


The Reward System and Post-Traumatic Stress Disorder: Does Trauma Affect the Way We Interact With Positive Stimuli?

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Abstract

Post-traumatic stress disorder (PTSD) is a highly prevalent disorder and a highly debilitating condition. Although anhedonia is an important construct of the disorder, the relationship between PTSD and reward functioning is still under-researched. To date, the majority of research on PTSD has focused on fear: fear learning, maintenance, and extinction. Here we review the relevant literature—including clinical observations, self-report data, neuroimaging research, and animal studies—in order to examine the potential effects of post-traumatic stress disorder on the reward system. Our current lack of sufficient insight into how trauma affects the reward system is one possible hindrance to clinical progress. The current review highlights the need for further investigation into the complex relationship between exposure to trauma and the reward system to further our understandings of the etiology of PTSD.

Keywords

anhedonia, emotional numbing, MDD, negative valence, positive valence, PTSD, reward system, stress, trauma

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Introduction

Post-traumatic stress disorder (PTSD) was established as a psychiatric disorder in 1980.¹ Approximately 6–8% of the general population will be diagnosed with PTSD at some point in their lives,^{2–5} which makes the diagnosis quite prevalent. This is slightly lower than previous estimates because the 5th edition of the American Psychological Association's (APA) Diagnostic Statistical Manual (DSM) narrowed its diagnostic parameters on criterion A (see Table 1) by excluding illness-related traumas and traumas witnessed only via electronic media.^{3,6}

PTSD is often characterized by its maladaptive fear responses following a traumatic event (e.g., a combat veteran with a startle response to ordinary noises and movements). To date, the majority of PTSD research has focused on the inhibition system—fear learning, maintenance, and extinction—due to the conspicuous nature of the disorder's fear-related symptoms. Though less researched, PTSD also involves depressive symptoms, such as anhedonia and emotional numbing (which relate to the reward system). The relationship between PTSD and the reward system is not well

characterized,^{7–10} and current research is not definitive regarding whether reward system deficits are distinct from negative valence symptoms.^{11–13}

Appropriately characterizing PTSD symptomatology related to the reward system enables professionals in the field to effectively identify predictive factors for PTSD and properly diagnose the disorder. Additionally, diagnostic clarity would enable the development of treatments that go beyond reversal of fear,^{8,14} which may assist in avoiding poor outcomes, such as health risk behaviors and chronic PTSD (which have been positively correlated with depressive symptoms in PTSD^{8,15}). The aim of this article is to synthesize relevant literature and aid in the overall understanding of the effect of trauma on the reward system.

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Table 1. PTSD criteria across DSM editions.

Criteria	DSM III (1980)	DSM III-R (1987)	DSM IV (1994), DSM IV-R (2000)	DSM V (2013)
A	Significant stressor	Significant stressor <ul style="list-style-type: none"> • Outside of normal experience 	Significant stressor <ul style="list-style-type: none"> • Possibility of death, serious injury, or risk to physical integrity (e.g. sexual assault) • Induced fear, helplessness, or horror • Was experienced, witnessed, or learned about • Can be illness-related 	Significant stressor <ul style="list-style-type: none"> • Possibility of death, serious injury, sexual violence • Must be violent or accidental (not medical or illness-related) • Was experienced, witnessed, or learned about (not through media)
B	(1x) <ul style="list-style-type: none"> • Intrusive memories • Nightmares • Flashbacks 	(1x) <ul style="list-style-type: none"> • Intrusive memories • Nightmares • Flashbacks • Symptom triggering 	(1x) <ul style="list-style-type: none"> • Intrusive memories • Nightmares • Flashbacks • Symptom triggering • Physiological reactivity 	(1x) <ul style="list-style-type: none"> • Intrusive memories • Nightmares • Flashbacks • Symptom triggering • Physiological reactivity
C	(1x) <ul style="list-style-type: none"> • Anhedonia • Detachment • Limited affect 	(3x) <ul style="list-style-type: none"> • Anhedonia • Detachment • Limited affect • Dread • Avoidance (thoughts/feelings) • Avoidance (activities/situations) • Inability to recall trauma details 	(3x) <ul style="list-style-type: none"> • Anhedonia • Detachment • Limited affect • Dread • Avoidance (thoughts/feelings) • Avoidance (activities/situations) • Inability to recall trauma details 	(1x) <ul style="list-style-type: none"> • Avoidance (thoughts/feelings) • Avoidance (activities/situations)
D	(2x) <ul style="list-style-type: none"> • Hypervigilance • Sleep disturbance • Guilt • Impaired memory • Avoidance • Symptom triggering 	(2x) <ul style="list-style-type: none"> • Hypervigilance • Sleep disturbance • Anger • Impaired concentration • Exaggerated startle • Physiologic reactivity 	(2x) <ul style="list-style-type: none"> • Hypervigilance • Sleep disturbance • Anger • Impaired concentration • Exaggerated startle 	(2x) <ul style="list-style-type: none"> • Guilt/blame • Anhedonia • Detachment • Inability to recall trauma details • Emotional numbing • Negative beliefs • Negative emotional state
E	N/A	N/A	N/A	(2x) <ul style="list-style-type: none"> • Hypervigilance • Sleep disturbance • Anger • Impaired concentration • Exaggerated startle • Impulsivity
Classification	Anxiety disorder	Anxiety disorder	Anxiety disorder	Trauma/stress disorder

DSM: Diagnostic and Statistical Manual of Mental Disorders.

The Reward System: Function and Dysfunction

The reward system refers to the aggregate of neural circuits that process appetitive stimuli—including the limbic system (septal area, thalamus, hypothalamus, amygdala), basal ganglia (containing the ventral and dorsal striatum), prefrontal cortex (ventromedial

prefrontal cortex, in particular), ventral tegmental area (VTA), and substantia nigra.⁸

Neural structures were first implicated in reward processes in the mid-1900s when studies started employing deep-brain stimulation^{16–23} to elicit reward responses, resulting in self-reported feelings of pleasure and a disinclination to cease self-stimulation.^{17,18,24} Findings were

replicated across settings with the use of multiple neuroimaging technologies.^{18,25}

The reward system is now understood to rely on the functioning of neural structures, circuits, and neurotransmitters including dopamine, serotonin, and norepinephrine. In a properly functioning reward system, the anticipation or acquisition of a reward will catalyze a cascade of events, eventuating in the release of dopamine, which travels along the dopaminergic pathways^{26–30} to critical structures (the cerebral cortex, striatum, and pituitary gland).^{29,31–33} Dopamine is involved in signaling reward prediction error and creating incentive salience, therefore motivating and reinforcing behavior.^{29,33–37} Serotonin has a large role in mediating reward enjoyment, motivation, and learning. High serotonin levels will increase inhibition and feelings of satiation, modulating dopamine's effects on reward reinforcement.^{38–42} Norepinephrine affects the mobilization efforts required to obtain a reward.^{32,43} The cannabinoid and opioid systems are also implicated in this process. Some evidence exists to suggest that the cannabinoid system has a role in reward signaling and in modulating one's risk for developing reward-related deficits following stressors.^{44,45} Additionally, the opioid system may help to regulate motivation and reward seeking.^{46–48} All of these mechanisms work in tandem to create reward satisfaction and encourage reward-seeking behavior—though the scientific community's understanding of these mechanisms is relatively new, and further research is warranted.

Dysfunction in reward mechanisms can occur naturally (e.g., natural dopamine decline caused by social isolation^{49–51} or serotonin decline caused by aging⁵²), or artificially (e.g., introduction of dopamine antagonist,^{53–55} drug-induced blocking of CB1 receptors,⁴⁵ or opioids modifying their receptors as addiction develops⁴⁶), or caused by illness^{34,56,57} and genetic disorders (e.g., genetic depletion or malformation of the CB1 receptors^{44,45}). Dysfunction in these mechanisms is characterized by reward learning and motivation deficits⁵⁶ and emotional abnormalities:⁸ a lack of pleasure or satisfaction (anhedonic symptoms^{34,57,58}), reduction in motivation,^{29,33,37,59} and emotional numbing.⁶⁰

PTSD and Dysfunction in Reward System Mechanisms

Researchers are still trying to understand if and to what extent trauma may be a natural cause of reward system dysfunction. Evidence does support the existence of reward learning deficits in the neural circuitry of those with PTSD, as demonstrated by limited activation in brain regions during both the anticipation of (“expectancy phase”) and experiencing of (“outcome

phase”) reward.^{61–63} When actively participating in a rewards task, participants with PTSD displayed reduced neural activation in the nucleus accumbens and in the mesial prefrontal cortex, and they were slow to learn which series of responses would lead them to a reward.⁶⁴ When passively experiencing rewards, PTSD-sufferers have shown limited blood oxygen level-dependent (BOLD) changes in the amygdala and striatal subregions (bilateral nucleus accumbens, bilateral caudate, bilateral putamen) in comparison to healthy controls.⁶² Abnormalities, varying by factors such as age of trauma onset, brain region, and sex, also exist in the cannabinoid^{65,66} and opioid systems as a result of trauma.^{67,68} Neurobiological symptoms are supported by animal model research, which correlates core PTSD symptomatology (e.g., mouse behaviors related to avoidance, fear extinction, arousal) to impaired neurocircuitry in the amygdala, nucleus accumbens, caudate, and putamen.⁶⁹

Trauma is also associated with emotional abnormalities in neural structures. One study found that, compared to their healthy counterparts, those with PTSD have less activation in left parahippocampal gyrus, left fusiform gyrus, left bilateral temporal pole (regions often indicated in emotional processing), when they are exposed to positive valence images.⁷⁰ Another study found that participants with PTSD had less activation in their amygdala and in their ventral striatal-limbic networks than healthy controls did when passively presented with a happy face. The PTSD group also reported the faces as appearing less happy than they appeared to the control group.⁷¹

Clinical observation has supported the relationship between trauma and emotional abnormalities: anhedonia,⁷² emotional numbing,^{72,73} diminished positive affect⁸ (present even without comorbid diagnoses of MDD⁷⁴), and deficits in motivation (present without any coexisting cognitive deficits that could be confounding⁶⁴). Similar symptoms are observed in animal models. (When stress is introduced, rats show a decreased interest in rewards⁷⁵).

Self-report data corroborates these findings, further suggesting a correlation between PTSD and deficits in reward learning. Those with PTSD report getting less satisfaction from rewards than their healthy control counterparts report getting; this includes both passively and actively experiencing the anticipation and outcome of a reward.^{62,76} Participants with PTSD have reported fewer positive emotions associated with rewards⁷⁶ and less satisfaction with the rewards even when there was little expectation of receiving a reward at all.⁷⁷ Healthy controls, comparatively, reported experiencing more pleasure from and satisfaction with the rewards, and they were particularly pleased when the reward was unexpected.⁷⁷ One theory postulates that this phenomenon exists because those with PTSD have a higher

threshold for positive stimuli and require stronger positive stimuli to meet said threshold.⁷⁸

In addition to trauma being linked to emotional abnormalities and reward learning deficits, there is also a connection between PTSD and dysfunction in reward-related neurobiology. Dopamine is crucial to reward functioning,^{55,58} and severe stress has been associated with a reduction in dopaminergic neural activity in humans and in animal models.^{75,79–82} The reciprocal is also true: In mice, the introduction of dopamine agonists has been shown to reduce PTSD-like stress symptoms.⁸³ In human neuropharmacological studies, artificial increases in dopamine, norepinephrine, or serotonin can have antidepressant effects in PTSD.^{84,85}

PTSD and External Reward Seeking

Abnormal reward-seeking and maintenance behaviors (risky behaviors) are innate in the PTSD diagnosis (criterion E2⁶). PTSD symptom severity positively correlates with risk-taking behaviors, including thrill seeking and risky sexual behaviors.^{86–88} Notably, PTSD is also highly comorbid with substance abuse:^{80,89–92} According to the American Addiction Center, 50–66% of PTSD-sufferers have comorbid substance abuse issues. According to the European Monitoring Centre for Drugs and Drug Addiction, depression—a disorder highly defined by its reward-system deficits—has the highest substance abuse comorbidity prevalence of all psychiatric disorders.⁹³

High levels of external reward seeking may be evidence of reward system dysfunction in PTSD. Dopamine-inducing behaviors and substances (such as alcohol, opioids, nicotine, and cannabis) are self-reinforcing.^{37,94} The self-proctoring of external thrills and psychoactive substances is an in-vivo representation of neurobiological self-stimulation studies,^{93,95} which have been mainstays in reward system research. Those experiencing emotional abnormalities may be in search of external stimulation. This can lead to risky behaviors and addictive substances—as dopamine levels increase as a result of reward system activation, serotonin levels decrease,^{38,41} and leave the individual without a sense of satiation and in search of additional stimulation.^{39,42,96} Possibly adding to the effect: High levels of dopamine are thought to encourage impulsive behaviors, while high levels of serotonin will suppress them.⁹⁷ With low levels of serotonin and high levels of dopamine, reward seekers are left unencumbered by inhibition and in search of an ever-elusive feeling of satiation.

Opposing Theories

Despite strong evidence, the theory that trauma affects the reward system is not universally accepted. There

have been alternative theories as to why neural correlates present differently in PTSD patients than they do in healthy controls. One theory speculates that hyperarousal (a negative valence symptom associated with PTSD (see Table 1)) uses up cognitive resources, diverting resources away from tasks and non-trauma-related topics.⁷⁸ Animal model studies have suggested that performance in cognitive and memory-based tasks is impaired when there is exposure to high stress,⁹⁸ which could explain why human research studies detect limited BOLD activation during reward learning tasks.^{61–63}

Another theory states that PTSD sufferers are not lacking the ability to feel positive emotions. Rather, they lack the ability to label and/or express these emotions. Comparisons between PTSD and non-PTSD samples have suggested a correlation between alexithymia and a diagnosis of PTSD.^{99–101} Alternatively, individuals could be strategically concealing their emotions. One study found that veterans with PTSD reported purposely concealing their emotions more often and more intensely than veterans without PTSD did.¹³ Researchers in this study, however, did acknowledge that any active retention of emotion was most likely a facet of an overarching emotional deficiency and not a primary symptom in itself.

Another differential conclusion is that comorbidities are the primary contributor to reward deficits in PTSD, given the high rate of comorbidity with substance abuse^{89–91} and MDD.¹⁰² With this explanation, comorbid disorders would explain a majority of the observed reductions in appetitive functioning.¹² PTSD symptomatology is complex and can have significant overlap with other disorders, which makes understanding the source of reward system deficiencies challenging.

Complexity and Heterogeneity

If the field is to carefully consider the possible effects of PTSD on the reward system, it will be important to reconcile these opposing theories, potentially by transparently acknowledging the disorder's complexity and heterogeneity. One facet to consider is that the existence and severity of reward dysfunction in PTSD may vary across demographic categories. Research has suggested that older adults may suffer more reward dysfunction following trauma than younger adults will.^{103,104} Although not fully studied, broader research does support the existence of variation in PTSD symptom expression across race, gender, and age—affecting arousal,^{105–107} avoidance behaviors,^{103,107–109} and emotional regulation^{110,111}): Evidence suggests that Black individuals are generally likely to experience more avoidance symptoms than white individuals will,^{108,109} and women more so than men.^{103,107} Also suggested is that Black individuals are generally likely to experience more

hyperarousal symptoms than white individuals will,¹⁰⁵ elderly adults more than younger adults,¹⁰⁶ and women more than men.¹⁰⁷ More research in this area is warranted, specifically as it relates to the reward system.

Symptom variation may, in part, be explained by differing trauma types across populations.^{109–114} For example, racial trauma may produce a different set of coping strategy than other traumas¹⁰⁹ and sexual trauma may affect emotional regulation skills, mitigating gender differences.^{110–112}

Heterogeneity across populations is important to address, as ignoring it could lead to systematically underdiagnosing or overdiagnosing certain groups, which may further result in undertreating or mistreating. Chart-review research on the detection of PTSD offers a look into the real-world consequences involved when diagnostic criteria do not equally represent different groups of people and their respective symptomatology: Among those with a confirmed diagnosis of PTSD, women, Native Americans, and Black men were most likely to have been diagnosed with PTSD upon first consulting with a mental healthcare professional (as opposed to later on in treatment).^{2,115} A gap in knowledge exists when it comes to understanding presentations of PTSD across groups.

Some theories propose that heterogeneity in PTSD symptomatology would be best explained and controlled for in diagnosis by considering the possible existence of PTSD subtypes. The theory that PTSD has two emotional dysregulation subtypes^{68,116,117} is already relatively accepted in the literature. Relevant theories propose that there are also subtypes of PTSD that can be characterized by reward dysfunction.^{90,116,118} Neuroimaging research also shows that individuals with PTSD can display both overmodulation and under-modulation of the medial prefrontal cortex (PFC), which dynamically and continuously regulates the limbic system (relating to emotional modulation).¹¹⁶ These findings suggest that the expression of PTSD not only varies across people but is also dynamic within subjects. A unipolar definition of any PTSD symptomatology may be neglecting these complex aspects of PTSD presentation.

Limitations

Current research on PTSD's effect on the reward system is limited, and there are a number of obstacles involved in exploring the topic—barriers that stunt research but are also a reflection of gaps in knowledge and the need for further exploration. For one, researchers face a lack of definitional clarity.¹⁴ The American Psychological Association offers vague—if not conflicting—information about the current PTSD diagnosis criteria as they relate to the valence systems; the DSM 5 cites examples of valence over-reactivity and valence under-reactivity.⁶ Looking specifically at emotional abnormalities, we see

DSM discussion of both increased emotional reactivity (criterion B) and emotional numbing (D) (see Table 1). And while some reward system dysfunction is acknowledged (e.g., emotional numbing, anhedonia), the DSM evades a commitment to these criteria by allowing a diagnosis in the absence of any reward deficit symptoms.

Substantial changes between DSM editions (see Table 1) pose a similar obstacle. The DSM IV, for example, presents emotional numbing and avoidance in a single cluster of PTSD symptoms, whereas the DSM V separates these symptoms and goes on to define emotional numbing solely as the numbing of *positive* emotions—a specification not previously noted and one that is still not ubiquitously accepted or unanimously utilized in discourse.^{4,6,119–121} What may seem like technicalities create barriers in communication and can ultimately affect the reexamination of PTSD criteria and treatment.

Future Directions

Nonresponse and drop-out rates are high in PTSD treatments,^{122–127} and only about a third of people diagnosed with PTSD fully recovered from it.¹²⁸ Much work needs to be done on researching the disorder and its treatment, and part of this work needs to be on understanding trauma and the reward system. In line with the Research Domain Criteria (RDOC^{10,100}), future research could use multi-method procedures to assess the effects of PTSD on the reward system as well as potential bidirectional effects.

Research should, first, work to confirm if there are reward system deficits that accompany PTSD. While there is evidence to this end, some suggest alternative readings of the findings.^{78,98,99,101,102,129} One method to approach this question might be to try to measure reward deficits in PTSD subjects when comorbidities, such as threshold and subthreshold substance abuse, MDD, and alexithymia, are controlled for.

If reward dysfunction in PTSD attracts further scientific support, the next step may be to work toward understanding the heterogeneity of reward functioning in PTSD. Not understanding how the presentation of PTSD can differ across individuals and groups will affect diagnosis prevalence and may leave certain groups of people underdiagnosed or overdiagnosed. A proper diagnosis is a gateway to a proper treatment. If certain groups of people are underdiagnosed or misdiagnosed due to their symptom presentation, clinicians may be systematically excluding these groups from receiving appropriate treatment.^{105,108,112} Does chronic trauma put an individual at risk of developing reward deficits more than an acute trauma does? Does one's age, gender, race, ethnicity, or type of trauma increase the chances of developing these deficits?

Once these steps are taken, future research may include efforts to improve the effectiveness and tolerability of PTSD treatments by targeting reward-related symptoms. Prolonged exposure is currently regarded to be the gold standard of PTSD-targeted psychotherapies, particularly for those with chronic PTSD.¹²³ While generally considered effective, aiding in about a 40%–70% reduction in symptoms,¹³⁰ up to 50% of patients do not respond, and the treatment's rigor results in high drop-out rates.^{123–126} Medication trials also result in high rates of discontinuation prior to completion of the recommended course.¹³¹ If reward-related deficits in PTSD were better correlated to certain factors (e.g., trauma type or age increasing the likelihood of developing reward dysfunction) or if subtypes were identified and clearly defined, perhaps treatments could be better targeted. More precise treatments would not only carry the potential to become more effective than generalized treatments but would also aid in possibly attenuating patient exhaustion. One avenue to explore may be investigating the use of rapid-acting medications and their use in augmenting traditional psychotherapies. Ketamine, in particular, is promising, as it has been lauded for its antidepressant effects in patients with severe MDD^{132,133} and is currently being researched for its potential use in PTSD treatment.^{122,134–136} As a fast-acting antidepressant with potential long-term benefits, ketamine could make for a tolerable and effective therapeutic tool for addressing reward dysfunction in PTSD.

A final suggestion of future directions is to study the bidirectionality of trauma and reward dysfunction. Studies have shown both predisposing and acquired neural abnormalities related to PTSD. Hyperactivity in the amygdala and the dorsal anterior cingulate cortex are recognized as predisposing factors for vulnerability to PTSD; while decreased connection between the hippocampal and ventromedial prefrontal cortex is regarded as acquired impairment.¹³⁶ This line of research has historically focused on the processing of threats and fear, and more needs to be studied regarding the processing of positive rewards. While it is important to recognize the contribution of trauma to reward system impairments, it is likewise useful to be aware of the concern that this relationship could move in both directions. Does an altered reward system predispose an individual to be more susceptible or more resilient to psychopathology following a traumatic event? Understanding such questions will give new insights to preventive interventions and treatments for PTSD.

Conclusion

It has been 40 years since the term “post-traumatic stress disorder” was coined. PTSD remains highly prevalent.^{2–4} Our lack of insight into trauma's effect on the reward

system is one possible hindrance to clinical progress. When we start examining the relationship between trauma and the reward system, important questions come about regarding the diagnosis and treatment of PTSD. For example: Should reward system deficits be more central in PTSD diagnostic criteria? Should we be treating PTSD based on its subtype? Should psychotherapy for PTSD include affect-focused treatment? What is known is that those with PTSD have low levels of dopamine, reduced reactivity in neural areas associated with the reward system, lower reports of reward satisfaction, and a higher tendency to seek external—sometimes risky—rewards. These correlations merit some attention. Ultimately, further research is necessary in order to address PTSD in a more complete way, one that appreciates the heterogeneity of the disorder and is culturally competent. When more is known about PTSD criteria, including their relation to reward system deficits, the field will be better enabled to create targeted, effective, and efficient treatments for the disorder.



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