

The Relationship between Traumatic Stress, PTSD and Cortisol

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Many researchers have studied how trauma and posttraumatic stress disorder (PTSD) impact cortisol, a primary stress hormone. Although cortisol dysregulation is common in the general population, PTSD appears to hasten cortisol imbalance and its extensive consequences, making it an important area of continued study. The following paper provides a brief review of the documented relationship between traumatic stress and cortisol, as well as an overview of how cortisol responds to clinical treatments targeting PTSD.

The stress response

In his seminal work, Hans Selye¹ defined stress as “the non-specific response of the body to any demand placed upon it” and noted that the body’s reaction to stress (also referred to as the *general adaptation syndrome* or the *stress response*) can be activated by both actual and perceived demands.

When a stressor is identified, the hypothalamic-pituitary-adrenal (HPA) axis activates the “fight-or-flight” response. Cortisol, released by the adrenal gland, plays a key role in directing physiological and metabolic processes away from long-term management to immediate survival (e.g., increases in heart rate, decreases in digestion, alterations in immune functioning) and then works together with dehydroepiandrosterone (DHEA) to bring the body back to a normal state². For a detailed review of the stress response, please refer to Selye³.

Cortisol dysregulation and its consequences

Prolonged activation of the stress response can compromise the body’s internal stability, resulting in HPA axis dysregulation and alterations in cortisol levels. Chronic illness and disease can then ensue due to cortisol’s impact on the immune system. Enhanced cortisol activity suppresses cellular immunity, increasing susceptibility to infection and

neoplasm (abnormal growth of tissue), while low cortisol levels stimulate pro-inflammatory cytokines, which can lead to autoimmune diseases and malignancy².

Endocrine disorders can be another consequence of abnormal cortisol functioning. High levels of cortisol decrease the liver's sensitivity to insulin (i.e., insulin resistance), which increases glucose levels in the blood. If left untreated, high blood glucose can lead to kidney, neurological and cardiovascular damage⁴.

Mental health and cognitive problems may also develop from cortisol dysregulation. Hypercortisolism is associated with obsessive-compulsive disorder, panic disorder and melancholic depression, while hypocortisolism has been linked to depressed mood, chronic pain, sleep disturbances and fatigue². Additionally, cortisol's ability to bind to receptors in the hippocampus (the brain region involved in memory) can impact memory and consciousness. An over-production of cortisol can shrink and cause atrophy of the hippocampus, leading to memory difficulties⁵. Severe hippocampal atrophy may result in periods of dissociation⁶.

Cortisol and PTSD

It is well documented that individuals with PTSD have altered cortisol levels, yet the direction of impairment (i.e., too high or too low) is mixed. Yehuda and colleagues⁷ showed that chronic PTSD was associated with lowered cortisol activity compared to those without a PTSD diagnosis and suggested that chronically high stress levels may exhaust the HPA axis. Other studies have found higher cortisol activity in those with PTSD. One research team found that compared to controls, Vietnam combat veterans with PTSD had higher overall cortisol levels⁸. Another study documented that Croatian combat veterans had fewer glucocorticoid receptors (receptors that cortisol binds to) compared to healthy controls⁹, which could also contribute to higher levels of circulating cortisol.

One study found that child abuse victims with PTSD experienced enhanced cortisol activity in response to exposure to traumatic reminders, bringing researchers to conclude that low levels of baseline cortisol may compensate for periods of higher cortisol levels that accompany stress¹⁰. Still, some researchers have documented normal

cortisol levels in a sample that consisted of individuals diagnosed with PTSD from varying types of events (e.g., childhood trauma, domestic violence, war)¹¹.

Family and individual factors are important to consider when examining the relationship between PTSD and cortisol activity. Yehuda and colleagues¹² documented lower cortisol excretion in children of holocaust survivors with PTSD compared to healthy controls. Further, children who had two parents with PTSD had lower levels compared to those who only had one parent with PTSD. The authors note that the impact of parental PTSD on the child's cortisol level could be related to both biological mechanisms and the environment in which the child is raised (e.g., parental neglect).

Avoidance, a hallmark symptom of PTSD, may also play a significant role in the relationship between cortisol and PTSD. Research has shown that the engagement-nonengagement style of coping influences cortisol levels^{13,14} and that nonengagement has been associated with low levels of cortisol¹⁵. These findings may explain some of the variability in cortisol findings across PTSD populations. Those patients who avoid and withdraw to a greater extent may have lower cortisol levels. Additionally, cortisol levels vary throughout the day and in different situations within the same individual¹⁶. Thus, times of avoidance, withdrawal and isolation may be associated with lower cortisol levels, while re-experiencing and hyperarousal are related to enhanced cortisol activity.

Cortisol and PTSD treatment

Given the neuroendocrine dysregulation in those with PTSD, researchers have begun to study the impact of PTSD treatments on cortisol levels. Olff, de Vries, Guzelcan, Assies and Gersons¹⁷ examined cortisol response to trauma-based cognitive-behavioral therapy (CBT) in 21 individuals with PTSD due to civilian traumas. They found that successful treatment, assessed by the Structured Interview for PTSD (SI-PTSD) and self-report symptom measures (i.e., Impact of Event Scale [IES], Beck Depression Inventory [BDI]), was associated with enhanced levels of basal cortisol and DHEA at post-treatment. However, the improvements in hormonal measures were only seen when depression symptoms were included in the model.

Using a sample of 28 trauma survivors from the 9/11 attack of the World Trade Center, Yehuda and colleagues¹⁸ monitored individuals' cortisol levels as they participated in psychological treatment. Basal cortisol and PTSD severity (assessed by the Posttraumatic Stress Symptom-Interview [PSSI] and the PTSD Symptom Scale-Self Report [PSS-SR]) were collected before and after treatment. At pre-treatment, cortisol indicators (5-alpha reductase activity, total glucocorticoids) were lower for those who had higher avoidance scores but for no other symptom cluster (i.e., re-experiencing, hyperarousal). At post-treatment, 5-alpha reductase activity was significantly correlated with all three PTSD symptom clusters, as well as total severity scores. Overall, these findings indicate that those who were highly avoidant showed lowered cortisol activity, and successful treatment increased cortisol levels.

Gerardi, Rothbaum, Astin and Kelly¹⁹ were the first researchers to use a randomized control design when examining cortisol response to PTSD treatment. Sixty women with PTSD were randomly assigned to prolonged exposure (PE), eye-movement desensitization and reprocessing (EMDR) or waitlist. Measures were taken at three time points: at baseline, immediately after session 3 (first exposure session) and immediately after session 9 (last exposure session). Results showed that treatment response (i.e., at least a 50 percent reduction in PTSD symptoms assessed via the Clinician-Administered PTSD Scale [CAPS]) was associated with decreased cortisol levels. Cortisol measurements taken immediately after exposure sessions may explain why effective treatment was related to lower cortisol levels, whereas previous studies documented increased cortisol in response to treatment.

Summary and future directions

Cortisol plays a key role in physical and mental well-being. Research has shown that those with chronic PTSD often have dysregulated basal cortisol levels, yet individual and family factors (i.e., extent of isolation, parental PTSD) may also play a role. Investigators have begun to study the impact of PTSD treatment on cortisol activity and have found that clinical treatments have the potential to stabilize cortisol levels. However, a significant limitation in this line of study is the lack of prospective designs. Since cortisol activity prior to the traumatic event is often unknown, causation cannot be

established. Although it is often assumed that traumatic events alter cortisol levels, it is also possible that trauma survivors who develop PTSD had low cortisol activity before the event, which increased their vulnerability to developing the disorder. Another limitation is the various ways in which cortisol is assessed, making these measurements difficult to compare without the use of meta-analytic strategies. With continued research, further inquiry to how cortisol interacts with trauma may hold promise in helping improve detection and treatment of PTSD and other trauma-related problems.

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