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Post-Traumatic Stress Disorder and its Somatic Comorbidities: A Review

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Abstract

As of today, research has shown that exposure to traumatic events and psychological trauma is a widespread phenomenon. Its association with PTSD and other various mental disorders has been widely studied. However, research suggests the exposure to such events is said to be correlated with early onset of specific diseases, increased healthcare utilization, and premature death. The aim of this review is to summarize the available literature and explore various diseases and their relations to PTSD. The areas of interest for this particular review will include cardiovascular, metabolic, and autoimmune diseases as well as inflammation. Ample evidence of the link between PTSD and somatic diseases (specifically cardiovascular, metabolic, and autoimmune disease as well as additional comorbidities that do not fit into either category) following it is presented, as well as some additional studies showing bidirectional relationships between PTSD and several illnesses. Several other notable comorbidities are discussed as well, followed by a brief overview of presented studies and their limitations as well as a conclusion. Last presented is a list of potential future research directions, followed by a generalized discussion of the review at hand. A strong recommendation for rigorous implementation of trauma-informed care in healthcare facilities is presented, along with research corroborating the need for such practice.

Key words: Post-Traumatic Stress Disorder **(**PTSD); cardiovascular diseases; metabolic diseases; inflammation; autoimmune diseases

Introduction

Post-traumatic stress disorder (PTSD) is a mental disorder, which may develop in response to exposure to trauma - experiencing or witnessing an event when one's health, life, or well-being are threatened; it is characterized by many symptoms such as flashbacks or emotional numbing, but also a constant state of physiological arousal leading to an exaggerated startle response, disturbed sleep, and difficulties with concentrating or remembering (APA Dictionary of Psychology, 2023). It is known that some of the most common comorbidities are other anxiety disorders, depressive disorders, and substance abuse (Qassem et al, 2020). However, the impact of PTSD on physical health has become an area of growing interest in recent years. This work aims to be a comprehensive review of the findings in that specific field, providing a detailed analysis of the most recent findings of PTSD and its non-psychiatric comorbidities. It is important to fully understand the disorder and its implications in order to optimize possible treatment plans and offer patients the best care possible in their time of need.

The first area of interest this review will be examining is the prevalence of comorbid cardiovascular disease. To date this field has been extensively studied, as evidenced by numerous reviews and meta-analyses published on it, and sufficient studies have been conducted to conclude PTSD is indeed a risk factor for cardiovascular disease (Edmondson & Känel, 2017). This article will review the available data and examine its findings as well as shortcomings.

Next, due to their often-inextricable link to cardiovascular issues, this review will focus on metabolic diseases. Studies suggest there is a positive correlation between PTSD and obesity (Kubzanksy et al, 2014). This article will review the available literature on the topic of comorbid PTSD and obesity as well as other metabolic diseases, such as type II diabetes.

The next area of interest of this review is inflammation and, subsequently, autoimmune disease. There is some evidence that an increase in inflammatory factors is a predictor for PTSD (Bielas et al, 2018) and that PTSD is correlated with pro-inflammatory activity (Tursich et al, 2014). Research has also shown that PTSD is a risk factor for developing autoimmune diseases

(Song et al, 2018). This work will present the available data on the matter as well as highlight possible issues with studies conducted to date.

Lastly, before moving on to general discussions and future study directions, this paper will briefly discuss other notable studies of PTSD and its various somatic comorbidities.

PTSD and cardiovascular disease

As of today, cardiovascular diseases remain among most well-studied comorbidities of PTSD. As such, substantial literature documenting the potential relationship between the disorder and increased cardiovascular reactivity to trauma-related cues, sleep disturbances, near-perpetual autonomic hyperarousal and altered HPA activity can be found. Thus, a model of this relationship has been proposed as seen in Figure 1.

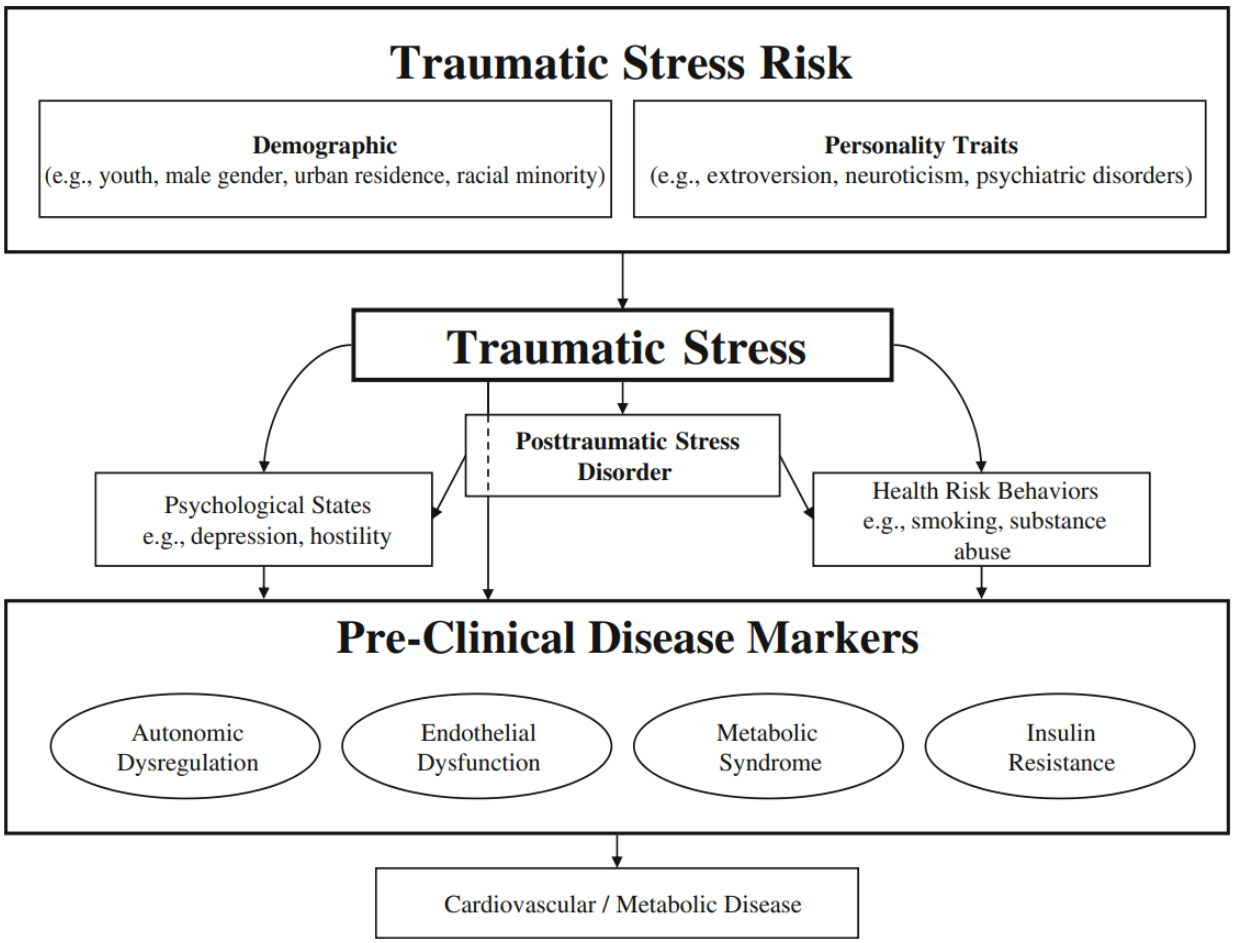

Figure 1. Model of the complex relationship between traumatic stress risk, PTSD, and cardiovascular diseases (Dedert et al, 2010).

This model proposes that both PTSD and traumatic stress are directly related to several biomarkers and physiological reactivity indices. This hypothesis is backed by several studies tackling the correlation between PTSD and several different biomarkers, with one of the most thoroughly studied among them being heart rate. A monozygotic twin study discordant for combat exposure showed that being exposed to sudden loud noises elicited an accelerated heart rate response in Vietnam veterans as opposed to their non-combatant twins (Orr et al, 2003). The aforementioned reactivity may be relevant to cardiovascular disease, as resting heart rate has been shown to be predictive of earlier mortality from cardiovascular disease (Greenland et al, 1999). In addition to this, several studies found that individuals with PTSD suffer from an increased basal heart rate even in the absence of a traumatic stimulus (Obilom & Thacher, 2008; Rabe et al, 2006). Another biomarker, blood pressure, has not been studied as extensively or consistently as heart rate. It has yielded some significant results regardless; one meta-analytic study found that PTSD was associated with higher basal blood pressure relative to both non-trauma exposed individuals as those without a PTSD diagnosis, however these results were smaller in magnitude than the results obtained for increased basal heart rate among individuals with PTSD (Buckley & Kaloupek, 2001). A different study has shown a more drastic increase in ambulatory blood pressure among PTSD veterans as opposed to veterans with no such diagnosis (Buckley et al, 2004). These findings were corroborated by another study done on a sample of individuals with a PTSD diagnosis, who had elevated systolic and diastolic blood pressure (Kellner et al, 2003). Another biomarker relevant to the topic at hand is endothelial dysfunction. Studies have shown endothelial dysfunction to be the first step before cardiovascular disease (Lüscher & Noll, 1995; Harrison, 1994). As such, assessment of endothelial functioning may serve as a marker of development of arterial disorders. Additionally, it may provide an additional method to utilize in studies on the development of cardiovascular disease in PTSD. One way of monitoring endothelial functions is via flow-mediated dilation (FMD), which uses ultrasound imaging to follow changes in vessel diameter in response to blood flow. In vessels lined with healthy endothelium increased blood flow leads to dilation of the vessel; this response is disrupted by endothelium dysfunction. Studies have shown that FMD can detect endothelial dysfunction among individuals at high risk of atherosclerosis prior to the onset and clinical manifestation of vascular diseases (Celermajer et al. 1992; Celermajer et al, 1994). Such methods of early detection are crucial for a multitude of reasons, one of them being the increased risk of development and onset of arterial disorders among patients with self-reported PTSD symptoms (Schnurr et al, 2000). Another study examining a sample of mixed male and female police officers showed that individuals who

reported a higher severity of PTSD symptoms exhibited half the brachial reactivity when compared to those who declared lower symptom severity (Violanti et al, 2007). It is important to note that this study was largely limited by its reliance on self-report measures instead of tools sufficient for a clinical PTSD diagnosis. Another biomarker to take note of when discussing PTSD and CVD is baroreceptor sensitivity. Baroreceptors are nerves that detect changes in pressure and are responsible for regulating parasympathetic activity. Consequently, baroreceptor sensitivity has been used to measure PNS tone. Several studies conducted in controlled laboratory settings have shown a relationship between PTSD and reduced parasympathetic functioning, as well as PNS control of the heart (Cohen et al, 1997; Hughes et al, 2007). However, as illustrated by a study where children with PTSD were monitored by an electrocardiogram while interacting with their parents, parasympathetic components of cardiovascular functioning may be influenced by an individual's immediate environment (Scheeringa et al, 2004). Another noteworthy finding was the discrepancy in baroreceptor sensitivity between male and female smokers with PTSD, whereas a study has shown that while baroreceptor sensitivity remained unaltered in men it was significantly reduced in women (Hughes et al, 2006).

Even in the more recent years, PTSD symptoms have been shown to be correlated with an increased risk of incident CVD in a sample of 49,978 women (Sumner et al, 2015). One study showed that a group of women who were victims of assault did not exhibit differences in heart rate one month after the event, but did six months after (Griffin, 2006). That, however, may be explained by the changes made within the most recent fifth edition of the *Diagnostic and Statistical Manual for Mental Disorders*, which added a specification of PTSD with delayed expression whereas some symptoms may not appear up until six months after the traumatic event (American Psychiatric Association, 2013). Another epidemiological study on the general population of Denmark examined the relationship between PTSD and CVD events (specifically myocardial infarction, stroke, ischaemic stroke, and venous thromboembolism). It found that individuals suffering from PTSD faced a 60% increased risk of cardiovascular disease (Gradus et al, 2015). Some research also points to PTSD as a consequence of CVD events, which consequently worsens the prognosis for the cardiovascular disease (Edmondson & von Känel, 2017). Interestingly, recent findings indicate that alleviating PTSD symptoms shortly after its initial onset may mitigate the risk of developing cardiovascular disease (Glisanz et al, 2017).

PTSD and metabolic disease

According to the model presented in Figure 1, traumatic stress risk and consequently post-traumatic stress disorder are linked to the development of metabolic disease. According to one meta-analysis, individuals suffering from PTSD are at a higher risk of developing type II diabetes (Vancampfort et al, 2016), however it is important to note that the authors of said review stress that the results should be approached with caution, since they relied on self-reports. One point of contention regarding the comorbidity of PTSD and type II diabetes is obesity; one study showed that while there was a link between PTSD and T2D, said risk lost significance after accounting for obesity (Kubsanzky et al, 2014). This finding was corroborated by a later study which investigated the role of obesity among PTSD patients with incident diabetes and found that type II diabetes was not a risk factor in the absence of obesity in individuals suffering from PTSD (Sherrer et al, 2018). While this clearly shows there is no direct link between a PTSD diagnosis and development of type II diabetes, PTSD may be an indirect risk factor. Some suggest that PTSD is the psychiatric disease most commonly associated with obesity (Jakovljević et al, 2008). A possible reason for that, a study showed that when compared to patients with major depressive disorder those with PTSD were related to various lipid variables, namely higher cholesterol levels, low-density lipoproteins, and triglycerides, as well as decreased high-density lipoproteins (Solter et al, 2002). The aforementioned lipid variances - dyslipidemia - have recently shown to be correlated with development of obesity (Bays et al, 2024). And while dyslipidemia is a known likely cause of myocardial infarction and ischemic heart disease (Thomsen & Nordestgaard, 2014), it is important to examine their prevalence among PTSD patients, since as mentioned in the introductory paragraph metabolic and cardiovascular diseases remain inextricably linked, which makes them difficult to discuss separately. One study highlights that PTSD was linked to ischemic changes on exercise treadmill tests after adjusting for traditional cardiac risk factors, c-reactive protein, several health behaviors as well as certain psychosocial factors (Turner et al 2013). Interestingly, some studies suggest something of a bidirectional relationship between the two, seeing how individuals who have developed PTSD after a myocardial infarction were more likely to develop myocardial ischemia (Lima et al, 2020).

It is now known that one of the most common risk factors for type II diabetes is obesity (Yashi & Daley, 2023). With PTSD being another known risk factor for obesity, it is important to know that obesity is the most critical factor for coronary heart disease (Manoharan et al, 2022). Another way of looking at PTSD and its relationship with obesity is through a behavioral

and neuropsychological lens. Patients with PTSD appear to have alterations in their cerebral reward systems, specifically the nucleus accumbens which is known as a key factor in not only developing drug and gambling addictions, but also disturbed eating behaviors (Salamone et al, 2007). One study illustrated the link between PTSD and food addiction specifically, showing the increased prevalence in a sample of women with a PTSD diagnosis (Mason et al, 2014). This shows yet another connection illustrated in Figure 1, which shows the possible link between metabolic disease and health risk behaviors.

PTSD, inflammation, and autoimmune disease

Studies have shown that a PTSD diagnosis as well as the severity of exhibited symptoms are correlated with inflammation, as evidenced by a meta-analysis which showed significant correlations between trauma exposure and circulating concentrations of proinflammatory cytokines IL-1β, IL-6, and TNF-α and of the acute-phase protein CRP; the relationship between the presence of proinflammatory cytokines and trauma exposure was the strongest in the clinical population of symptomatic individuals (Tursich et al, 2014). Additionally, one meta-analysis showed the correlation between PTSD and decreased levels of anti-inflammatory markers (Passos et al, 2015). One study suggests that the reason for the prevalence of inflammation among individuals with PTSD is that it promotes a positive feedback loop, whereas IL-1β, IL-6, and TNF-α induce the CRP to activate the complement system which in turn leads to a multitude of inflammation-promoting events being triggered (Hori & Kim, 2019). In spite of all that, there is little evidence determining the causality of PTSD in inflammation. However, a growing body of research reports a bidirectional relationship between the two; one review finds that pre-diagnosis increased levels of inflammation markers may be predictive of a higher sensitivity to developing PTSD while a later diagnosis may lead to worsening the inflammation (Sumner et al, 2020) and another suggests that while there is a genetic predisposition for risk of developing PTSD and increased levels of CRP the relationship is not causal in either direction, but rather influenced by a third, outside factor (Carvalho, 2020).

It is now known that inflammation is a key driving factor behind the development of autoimmune disease (Xiang et al, 2023). As such, it seems appropriate to now shift the focus to the relationship between PTSD and development of autoimmune disease. A population- and sibling-matched study on the Swedish population has shown that compared to non-disordered individuals, those with PTSD not only ran a higher risk of autoimmune diseases but also numerous autoimmune syndromes, and the risk was found to be even higher among younger

individuals (Song et al, 2018). A different retrospective study of US veterans of the wars in Afghanistan or Iraq found that those suffering from PTSD had elevated risk of developing rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis, inflammatory bowel disease, and thyroids (O'Donovan et al, 2015).

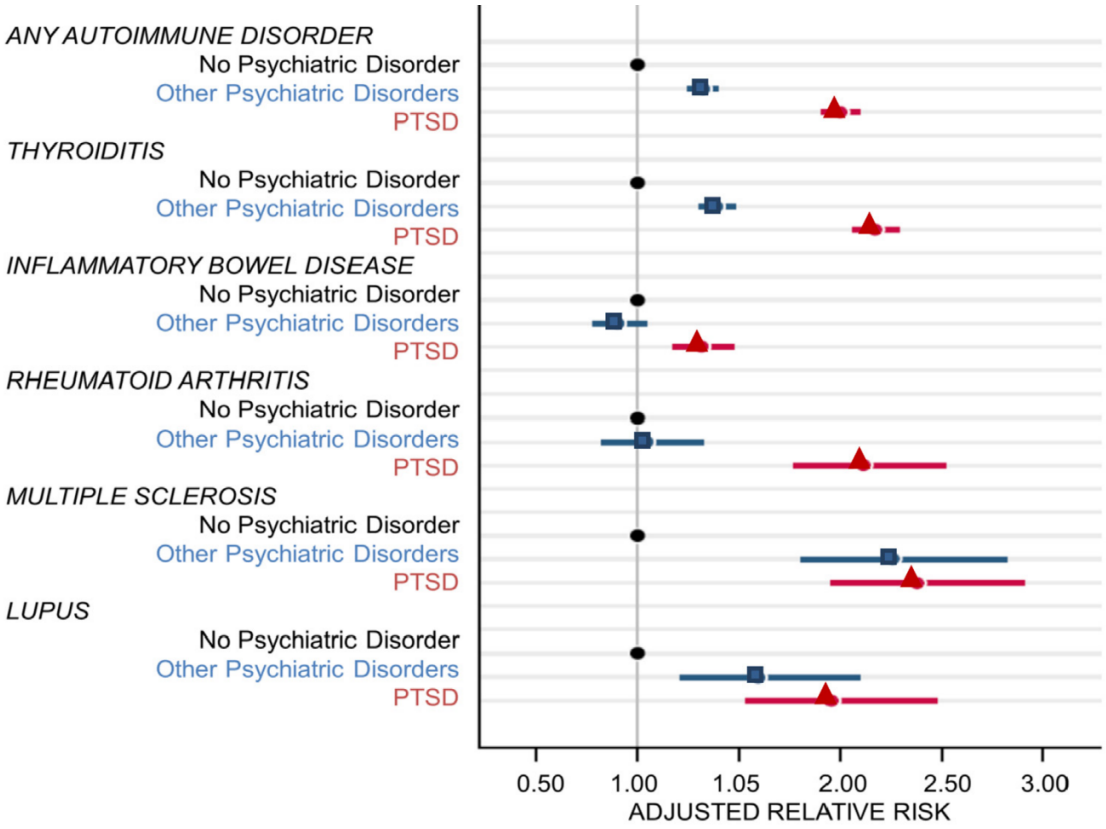

Figure 2. Adjusted relative risk for autoimmune disorder diagnoses in veterans without any psychiatric disorder (black circles), with psychiatric disorders other than posttraumatic stress disorder (PTSD) (blue squares), and with PTSD with and without other psychiatric disorders (red triangles) adjusted for age, gender, race, and primary care visits. Dots represent adjusted relative risk, and lines represent 95% confidence intervals (O'Donovan et al, 2015).

These studies provide ample evidence for the association of PTSD with autoimmune disease. However, there are several notable limitations to individual studies examining the relationship between PTSD and autoimmune disease, namely that their only focus were military veterans who have finished their service (Boscarino et al, 2010), only a single gender (Roberts et al, 2017), or civilian populations (Lee et al, 2015). One study aimed to tackle this by using the Millenium Cohort Study (Ryan et al, 2007) to investigate the risk levels of autoimmune disease in active veterans with history of PTSD; they found that individuals with said history

ran an increased risk of developing rheumatoid arthritis, systemic lupus erythematosus, multiple sclerosis or inflammatory bowel disease which were the main focus of the study (Bookwalter et al 2020).

Other notable comorbidities

A recently published study uncovered a link between PTSD and an increased risk of incident stroke in a sample of female veterans (Ebrahimi et al, 2024). One other review corroborates those findings while postulating the bidirectionality of this relationship, presenting additional studies which show PTSD as an independent risk factor for stroke (Perkins et al, 2021).

There is a different, rapidly growing area of interest in the field of the somatic comorbidities of PTSD, namely asthma. One review presented a strong case for a link between asthma and PTSD, although it did not establish the directionality of said relationship (Algire et al, 2021).

One other study found that patients with PTSD had a higher risk of developing Parkinson's disease (Chan et al, 2017). Another study which tested a cohort of 8336 patients suffering from PTSD matched with an equal number of those without it showed that the individuals with PTSD had an increased risk of developing Parkinson's disease (Barer et al, 2022).

Notably, studies of the relationship between PTSD and cancer have not shown any link between the two. One nationwide cohort study of the Danish population reported a lack of evidence of any association (Gradus et al, 2015), which falls in line with the reports of previous research on the same topic (Kisley et al, 2013).

Limitations

To date, relatively few studies targeted generalized populations, instead focusing rather on specific at-risk subgroups (eg. veterans). This, while insightful, may not be applicable to greater populations. Another limitation was focusing on self-reports for post-traumatic stress disorder, which may have skewed the results and led to obtaining results not reflective of the facts. Several studies and reviews have not been updated, thus still relying on outdated definitions. A noticeable lack of data from longitudinal studies can render the findings inaccurate.

Future research directions

In the case of many a disease, the direction of the relationship needs to be studied. A more in-depth, holistic approach to PTSD comorbid with physical illness should be considered and tested. It is also important to establish whether there is a relationship between the cause of trauma and its somatic comorbidities or not. The relationship between the severity of PTSD symptoms and the severity of the physical disease in question should be studied. It is crucial to examine the influence of various potential mediators, such as psychosocial, biological, and behavioral factors, in the development of somatic diseases in the aftermath of a PTSD diagnosis.

Conclusion

The goal of this literature review was to examine the current state of knowledge on post-traumatic stress disorder, a psychiatric disease developing in response to experiencing a traumatic event, and various physical illnesses. The main areas of interest were cardiovascular, metabolic, and autoimmune disease with an additional focus on inflammation. Other diseases of note, such as cancer, asthma, or Parkinson's disease, have also been examined.

This paper has summarized the currently available data on the matter and presented their strengths as well as weaknesses before scrutinizing the limitations of available studies. Lastly, potential directions for future research were presented.

Discussion

As of right now, ample evidence is available to show the severe implications of trauma, not only on the mind but the whole body. Not only can it lead to a debilitating condition that is post-traumatic stress disorder, said disorder is a getaway to a myriad other, somatic issues.

Understanding the various ways trauma can impact the body is crucial to proper treatment, not only of the disorder but also the physical aftermath of it - the deadly diseases that oftentimes follow. Thus, it is necessary to highlight the importance of trauma-informed care; it is a framework for helping those who faced adverse circumstances (Van der Kolk, 2014). There are various frameworks conceptualizing it, however one study postulated that the understanding of structural and interpersonal impact of traumatic events on people's lives and behaviors as well as creating emotionally and physically safe spaces for both service providers and users (Wathen et al, 2023) are key principles of said approach. One review examines studies that have

shown the beneficial effects of implementing trauma-informed care in medical and healthcare facilities, as illustrated (Goldstein et al, 2024).

The first step to curing a disease is understanding it. Understanding when disorders are caused by another affliction may yet save many lives.

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