252265]
- Whitaker KL, Watson M, Brewin CR. Intrusive cognitions and their appraisal in anxious cancer patients. *Psycho-Oncology*. 2009; 18(11):1147–1155. [PubMed: 19140125]
- Wiedemar L, Schmid J-P, Muller J, Wittmann L, Schnyder U, Saner H, von Kanel R. Prevalence and predictors of posttraumatic stress disorder in patients with acute myocardial infarction. *Heart & Lung*. 2008; 37(2):113–121. [PubMed: 18371504]
- Wikman A, Bhattacharyya M, Perkins-Porras L, Steptoe A. Persistence of posttraumatic stress symptoms 12 and 36 months after acute coronary syndrome. *Psychosomatic Medicine*. 2008; 70(7):764. [PubMed: 18725431]

Yehuda R. Advances in understanding neuroendocrine alterations in PTSD and their therapeutic implications. *Ann N Y Acad Sci.* 2006; 1071:137–166. doi: 10.1196/annals.1364.012. [PubMed: 16891568]



Note: Arrows represent associations. For associations with behavioral and health outcomes, dark arrows represent associations with the strongest and/or most consistent empirical evidence, and light arrows represent associations with some empirical evidence.

Figure.
Enduring Somatic Threat Model of PTSD due to Acute Medical Events

Table 1

DSM-V Diagnostic Criteria for PTSD

Criteria		
A. Stressor	The person was exposed to: death, threatened death, actual or threatened serious injury, or actual or threatened sexual violence: (1 required)	<ol style="list-style-type: none"> 1. Direct exposure. 2. Witnessing, in person. 3. Indirectly, by learning that a close relative or close friend was exposed to trauma. If the event involved actual or threatened death, it must have been violent or accidental. 4. Repeated or extreme indirect exposure to aversive details of the event(s), usually in the course of professional duties (e.g., first responders, collecting body parts; professionals repeatedly exposed to details of child abuse). This does not include indirect non-professional exposure through electronic media, television, movies, or pictures.
B. Intrusion symptoms	The traumatic event is persistently re-experienced: (1 required)	<ol style="list-style-type: none"> 1. Recurrent, involuntary, and intrusive memories. Note: Children older than 6 may express this symptom in repetitive play. 2. Traumatic nightmares. Note: Children may have frightening dreams without content related to the trauma(s). 3. Dissociative reactions (e.g., flashbacks) which may occur on a continuum from brief episodes to complete loss of consciousness. Note: Children may reenact the event in play. 4. Intense or prolonged distress after exposure to traumatic reminders. 5. Marked physiologic reactivity after exposure to trauma-related stimuli.
C. Avoidance	Persistent effortful avoidance of distressing trauma-related stimuli after the event: (1 required)	<ol style="list-style-type: none"> 1. Trauma-related thoughts or feelings. 2. Trauma-related external reminders (e.g., people, places, conversations, activities, objects, or situations).
D. Negative alterations in cognitions and mood	Negative alterations in cognitions and mood that began or worsened after the traumatic event: (2 required)	<ol style="list-style-type: none"> 1. Inability to recall key features of the traumatic event (usually dissociative amnesia; not due to head injury, alcohol or drugs). 2. Persistent (and often distorted) negative beliefs and expectations about oneself or the world (e.g., "I am bad," "The world is completely dangerous.>"). 3. Persistent distorted blame of self or others for causing the traumatic event or for resulting consequences. 4. Persistent negative trauma-related emotions (e.g., fear, horror, anger, guilt or shame). 5. Markedly diminished interest in (pre-traumatic) significant activities. 6. Feeling alienated from others (e.g., detachment or estrangement). 7. Constricted affect: persistent inability to experience positive emotions.
E. Alterations in arousal and reactivity	Trauma-related alterations in arousal and reactivity that began or worsened after the traumatic event: (2 required)	<ol style="list-style-type: none"> 1. Irritable or aggressive behavior. 2. Self-destructive or reckless behavior. 3. Hypervigilance. 4. Exaggerated startle response. 5. Problems in concentration. 6. Sleep disturbance.
F. Duration	Persistence of symptoms (in Criteria B, C, D and E) for more than one month.	
G. Functional significance	Significant symptom-related distress or functional impairment (e.g., social, occupational).	