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The Link Between PTSD and Autoimmune Diseases: A Path to Effective Treatment. Literature review

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Abstract

Introduction and Objective:
PTSD is a psychiatric disorder associated with the experience of a traumatic event in the past. Changes in the immune system are presented as one of the mechanisms involved in its pathophysiology. The purpose of this review is to investigate the potential link between PTSD and autoimmune diseases, as well as to highlight the therapeutic implications.

Review methods:
The review is based on scientific publications in PubMed, Science Direct and NCBI databases. After an initial evaluation of the articles, primarily observational studies and meta-analyses on the relationship between PTSD and autoimmune diseases, the underlying pathophysiology, and new potential therapeutic options were selected.

Abbreviated description of the state of knowledge:
PTSD is associated with numerous changes in the immune and neuroendocrine systems. As a result, patients affected by the disorder are susceptible to developing somatic diseases, including autoimmune diseases. Numerous studies indicate a reciprocal effect on the increased risk and aggravation of symptoms for PTSD and autoimmune diseases due to certain similarities in pathophysiology. New treatment options for PTSD targeting the immune system with anti-inflammatory and immunomodulatory drugs are currently being explored.

Summary:

This review article highlights the existing reciprocal relationship between PTSD and autoimmune diseases, which carries important clinical implications. The authors of the review emphasize the importance of better understanding the pathophysiology of PTSD and the early diagnosis and treatment of people with PTSD, as well as the need to develop new effective treatments for PTSD that can help reduce the risk of developing autoimmune diseases in people with the disorder. As suggested, treatment targeting the immune system may be an effective therapeutic option for patients with PTSD.

Keywords: autoimmune diseases; post-traumatic stress disorders; trauma and stressor related disorders

Introduction

Post-traumatic stress disorder (PTSD) and autoimmune diseases are two common conditions that significantly affect the functioning of sufferers. While the two entities are considered distinct, recent research nevertheless suggests a potential link between the two. This review article delves into the current understanding of how PTSD may affect the development of autoimmune diseases.

PTSD is a psychiatric disorder that can occur because of exposure to a traumatic event, such as accidents, natural disasters, violence or warfare. Typical symptoms include intrusive thoughts and memories, avoidance of reminders, hypervigilance, anxiety, depression, sleep disturbances, hyperactivity and difficulty concentrating.

Autoimmune diseases, on the other hand, encompass a spectrum of conditions in which the immune system begins to recognize healthy tissues as foreign and thus begins to attack them. This situation can lead to a variety of symptoms depending on the specific autoimmune disease. Some of the more common autoimmune diseases are rheumatoid arthritis, lupus erythematosus, Graves-Basedow disease and type 1 diabetes. The exact etiology of autoimmune diseases is unknown, likely involving a complex interaction of environmental, genetic and immunological factors. Recently, there has been growing interest in understanding how

psychological factors, particularly the stress accompanying conditions such as PTSD, may influence the onset and progression of these diseases.

Although research into the relationship between PTSD and autoimmune diseases is ongoing, accumulating evidence points to a potential link. Autoimmune diseases appear to be more prevalent in people with PTSD, but there are also reports that people with autoimmune diseases appear to be more likely to develop PTSD. Several mechanisms may explain this association. Chronic stress associated with PTSD may disrupt the balance of the immune system in various ways, thereby increasing susceptibility to autoimmune disorders. An additional mechanism is the potential effect of stress on gene expression and epigenetic processes that contribute to the development of autoimmune diseases.

In this review article, we will examine existing research on the relationship between PTSD and autoimmune diseases and discuss the potential mechanisms underlying this relationship, as well as provide an update on the current state of knowledge on this topic. We will also address areas including clinical implications and implications for future research and clinical practice. The goal is to draw attention to better diagnosis and treatment of people with PTSD, with a particular focus on the prevention of autoimmune diseases in these individuals.

PTSD

PTSD is a mental disorder that can develop after exposure to a traumatic event. Epidemiological studies indicate that 3.9% of the world's population experiences PTSD. Approximately 354 million adult survivors have PTSD and/or major depression, with 26% meeting diagnostic criteria for PTSD. [1] [2]

PTSD has been linked to several different psychiatric conditions including anxiety disorders, depressive disorders, impulse control disorders, addiction to various substances and suicidal behavior. [3] Furthermore, chronic stress associated with PTSD is associated with a number of changes in the immune, nervous and endocrine systems. There is growing evidence that post-traumatic stress disorder is associated with increased morbidity, particularly from autoimmune and cardiovascular diseases.

Patophysiology of PTSD

Pro-inflammatory environment

Inflammation is an important component of pathophysiology in both autoimmune diseases and PTSD.

Analyses indicate that pro-inflammatory populations of CD4+ T cells, Th1 cells, and Th17 cells are increased in individuals with PTSD, while Treg lymphocytes are decreased. [4] Treg lymphocytes play a key role in modulating the inflammatory response and protecting the body from the effects of excessive inflammation that occur in autoimmune diseases, or allergies. Reduced ability to suppress the immune response, which leads to increased immune reactivity and chronic inflammation in individuals, is a significant factor in the increased susceptibility to the development of autoimmune diseases such as Hashimoto's disease, IBD and rheumatoid arthritis in people with PTSD. [5] Moreover, there is evidence of premature aging of the immune system in PTSD, associated with a lower ratio of CD4+ T cells to CD8+ T cells. Such a condition is usually characteristic of the elderly, whereas it occurs earlier in PTSD. [6]

Several studies have found elevated serum CRP (C-reactive protein) levels in people with PTSD. [7] CRP is an important element involved in the activation of the complement system after stimulation with IL-6 or other pro-inflammatory cytokines such as TNF-α and IL-1β. In addition, high levels of CRP, a frequently used clinical marker of inflammation, have been found to correlate with the severity of PTSD symptoms. [8]

One of the immune mechanisms in response to stress is increased secretion of pro-inflammatory cytokines - IL-6, IFN-γ, IL-1β, IL-10, TNF-α, IL-17. [9] [10] One study found that participants with a prior diagnosis of post-traumatic stress disorder, who had levels of pro-inflammatory cytokines compared at 3 time points - before experiencing the stressful situation, during the stressful situation, and 1 hour after experiencing the stressful situation - had higher levels of pro-inflammatory cytokines at each of these time points compared to those without the diagnosis. Interestingly, in the results for PTSD, concentrations of pro-inflammatory cytokines were also positively correlated with the severity of the level of exposure to traumatic events on the LEC-5 scale. [11] Increased secretion of pro-inflammatory cytokines causes secondary symptoms after exposure to stress such as fatigue and sleep problems. At the same time, sleep deprivation, which is one of the symptoms in PTSD, also causes an increase in pro-inflammatory cytokines, and is associated with oxidative stress. During the inflammatory

response, the release of various biological catalysts including pro-inflammatory cytokines, reactive oxygen species and nitrogen induces oxidative stress. At the same time, as one of the elements of PTSD pathophysiology, oxidative stress promotes inflammatory pathways - causing the development of diseases, with studies confirming its role in the pathophysiology of autoimmune diseases. [12] The role of antioxidants such as polyphenols in counteracting the effects of oxidative stress, particularly in neurodegenerative and psychiatric diseases, is increasingly being pointed out [13] This may point to additional treatment options in people with PTSD or autoimmune diseases.

Some studies also indicate the importance of the gut microbiota in regulating inflammation by, among other things, maintaining the CD4 + T-cell population in the spleen and systemic neutrophil communities in the blood circulation, suggesting that the microbial population present in the gastrointestinal tract plays a role in regulating immune function. [14]

Endocrine implications

It has been suggested that disruption of the hypothalamic-pituitary-adrenal (HPA) axis is an important mediator of the processes leading to increased vulnerability to disease development in PTSD in addition to significant changes in the immune system. The immune system interacts with the hypothalamic-pituitary-adrenal axis in a bidirectional manner to maintain homeostasis. The presence of dysregulation of the hypothalamic-pituitary-adrenal axis, the primary physiological pathway responsible for the stress response, has been extensively studied as being linked to the pathophysiology of PTSD. [15] Activation of the HPA results in elevated blood levels of glucocorticoids, epinephrine and norepinephrine. After the threat subsides under physiological conditions, HPA axis activity returns to baseline. In a situation of excessive, prolonged stress and exposure to a threatening situation, as in the case of PTSD, there is repeated activation of the HPA axis, secreting large amounts of cortisol, which ultimately leads to impaired reactivity of the hypothalamic-pituitary-adrenal axis. [16] This can result in deregulated glucocorticoid signaling, thereby activating the immune system and increasing inflammation. Physiologically, elevated cortisol levels are observed shortly after exposure to a traumatic experience. However, we often see a different phenomenon in people with PTSD - a meta-analysis found reduced cortisol levels over 24-h. [17] A recent meta-analysis also found lower cortisol levels in morning saliva in patients with post-traumatic stress disorder compared to healthy individuals. [18] At the same time, increased dexamethasone

inhibition has been observed in PTSD [15] Thus, given the current state of knowledge, PTSD is thought to be associated with low plasma cortisol levels and higher glucocorticoid receptor (GR) expression, suggesting increased sensitivity to cortisol feedback. [19] Meta-analyses indicate altered function of the HPA axis, but conclusions remain partially inconsistent. Results of studies that assessed standard hypothalamic-pituitary-adrenal axis activity in people diagnosed with post-traumatic stress disorder have been inconclusive, ranging from lower cortisol secretion in some cases to higher in others. One putative reason for this may be differences in the duration of allostatic stress, as well as single measurements of cortisol levels taken in many studies, which are not considered reliable indicators of HPA axis function. [20] Further studies should be conducted to clarify these mechanisms.

There are studies indicating that people who have experienced trauma and those with PTSD have reduced endogenous oxytocin secretion. Moreover, dysregulation of oxytocin secretion, which has a protective effect in traumatic events, can lead to an increased stress response and increase the risk of developing PTSD. However, it should be considered that the relationship between PTSD and reduced oxytocin is complex and depends on many additional factors. [21]

It seems interesting that the changes in the immune and endocrine systems in the course of PTSD are not static but undergo changes during the course of the disease in parallel with the clinical symptoms of the disorder. This was assessed in one study conducted with Croatian veterans using the CAPS scale - subsequent stages of PTSD are associated with lower inflammatory parameters. These changes depend on the duration of allostatic load and its effect on interactions involved in the stress response. [22]

Genetic implications

Research suggests that genetics is one of the elements contributing to the differential risk of PTSD after trauma. Increasingly, the role of genetic variants involved in the immune response is being highlighted, as this is one important determinant of the increased inflammation found in people with PTSD. The results of genetic and epigenetic studies are promising, identifying similar genes in various research papers. Nevertheless, they are still not entirely conclusive. Understanding the genetic underpinnings of immune dysregulation in PTSD is important because they offer a chance to expand prevention and treatment options.

Genome-wide association studies of GWAS, which identify which genes are responsible for specific pathologies, have identified variants in genes and pathways that are involved in immune regulation and are associated with autoimmune and inflammatory disorders. Ankyrin Repeat Domain-55 gene, whose role has been linked to a number of inflammatory and autoimmune diseases, such as multiple sclerosis, type 2 diabetes, gluten-sensitive enteropathy and rheumatoid arthritis, has been found to be significantly associated in groups suffering from post-traumatic stress disorder. [23] A recent GWAS conducted on Danish soldiers deployed in war and conflict zones identified a significant 4q31 locus highlighting the link between PTSD and inflammation. [24]

A recent study also found an association between HLA alleles and PTSD. [25] HLA allelic variation is associated with a change in haplotype-specific amino acids in antigen recognition binding grooves, which affects the ability to recognize antigen. Some HLA alleles are characterized by increased antigen presentation capacity, resulting in intense T-cell activation and significant production of pro-inflammatory cytokines, which may be associated with the creation of a pro-inflammatory environment in PTSD. In addition, specific HLA alleles have been linked to autoimmune diseases such as rheumatoid arthritis and multiple sclerosis, which also co-occur with PTSD. [26]

One study identified eight HLA alleles associated with PTSD: HLA-B, HLA-C, HLA-DQA1, HLA-DQB1 and HLA-DPB1 were more common in PTSD cases, while HLA-A, HLA-DQA1 and HLA-DRB1 were more common in the control group. [27]

According to evidence in the field of genetics, peripheral HLA gene expression is therefore altered in PTSD.

Changes in inflammatory gene expression associated with PTSD are consistent with changes in epigenetic profiles, due to the regulation of gene expression through changes in DNA methylation. These changes are observed in PTSD. Other studies have shown that individuals with PTSD have reduced methylation at CpG sites located in DOCK2 ZFP57, RNF39 and HIST1H2APS2, among others. [28]

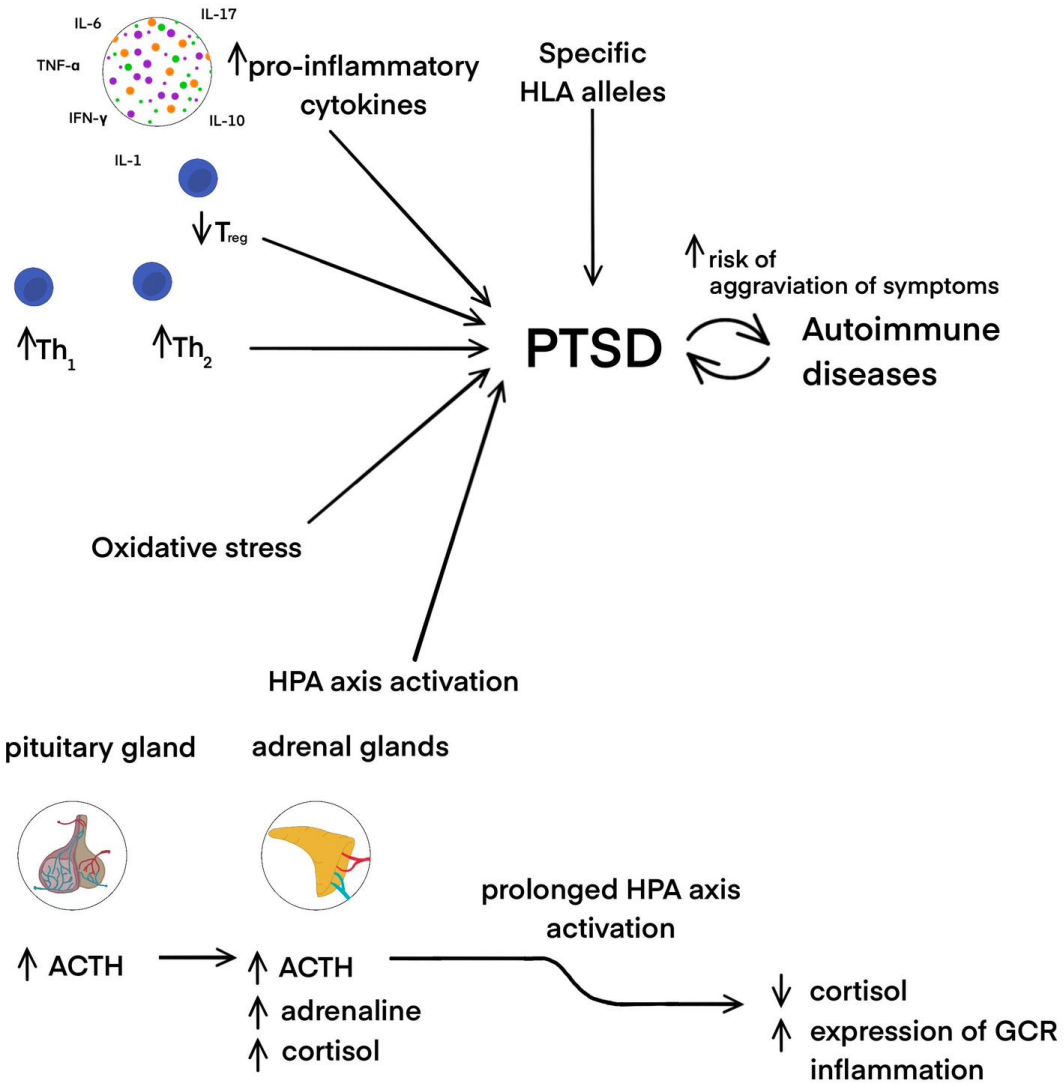

Figure 1. The pathophysiology of PTSD and the interplay between PTSD and autoimmune diseases.

The relationship between PTSD and autoimmune diseases

Autoimmune diseases are a diverse group of conditions associated with abnormal reactivity of B and T cells against healthy host tissues. Autoimmune diseases are more common in women compared to men, due to increased humoral response in women, differences in immunomodulation and immune response to stress. [29] Genetic predisposition, infectious and environmental factors are identified as the most likely etiological factors. Due to their early onset and chronic nature, autoimmune diseases are an important health and economic problem. Recently, there has been a steady increase in the incidence of autoimmune diseases. Estimated

annual increases in the overall global incidence and prevalence of autoimmune diseases are 19.1% and 12.5%, respectively. [30] Due to the increasing incidence, the health care costs associated with these diseases are also increasing. [31] This prompts special attention to understanding the risk factors and pathogenesis of autoimmune diseases.

To date, there have been many studies evaluating the relationships that exist between PTSD and autoimmune diseases.

One study suggesting a link between PTSD and increased risk of autoimmune diseases is a retrospective cohort study of 666,269 Iraq and Afghanistan veterans, which found that veterans with PTSD had twice the risk of being diagnosed with an autoimmune disorder compared to those without a mental disorder and a 51% higher risk compared to veterans with mental disorders other than PTSD. Of all those surveyed, 9,743 (1.5%) received an autoimmune diagnosis. Although the magnitude of the increase in risk associated with PTSD was similar in men and women, women overall had nearly three times the risk of being diagnosed with an autoimmune disorder, due to the overall higher risk of autoimmune diseases in the population in women. Military sexual trauma (MST) was found to be an independent factor associated with the risk of autoimmune disorders in both men and women. [32] In contrast, in another study, results did not differ significantly by combat exposure and by type of physical or sexual trauma. This study, conducted with U.S. military personnel and involving 120,572 participants observed for an average of five years, found that the risk of any of the selected autoimmune diseases was 58% higher in those with a history of PTSD compared to those without a history of PTSD. It was concluded that active duty military personnel with PTSD may have an increased risk of a number of autoimmune diseases, regardless of combat experience or prior trauma. Also, the magnitude of the association between PTSD and each autoimmune disease was found to be similar in men and women. [33]

Also, one recent retrospective cohort study of Swedish civilians found that, those with PTSD and stress-related disorders were more likely to develop autoimmune disorders compared to those without such disorders [34] Other previous cohort work suggests that those who have experienced combat during deployment may be at increased risk of rheumatoid arthritis, compared to deployment without combat experience although it should be noted that participants in this study were not screened for PTSD. Nonetheless, such an association is possible. [35]

Another study estimated that Vietnam veterans with comorbid PTSD had a higher risk of autoimmune diseases, especially rheumatoid arthritis, hypothyroidism and psoriasis. At the same time, exposure to combat was an important predictor of post-traumatic stress disorder.

Social support appeared to be an important factor - the less social support veterans had, the more likely they were to develop PTSD [36] The importance of social support in people with PTSD was also highlighted as an important factor in reducing the severity of symptoms. [37] An association between PTSD and systemic autoimmune diseases was also shown in a study involving 43,133 people who were exposed to the September 11th, 2001, terrorist attack on the World Trade Center in New York, but not in all study groups. A statistically significant association was observed among community members who experienced intense exposure to dust from the collapsed building and who developed post-traumatic stress disorder. The risk of developing a systemic autoimmune disease in this group was more than double that of those without PTSD. In contrast, among rescue workers, the association between PTSD and autoimmune diseases was not statistically significant, although some upward trends were observed. Similar results were found in 2 studies involving mainly male firefighters, which showed that the type of rescue and recovery work and the increased duration of work in the WTC, defined in months, were associated with an increased risk of systemic autoimmune disease. They found that each additional month worked at the WTC site increased the likelihood of developing a systemic autoimmune disease by 13% [38].

A link has also been found between adverse childhood experiences and the development of autoimmune diseases. A US cohort study of 67,516 nurses reported 94 cases of SLE during a 24-year follow-up. They found a significantly increased risk of systemic lupus erythematosus (SLE) among women who had experienced physical and emotional abuse in childhood compared to women who had not. The study found results indicating that 23% of the violence-related SLE risk can be explained by PTSD. Moreover, exposure to trauma independent of PTSD symptoms was strongly associated with the occurrence of SLE. One model that may explain this phenomenon suggests that people with adverse childhood experiences, including socioeconomic disadvantage and abuse, may have increased sensitivity to stress, both psychological and physiological. Additionally, when unfavorable factors like limited stress-coping resources co-occur, psychological dysregulation can further impact the immune system, potentially leading to inflammation. [39] There is also an association between adverse childhood experiences such as emotional abuse, physical violence, sexual abuse, and an increased likelihood of first hospitalization for any autoimmune disease. Children exposed to 2 or more adverse childhood experiences were 2 times more likely to be hospitalized for rheumatic disease compared to unexposed children. [40]

Some researchers have concluded that the association between PTSD and autoimmune diseases may be at least partially explained by unhealthy behaviors such as increased smoking,

sleep disturbances, poor diet, and use of psychoactive substances and alcohol [41] However, another study found that health behaviors-body size, smoking history, and alcohol consumption-did not appear to affect the association between PTSD and risk of selected autoimmune diseases. [33] Thus, research findings on this topic are inconclusive.

There are also reports of an increased risk of PTSD after experiencing trauma in people with autoimmune diseases. A higher risk of PTSD in patients with connective tissue diseases was demonstrated in a prospective observational study conducted during the COVID-19 pandemic involving patients with systemic autoimmune diseases. [42] In a recent study from a group of 99 patients with lupus erythematosus who were tested for PTSD, 31% of patients had a TALS-SR score consistent with PTSD. Thus, post-traumatic stress disorder is likely to be much more prevalent in SLE patients than in the general population and has an adverse impact on their quality of life. [43]

Treatment Implications

The main treatments for PTSD are psychotherapy and pharmacotherapy with psychotropic drugs. However, these classic therapies still fail to bring remission of PTSD symptoms in many patients. Considering the heightened risk of autoimmune diseases in individuals with PTSD, effective treatment strategies may include immune modulation, in addition to previously mentioned therapies, to target the underlying inflammatory response.
The strongest evidence has been found for psychological treatments for PTSD, particularly cognitive-behavioral therapy with a focus on trauma (CBT-TF), cognitive processing therapy (CPT), cognitive therapy (CT), eye movement desensitization and reprocessing (EMDR) and long-term exposure (PE). In addition, pharmacologically assisted psychotherapy for adults with post-traumatic stress disorder is an important area of treatment, with MDMA-assisted psychotherapy appearing to be the most promising of the interventions considered. In contrast, pharmacological treatment primarily includes SSRIs, which are first-line medications for treating post-traumatic stress disorder. Fluoxetine, paroxetine, sertraline and venlafaxine have shown particularly good evidence of reducing PTSD symptoms in adults. In addition, they have also been shown to exert anti-inflammatory effects, which is important in the context of PTSD pathophysiology. [44] Prazosin is another drug used to treat post-traumatic stress disorder, and it also has additional anti-inflammatory effects.

As mentioned earlier, PTSD and inflammation appear to be closely linked. For this reason, the development of new drug therapies for PTSD based on biological mechanisms of inflammation is currently attracting considerable interest.

Studies in animal models are emerging proposing the use of drugs that affect the reduction of inflammation, such as ACE and ARB inhibitors and non-steroidal anti-inflammatory drugs. In addition, treatment with exogenous corticosteroids has been studied in patients with post-traumatic stress disorder, and has been shown to alleviate PTSD symptoms, either given alone or in combination with psychotherapy. Also, treatment with monoclonal antibodies such as infliximab (anti-TNF-α), adalimumab (anti-TNF-α), and tocilizumab (anti-IL-6 receptor) may be useful in suppressing increased inflammation by blocking cytokines in people with PTSD. Cannabis, as a psychotropic substance with anti-inflammatory properties, has been proposed as a potential treatment for PTSD. However, few studies have shown possible efficacy. At the same time, the method additionally poses a risk of addiction in cases of psychiatric disorders. There have also been proposals to use deep brain stimulation (DBS) to treat PTSD. In animal models, it has been shown that vagus nerve stimulation (VNS) can eliminate PTSD-like symptoms such as anxiety and social avoidance. In addition, the potential beneficial effects of health-promoting behaviors such as exercise and diet used as an alternative or adjunct to treatment have been reported in patients with post-traumatic stress disorder. [45] [46]

Intranasal oxytocin administration has also been proposed as a potential way to normalize brain function in PTSD. Oxytocin administration in patients with post-traumatic stress disorder was associated with normalization of functional connections between the ventral medial prefrontal cortex and the amygdala and enhanced the function of brain areas particularly involved in emotion processing, working memory and reward [47] For this reason, oxytocin administration appears to be a promising therapeutic strategy for patients with PTSD. However, current findings suggest that it may only be helpful for a limited, strictly selected group of patients. [48]

CURRENT TREATMENTS	POTENTIAL TREATMENTS
Psychotherapy (CBT-TF, CPT, CT, EMDR, PE) MDMA-assisted psychotherapy	ACE and ARB inhibitors (Captopril, Telmisartan)
SSRI (Fluoxetine, Paroxetine, Sertraline, Venlafaxine)	Glucocorticoids (Hydrocortisone, Prednisolone, Dexamethasone)
Prazosin	Monoclonal antibodies (Infliximab, Adalimumab, Tocilizumab)
	NSAIDs (Celecoxib, Ibuprofen, Naproxen)
	Deep brain stimulation (DBS) Vagus nerve stimulation (VNS)
	Intranasal Oxitocin
	Cannabis
	Ketamine

Figure 2. The current and potential treatment of PTSD.

Summary

PTSD is considered one of the more complex disorders, influenced by a number of biological, social and psychological factors. **A growing body of evidence suggests** that, in

addition to the well-known psychiatric symptoms of PTSD, distinct changes occur in the neuroendocrine and immune systems of individuals suffering from the disorder, likely due to chronic stress exposure. These dysregulations link stress-related disorders such as PTSD to somatic diseases, including autoimmune diseases, and may be associated with premature mortality. The link between PTSD, immune system function, and autoimmune diseases is bidirectional. Many studies indicate that both PTSD and immune disorders, due to certain similarities in pathophysiology, can exacerbate each other.

Current treatment options for PTSD still have limited efficacy in many patients. **Effective treatment for PTSD likely requires a multidisciplinary approach** including psychotherapy, social support, SSRI medications, and healthy lifestyle habits. Additionally, anti-inflammatory and immunomodulatory treatments, while still under investigation, hold promise for future inclusion.

Awareness of the potential increase in the risk of autoimmune diseases in patients with PTSD provides opportunities for early detection of these diseases, faster implementation of therapies when they occur, and finding new treatments. The literature summarized here suggests that intervention strategies targeting neuroendocrine and immune dysfunctions, such as anti-inflammatory and immunomodulatory treatments, may prove beneficial in the treatment of PTSD. Moreover, given the aforementioned link between PTSD and autoimmune diseases, recognizing and treating post-traumatic stress disorder may reduce the risk of autoimmune diseases, as well as improve treatment outcomes in people with concurrent autoimmune diseases. In turn, effective treatment of autoimmune diseases may reduce the risk of PTSD, as well as improve the course of PTSD in a group of patients with comorbidities.

Interestingly, the use of AI may be useful in the early detection of PTSD, as reported recently. This may affect the early implementation of treatment, and thus may lower the risk of developing PTSD-related psychiatric disorders and somatic diseases. [49]

Further research is needed to clarify the relationship between inflammation and post-traumatic stress disorder, as well as on the mechanisms affecting the risk of developing autoimmune diseases in people with PTSD. Also important is further research into new therapies, such as anti-inflammatory treatment, that have the potential to alter the course of PTSD and reduce the risk of autoimmune diseases.

Conclusions

Based on the studies analyzed in this paper, it can be concluded that there is a correlation in PTSD and autoimmune diseases. People with PTSD are more likely to develop autoimmune diseases. Some studies also indicate an inverse correlation - an increased risk of PTSD in people after experiencing trauma who had a previous diagnosis of autoimmune disease. These findings may indicate a reciprocal effect of these two disease entities, especially given the many correlating factors involved in their pathophysiology.

Thus, people with PTSD may need to be monitored for the potential development of an autoimmune disease and receive treatment if necessary. Conversely, people with autoimmune diseases may benefit from PTSD evaluation. Moreover, effective treatment for PTSD, with consideration of new potential treatments, could potentially alleviate the symptoms of both conditions.

Disclosure:

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Project administration: Anna Kajka;

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