

Viewpoint

# Enhancing Agency in Posttraumatic Stress Disorder Therapies Through Sensorimotor Technologies

Vladimir Adrien<sup>1,2,3</sup>, MD, PhD; Nicolas Bosc<sup>3</sup>, MD, PhD; Claire Peccia Galletto<sup>4</sup>, BSc; Thomas Diot<sup>5</sup>, MSc, MD; Damien Claverie<sup>6</sup>, MD, PhD; Nicco Reggente<sup>7</sup>, PhD; Marion Trousselard<sup>6,8,9</sup>, MD, PhD; Eric Bui<sup>10,11,12</sup>, MD, PhD; Thierry Baubet<sup>3,13,14</sup>, MD, PhD; Félix Schoeller<sup>7,15</sup>, PhD

<sup>1</sup>Department of Infectious and Tropical Diseases, Avicenne Hospital, AP-HP, Université Sorbonne Paris Nord, Bobigny, France

<sup>2</sup>Institute of Psychiatry and Neuroscience of Paris, Inserm UMR-S 1266, Université Paris Cité, Paris, France

<sup>3</sup>Department of Psychopathology, Avicenne Hospital, AP-HP, Université Sorbonne Paris Nord, Bobigny, France

<sup>4</sup>Unités René Diatkine, Association de Santé Mentale du 13<sup>e</sup> arrondissement, Paris, France

<sup>5</sup>Department of Adult Psychiatry, Impact, Mondor Hospital, AP-HP, Université Paris-Est Créteil, Créteil, France

<sup>6</sup>Institut de Recherche Biomédicale des Armées, Brétigny-sur-Orge, France

<sup>7</sup>Institute for Advanced Consciousness Studies, Santa Monica, CA, United States

<sup>8</sup>INSPIRE, Inserm UMR 1319, Université de Lorraine, Nancy, France

<sup>9</sup>ADES, CNRS UMR 7268, Aix-Marseille Université, Marseille, France

<sup>10</sup>Department of Psychiatry, Caen Normandy University Hospital, Normandie Université, Caen, France

<sup>11</sup>Physiopathology and Imaging of Neurological Disorders, UNICAEN, Inserm UMR-S 1237, Normandie Université, Caen, France

<sup>12</sup>Department of Psychiatry, Massachusetts General Hospital, Boston, MA, United States

<sup>13</sup>Unité Transversale de Psychogénèse et Psychopathologie, Université Sorbonne Paris Nord, Villetaneuse, France

<sup>14</sup>Centre National de Ressources et de Résilience, Lille, France

<sup>15</sup>Media Lab, Massachusetts Institute of Technology, Cambridge, MA, United States

**Corresponding Author:**

Vladimir Adrien, MD, PhD

Department of Infectious and Tropical Diseases

Avicenne Hospital, AP-HP

Université Sorbonne Paris Nord

125 rue de Stalingrad

Bobigny, 93000

France

Phone: 33 148955421

Fax: 33 148955428

Email: [vladimir.adrien@aphp.fr](mailto:vladimir.adrien@aphp.fr)

**Abstract**

Posttraumatic stress disorder (PTSD) is a significant public health concern, with only a third of patients recovering within a year of treatment. While PTSD often disrupts the sense of body ownership and sense of agency (SA), attention to the SA in trauma has been lacking. This perspective paper explores the loss of the SA in PTSD and its relevance in the development of symptoms. Trauma is viewed as a breakdown of the SA, related to a freeze response, with peritraumatic dissociation increasing the risk of PTSD. Drawing from embodied cognition, we propose an enactive perspective of PTSD, suggesting therapies that restore the SA through direct engagement with the body and environment. We discuss the potential of agency-based therapies and innovative technologies such as gesture sonification, which translates body movements into sounds to enhance the SA. Gesture sonification offers a screen-free, noninvasive approach that could complement existing trauma-focused therapies. We emphasize the need for interdisciplinary collaboration and clinical research to further explore these approaches in preventing and treating PTSD.

(*J Med Internet Res* 2024;26:e58390) doi: [10.2196/58390](https://doi.org/10.2196/58390)

**KEYWORDS**

posttraumatic stress disorder; PTSD; agency; proprioception; trauma; self-control; sensorimotor technology; enactive perspective; peritraumatic dissociation; proprioceptive reafferent fibers; gesture sonification devices

## Introduction

Posttraumatic stress disorder (PTSD) stands among the 10 major public health issues [1]. Remission is obtained for a mere one-third of patients at 1-year follow-up, while another third continues to grapple with symptoms a decade later [2]. The gold standard treatments are trauma-focused cognitive behavioral therapies (CBTs), including cognitive processing therapy and prolonged exposure [3,4]. These approaches aim at improving self-regulation [5] but lead to remission in only approximately 40% of patients [6], although criteria for treatment nonresponse are not well defined [7].

Virtual reality exposure therapy (VRET) has been used with immersive simulations of trauma-relevant environments [8,9]. However, its superiority over standard prolonged exposure remains controversial [10-12], probably due to some inherent limitations, such as the sensory conflicts virtual reality (VR) induces as well as how its visual interface decouples patients from their body and environment [13]. In this context, other sensorimotor technologies letting patients connect to their environment may be of interest due to their potential to modulate the sense of body ownership (SO) and sense of agency (SA).

Emerging data have in fact reported how the SO and SA may be impacted in PTSD [14-19]. While the loss of the SO and control has been extensively studied in the context of PTSD and is historically among the main targets of trauma-focused therapies, much less attention has been paid to the loss and restoration of the SA, as opposed to emotion regulation and cognitive control. Despite promising theoretical perspectives on this topic under the umbrella of enactive theories of mind [15,20-28], there is still a dearth of agency-based treatment and recovery options available to patients with PTSD and clinicians. In this perspective paper, we explore the SA in the context of PTSD and its importance in understanding symptoms and improving treatments. Our contribution entails providing a road map for the development of agency-based therapies in the future, along with offering an agency-based perspective on psychological trauma. Finally, we explore the potential of using screen-free innovative technologies such as gesture sonification (GS) to complement existing trauma-focused therapies that target internal regulation by directly influencing the SA, thereby paving the way for the creation of new and effective intervention tools.

## SO and SA: Definition and Brief Review

The SO refers to the perception of one's own body, feelings, thoughts, or movements, integrating somatosensory signals fundamental to distinguish between self and other, that is, to self-consciousness and control [29,30]. It depends on the interaction of afferent feedback and a top-down contribution of body representations [31,32]. The interplay between afferent internal (interoceptive and proprioceptive) and external multisensory (eg, visual and tactile) stimuli is sufficient for the SO [33], which is viewed as a psychophysiological baseline involving the brain's default mode network (DMN) [34]. This multisensory integration in the premotor cortex enables bodily self-attribution [35,36], with the prioritization of the most

relevant sensory domains [37]. Computational (Bayesian) models have been suggested to account comprehensively for this multisensory integration [38]. Conversely, the SA refers to the immediate feeling of initiating and controlling an action [39], that is, to the subjective perception of being an agent effecting changes in the external world with a sensorial reflection (ie, sensorimotor contingencies). That is, the SA refers to the feeling of being "the one who is causing some event in the external world" [40]. It depends on prior intention and prereflective perceptual monitoring of the consequences of self-generated action, achieved through basic efferent motor-control processes and sensory feedback [31,41-43]. The SA involves premotor, motor, and temporoparietal areas, intention, and action monitoring [34,44] and integrates sensory and motor signals into a coherent representation of the self-world system (ie, the sensory expectations deriving from motor output). The SA also relies on proprioception, which is broadly defined as the sense of (self-generated) movements, integrating signals arising from cutaneous, muscular, and joint receptors [45], and on causal attribution [46]. Therefore, the SA is best described as a multisensory process that integrates motor and nonmotor cues [47,48].

All in all, the SA is the sense of originating and controlling our actions, whereas the SO is the sense of being the one to act [29,39,47]. For instance, involuntary movement gives rise to the SO but not to the SA [42]. This phenomenological distinction has neural correlates [44], but recent work seems to show a more interactive model where the SA and SO are strongly related to one another [49-51]. For instance, both share a network in the left middle insula [52], and intereffector regions in the motor cortex display strong functional connectivity, suggesting that body control and action are part of a common circuit [53].

Prereflective SA and SO can be distinguished from attribution judgments [54-56], that is, the ability to attribute an action to its proper agent (self or other), which also integrates sensory information (the visual sense being determinant [57,58]). This integration processes multiple sources indirectly due to action and its congruence to the prediction of one's self-narrative and understanding [41]. These second-order reflective (ie, cognitively processed) attributions of agency and ownership involve *judgments* of agency and ownership [42,47,59] and can be distinguished from first-order prereflective (ie, motor processed) *experience* (or *feeling*) of the SA and SO, although experience can influence judgment [60]. It is worth noting that this separation between the SO and attribution of ownership [61,62], as well as the separation between the SA and attribution of agency [63], has been challenged.

The SO can be measured by experiments, such as the rubber hand illusion (RHI) [33,35,64,65], in which a lifelike left rubber hand is viewed by participants whose real left hand is hidden, and the same afferent signals (eg, tactile stroke by a paintbrush) are being delivered to both hands, generating the transfer of the SO from the real to the rubber hand (ie, the illusion that the rubber hand is the real hand). Participants are then asked to point to their left hand with the right one and point toward a position between both hidden and rubber hands, elucidating the proprioceptive drift.

To measure the SA, both direct and indirect measures have been proposed [66]. Direct measures are rating scales or self-report questionnaires [66], whereas indirect measures focus on intentional binding [60], that is, the perceived time interval between the action and its outcome in comparison to involuntary action [67]. There are ongoing debates about the reliability and validity of these measures, as no correlations were identified between both types that may assess different aspects of the SA [68]. In this context, the Sense of Agency Scale, a new psychometric scale, has been developed and validated to directly assess global SA [69]. This self-report questionnaire includes 2 factors, the sense of positive agency and the sense of negative agency (respectively the control and lack of control over the environment), and is thus interesting for assessing altered SA in psychopathology. The Sense of Agency Scale has been validated in multiple languages [70]. The SA can also be measured in the laboratory with the RHI, which has been adapted recently [71] to a “dynamic” RHI where the index finger of the rubber hand moves when the participants move their own finger, both hands being mechanically connected, and the participants transfer their SA to the rubber hand (ie, feel being the ones tapping on the table with their finger). Interestingly, the illusion stays equally strong for various combined sensory domains [72]. The SO and SA can be dissociated by varying the mode of movement (passive or active) and the position of the rubber hand (congruent or not). This strongly suggests that the SO and SA still represent distinct cognitive processes. The alteration of both SO and SA has been extensively studied across psychiatric conditions [42,57,58,73-75].

## *Psychological Trauma as a Major Loss of the SA*

Human experience constantly presents us with challenges that either meet our physical and cognitive skills or require that we develop new ones. When none of these are an option, or these options are overwhelmed, because of an unexpected event that goes largely beyond one’s representations and ability to adapt, threatens survival and physical or cognitive integrity of oneself or others [76], and induces a response of intense fear, helplessness, or horror, this event is called a *traumatic event* (TE; derived from  $\tau\rho\alpha\ \mu\alpha$ , the Greek word for wound, hurt, or injury). In the Bayesian approach of the mind as a hierarchical predictive model of its reality, the TE does not relate to any predictive models available to make sense of the external world, as no empirical priors exist to account for the incoming sensory signals or the most likely motor response. Significantly, in severe cases of psychological trauma, the lack of a predictive model results in the loss of both vision and hearing [77]. From a physiological perspective, both central and peripheral regulatory systems are ineffective in carrying out their retrocontrol functions. The stress response system, which involves the activation of the amygdala at the central level and the corticotrope and noradrenergic hormonal systems at the peripheral level, is hyperactivated in contrast to the underactivation of brain structures responsible for typical retrocontrol [78,79]. This includes the prefrontal cortex (PFC) and the hippocampus [80-83]. Regarding circuit regulation, this aligns with an overactive salience network (SN; eg, amygdala

with insula and the anterior cingulate cortex [ACC]), that is, with increased threat detection and fear learning [84], in contrast to weakly connected DMN and central executive network [85,86]. Along with the disruptions of the hypothalamic-pituitary-adrenal gland axis, the entire system is unable to effectively regulate the adaptive stress response.

Understanding the TE as a potential sensorimotor failure leads to a novel perspective of psychological trauma as *a major breakdown of the SA*. A lot of attention has been directed toward the SO and self-control, which depend largely on interoceptive signals (eg, “my body is unable to mitigate its own state of stress when being mugged”), whereas the clinical focus on the SA, directed toward exteroceptive signals, is insufficient. In the context of psychological trauma, the felt sense of helplessness (eg, “my body is unable to defend against an aggressor”), peritraumatic distress [87]; or tonic immobility, that is, the freeze response [88], translate into the failure of the SA. Hence, the breakdown of the SA automatically triggers a hardwired sensorimotor response, sometimes referred to as the defense cascade [88]. The alteration of both perception and action during the TE leads to dissociation [89], that is, disconnecting from incoming sensory information, generating a psychological distance from the traumatic experience, and allowing the patient to “tolerate” the intolerable [90]. Peritraumatic dissociation—an array of reactions to the TE that includes depersonalization, derealization, and emotional numbness [91]—has been thoroughly investigated as being the result of the absence of adequate sensory and motor representations (ie, “I do not understand what is happening to me and there is nothing I can do”). In derealization, the sense of the world is lost, whereas in depersonalization, the sense of self is lost. Emotional numbness corresponds to the absence of defensive emotions such as fear or anxiety. In certain cases, individuals may enter a state of sideration or extreme surprise, unable to move or plan any action, essentially losing their SA. Peritraumatic dissociation can be seen as a protective response to the intense emotional distress during the TE when there is no possibility of escape or avoidance and no sensory or motor representations of the event [23,92-94]. It provides a sense of safety and physical and psychic analgesia, reducing engagement with the TE [89,95]. Nevertheless and importantly, experiencing severe peritraumatic dissociation raises the risk of developing PTSD [96-99], interfering with trauma memory processing and coherence [89], thus, in this case, being deemed maladaptive, impeding the processing of the TE.

There is indeed a significant memory-related aspect of PTSD [100]: the emotions or sensory memories attached to the TE are either not integrated as long-term declarative memory, resulting in denial or the inability of verbalization of the TE, or are integrated as semantic instead of episodic memory [101,102], that is, as a factual general knowledge that does not seem to belong to the individual [103,104]. In other terms, the traumatic memory will be related to noetic instead of auto-noetic consciousness [105,106]. This correlates with reduced activity or dysfunction in the hippocampal structures [107,108]. When the traumatic memory remains unprocessed, it fails to integrate with the individual’s conscious perception of reality. Over time, the failure to integrate the traumatic memory leads to a default

in contextual processing [84,85,109,110], resulting in trauma-related cognitions, such as guilt and shame, which in turn induce a wide array of comorbid complications, such as depression [111], anxiety [112], and obsessive-compulsive disorder [113].

Posttraumatic dissociative states can also emerge later on as a protective reaction against the abnormal emotional associations linked to the traumatic memory [114]. Dissociative PTSD (D-PTSD) has been recognized in the *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5)* [115], and is supported by a neurophysiological basis that distinguishes it from conventional PTSD. Emotional overmodulation (ie, hypoarousal) in D-PTSD is thought to be linked to an underactivation of the amygdala and an overactivation of the PFC [114], in contrast to the standard PTSD model. The Clinician-Administered PTSD Scale for *DSM-5* [116] allows for differentiation between these 2 types of PTSD, and approximately 30% of individuals with PTSD may have the dissociative subtype [15,114,117]. It has already been demonstrated that patients with D-PTSD exhibit a higher proprioceptive drift in the RHI [15], suggesting that individuals with D-PTSD integrate the illusion more significantly due to their diminished SO. Further research is needed to determine if the SA is altered differently between these clinical subtypes. The latest version of the *International Classification of Diseases* [118-120] has recently introduced a subtype known as complex PTSD (C-PTSD), which is characterized by the symptoms of PTSD and 4 additional groups of symptoms: dissociation; difficulties in regulating emotions; a negative self-concept involving feelings of worthlessness, defeat, shame, and guilt; and challenges in social cognition, such as maintaining relationships and feeling emotionally close to others. This distinction is strongly supported by research [119,121,122].

C-PTSD affects approximately 40% of individuals with PTSD [123]. As for both D-PTSD and C-PTSD subtypes, there are currently no established guidelines for best practices.

Trauma-focused CBT may restore the functionality of the brain structures (PFC, ACC, and hippocampus) that are involved in executive retrocontrol, which is initially ineffective during the TE [124]. Notably, trauma-focused CBT is safe [125] and has been found to be as effective in treating D-PTSD [126,127], despite its reversed neurophysiological model, and C-PTSD [123] than conventional PTSD. By concentrating on the traumatic memory, the goal of this therapy is to alleviate the emotional dysregulation experienced with PTSD symptoms. Alternatively, the enactive perspective asserts that the traumatized brain is engaged, interconnected with the body and environment, and dynamically integrated [128].

## ***PTSD Symptoms Might Be Understood as an Attempt to Restore the SA***

### **Overview**

When considering psychological trauma from an enactive perspective and acknowledging that perception and action are intrinsically linked, the mechanisms described in the previous section and their accompanying symptoms can also be understood as an attempt to control oneself, the environment, and the causes of the TE that pervade both perception and action, whether physically or virtually. Thus, understanding PTSD symptoms as an adaptive response to psychological trauma can inform future treatment development, creating a more holistic approach to PTSD treatment that focuses on empowering patients to regain an SA and control over their environment. In this section, we briefly review PTSD symptoms and how they relate to the SA (Table 1).



**Table 1.** Posttraumatic stress disorder (PTSD) symptoms, their relation to the sense of agency (SA), and how intervening on the SA may target these symptoms.

|                                 | How does the symptom relate to the SA?  | How agency-based therapies can help?   |
|---------------------------------|---|--|
| Intrusion syndrome              | <ul style="list-style-type: none"> <li>Thoughts: attempt to get another shot at unfolding an optimal motor response to control the TE<sup>a</sup>.</li> <li>Repetitive behaviors: attempt to attribute to our own action the exposition or occurrence of the TE or the destruction of oneself during the TE.</li> </ul>   | Exposure therapy aims at self-control during intrusion symptoms, rather than the SA. Agency-based therapy may augment gradual exposure by letting the patient control exposure level and would aim at increasing environmental control and replacing repetitive behaviors by enhancing the SA. |
| Avoidance behaviors             | <ul style="list-style-type: none"> <li>Attempt to regain control over the environment and reminders of the TE or protect oneself from further harm.</li> </ul>  | Using gradual exposure therapy to triggers with body control of these triggers.  |
| Hyperarousal and hypervigilance | <ul style="list-style-type: none"> <li>Minimizes the expected loss of the SA (as a failure to avoid or control the TE, the patient anticipates it anytime).</li> </ul>  | The patient controls sensory triggers themselves with the help of the therapist to regain adapted and coherent salience.   |
| Trauma cognitions               | <ul style="list-style-type: none"> <li>Guilt: attempt to integrate the causes of the TE within a wider model of self, other, and reality in which oneself had some SA over what caused the TE to occur.</li> <li>Anxiety: signal anxiety, anticipate the expected loss of the SA, and work as a protection.</li> <li>Depression: loss of omnipotence or frustration of not having SA over the environment.</li> </ul> | By controlling the environmental sensorial triggers, the patient can get pleasure in return, reducing anxiety and frustration and regaining some SA over the environment.  |
| Dissociation                    | <ul style="list-style-type: none"> <li>Loss of the SA.</li> <li>In addition, adaptive response against the passive loss of the SA felt during the TE or PTSD symptoms.</li> </ul>   | Increasing the sensorial signals produced by one's action in the external environment reduces the disconnection felt between oneself and reality.  |

<sup>a</sup>TE: traumatic event.

## Intrusion Symptoms

Intrusive thoughts and memories as well as vivid re-experiencing, also called flashbacks, and traumatic nightmares are common symptoms in the aftermath of a TE [129,130]. These intrusive symptoms are distressing and make it difficult for patients to function in their daily life. Some patients with PTSD may have a tendency to replay the TE mentally in an obsessive-compulsive or addictive fashion, supposedly in an attempt to make sense of it and understand what had happened [131]. Intrusion symptoms can be regarded as a facet of dissociation [104]. Still, the recent findings on the activation of brain structures and neural circuits pointed out in the previous part, which would make PTSD and D-PTSD different entities, oppose this interpretation [114,132]. Intrusion symptoms have recently been analyzed from a Bayesian perspective, where the TE perceptual hypothesis gains a very high prior due to its life-threatening significance and is reselected independently of the actual sensory input [133]. From an enactive perspective, this can be understood as a way for the individual to try to regain some control over the TE to make it less distressing. Indeed, instructions to freely express intrusive thoughts led to a decrease in intrusive sexual assault thoughts over time, while suppressing them led to an automatic rebound in intrusive thoughts over time [134].

In addition to intrusive cognitions, repetitive behaviors, such as re-exposing oneself to potentially traumatic situations, are often seen in individuals with PTSD and can be a way for them to try to regain their SA. This can be observed in patients with

C-PTSD and premorbid personality disorders, who may engage in risky or self-destructive behaviors [135]. Perhaps one of the most striking examples of repetition syndrome would be patterns of hypersexuality in children who have been victims of sexual abuse [136]. Patients are at risk of repeated harm, either self-inflicted or at the hands of others, adopting self-destructive behaviors [136] or repeated exposure to violence (suicide attempts, self-mutilation, military enlistment, development of substance use disorders, risky sexual behavior, and inability to give consent for sexual intercourse leading to exposure to rape situations), which can be seen as a tentative to attribute to our own action the destruction of oneself experienced during the original TE [121]. The climax of this viewpoint is found in the classical example of survivors becoming executioners (children who experienced abuse, child soldiers, etc) after a phase of repetitive behaviors such as engaging in plays or reenactments, often seen in childhood traumas [137]. Freud [138] famously discussed the idea of individuals using objects or activities to regain a sense of control over their environment. He gave the example of a baby playing with a toy that represents his mother to feel like he has control over the situation and soothe his distress. This idea was later developed by Winnicott [139] into the concept of transitional objects, used by children to cope with the loss of the subjective omnipotence over their environment, deriving from the caretaker's attention to the child's need, a frustration that can be related to the one experienced during a TE. Separation (ie, autonomy) from the parents accompanies the rise of the SA in childhood, as the sum of the attempts to compensate for the loss results in the gained ability to be alone. Intrusive cognitions and repetitive behaviors

are thus common symptoms observed in individuals with PTSD, which can be seen as attempts to regain an SA and cope with the psychological trauma. However, these symptoms can also lead to repeated harm or victimization, making it crucial for therapists to develop treatment strategies that focus on restoring the SA while addressing the underlying causes of these symptoms.

### Avoidance Behaviors

The second category of PTSD symptoms is avoidance behaviors [140], that is, the active avoidance of thoughts, feelings, or external reminders of the TE. This can include avoiding certain places, people, or activities that may trigger memories of the event [141]. As patients have lost their SA over what triggers their flashbacks, the best way to control their occurrence is simply to avoid the stimulation. Here again, this might be understood as a displacement of the cause of the TE, where patients assume they can take responsibility for generating these sensory signals that largely surpassed their perception and action capabilities. One possible explanation for avoidance symptoms in PTSD is, therefore, that they may be an attempt by patients to regain their SA over their environment. For example, patients with PTSD due to a car accident may avoid driving, as driving may trigger memories of the TE [142].

An alternative explanation for avoidance symptoms in PTSD is that they serve as a means of self-protection, aiming to minimize the risk and vulnerability to risk. Social cognition impairment is commonly observed in PTSD [143]. By avoiding social situations, patients can shield themselves from re-experiencing the TE and encountering additional distress. Another instance is postdisaster PTSD, which is more likely to occur if the disaster originates from human actions as opposed to natural causes [144,145]. This underscores the greater psychological impact of interpersonal trauma, as it is perceived as potentially controllable, thus amplifying the distress. Patients may instinctively seek to regain the SA by avoiding situations associated with interpersonal trauma when addressing PTSD symptoms.

It should be noted that substance use is often seen as a form of avoidance in individuals with PTSD who may use drugs or alcohol as a way to numb or suppress their traumatic memories [146] or to escape from the difficult emotions that can accompany these memories. For example, self-reported PTSD is associated with increased use of 3,4-Methylenedioxymethamphetamine (MDMA) in adolescents with substance use disorders [147], and substance abuse is widespread in victims of childhood sexual abuse [148]. In addition to numbing their emotions, substance use can also be a way for individuals with PTSD to avoid trauma reminders (people, places, or situations).

### Negative Alterations in Cognition and Mood and Maladaptive Schemas

Negative cognitions are thoughts and beliefs about oneself, others, and the world that are negatively distorted and not based on reality. They include beliefs that one is to blame for the TE, that the world is dangerous and unpredictable, and that one is not safe. They also include decreased interest in activities,

negative emotions, and the feeling of being isolated. They may be a way of coping with the loss of the SA. Patients with PTSD often believe they are to blame (ie, the feeling of guilt) for the TE, perhaps as this belief provides them with a (virtual) sense of control over the situation. By experiencing guilt and self-blaming, they make sense of the event and feel like they have some SA over it—"I am responsible for exposing myself to the environment that generated the TE" [149].

In addition, negative cognitions in PTSD relate to the prediction of an *expected* loss of the SA. A TE can cause a person to feel vulnerable and at risk: negative cognitions may be a way of anticipating and preparing for future loss of control. For example, patients with PTSD may believe that the world is dangerous and unpredictable, as this belief helps them to be prepared for potential threats. This culminates with the concept of signal anxiety developed by Freud [150], one of the main defense mechanisms. By expecting the worst, patients are able to feel like they have some control over their environment and can protect themselves from further harm. Klein [151] related trauma negative cognitions to the "depressive position" in the early phase of existence. This preverbal infantile attitude comes with a loss of subjective omnipotence (ie, control) over external objects that coincides with the emergence of emotional valence in infancy: the child becomes aware that objects not only gratify ("good object") but can also frustrate ("bad object"), generating feelings of guilt and grief and a desire for reparation. This example of "proto-traumas" or "micro-traumas" experienced repeatedly during early childhood lightens what happens when a real TE occurs later in life, altering the sense of control and leading to greater PTSD susceptibility [99,152,153]. These negative thoughts and beliefs may be a way of coping with the distress and vulnerability caused by the TE, but in a vicious cycle, they can also have negative effects on daily life and overall well-being related to an expected loss of the SA, if nothing is done to remedy it.

### Alterations in Arousal and Reactivity: Disturbed Attentional Patterns

Patients with PTSD develop a heightened state of awareness, alertness, and physiological arousal to regain a sense of control and SA [154]. This is referred to as hypervigilance [114], characterized by emotional undermodulation, resulting in an increased threat detection and fear learning, and a state of alertness and readiness to respond to these threats [155]. This also includes irritability, difficulty concentrating, sleeping disorders, and an overall sense of feeling on edge. On a sensorimotor level, hypervigilance may manifest as an increased sensitivity to sensory stimuli, such as changes in light or sound, as well as increased physiological arousal, such as increased muscle tension or an elevated heart rate, which is a risk factor for developing PTSD when it immediately follows the TE [156,157]. PTSD is in return a risk factor and shares genetic risk for cardiovascular diseases [158]. By being constantly on guard and ready to respond to potential threats, patients may feel they are able to anticipate and prevent dangerous situations from occurring. This restores the SA that was lost as a result of the TE. Hypervigilance, as an attentional bias toward potential threats, can lead to anxiety and further hypervigilance [155,159]. This can cause the individual to misinterpret ambiguous cues

as threats and exaggerate minor threats, as shown by the overactive SN, whose function is to recognize and prioritize stimuli while regulating emotional reactivity [160]. These changes in attention and threat perception can also manifest in the individual's eye movements [161], leading to an inability to disengage from potential threats. This is referred to in the literature as "oculomotor reflexes" [162]. From a Bayesian inference perspective, dysfunctional SN amounts to aberrant precision control, where precision denotes the confidence placed in prediction errors (mirroring the reliability of the stimulation that causes them) within the hierarchy of information processing, that is, a high precision will favor bottom-up ascending prediction errors, while a low precision will bias perception toward top-down prior beliefs. Interestingly, the SN implies the ACC and insula, which are strongly related to interoception [163]. The dysregulation of bodily signals in psychiatric illnesses may, therefore, offer an important way forward in terms of phenotyping [164,165].

### Dissociative Symptoms

After Winnicott [139], Anzieu [166] developed the concept of "skin-ego," that is, the presence of a symbolic skin allowing for the creation of psychic and bodily boundaries. In PTSD, this symbolic frontier is blurred or constricted and no longer filters or organizes sensory and perceptual signals as easily. It becomes porous and loses its function as a protective structure between interoceptive cues and cues originating from the external world. This sudden immense vulnerability exacerbates the loss of self-integrity and SO and forces the maintenance of a state of psychic survival with various defensive symptoms involving both temporality and space: agitation, withdrawal, and dissociative symptoms.

The view of peritraumatic dissociation as a protective response can be related to the anthropological interpretation of dissociative experiences with their positive affective valence, with the DMN mainly activated in situations such as daydreaming, hypnotic responses, fatigue, anxiety, drug intoxication, and boredom [167]. In this case, dissociation has a social and discursive meaning rather than being seen as a mechanism. It can thus function adaptively, which depends fundamentally on context [94,168].

By contrast, dissociation interferes with the integration of sensory information and the creation of a coherent sense of self. If dissociative symptoms of PTSD are the reminiscences of peritraumatic dissociation and protect patients against the distress provoked by intrusion symptoms or hyperarousal, they are nevertheless severely debilitating. Patients often complain of the distress related to their dissociative states, which can be transitory or permanent and are associated with high disability. Dissociation can indeed be interpreted literally as the loss of the SA: "if I see myself from an external point of view, thus my body's actions do not belong to myself" and "if the external world is not real anymore, then I can no longer be an agent in the real world."

Dissociation can thus at the same time be a loss of the SA and a protective phenomenon against it. There is an ongoing debate on this matter, with the concept of protective dissociation culminating in at-risk professions such as the military or fire

service: workers experience deliberate dissociative states [169,170] to gain automatic action and execute tasks more efficiently without being emotionally overwhelmed. Further research is required to thoroughly investigate and clarify this phenomenon. One potential avenue is to distinguish between the *passive* loss of the SA resulting from external TE and the symptoms actively produced (deliberately or not) by patients as a means to combat passivity ("if it is due to my action or my mind that I lose SA, thus the weight of external events in the loss of SA is reduced"). This includes dissociation or dissociative behaviors (eg, drug use).

## Sensorimotor Technologies for Agency-Based Therapies

The enactive, agency-based perspective on PTSD suggests that the disruption of the SA results from the TE, overwhelming the patient's ability to adapt and make sense of sensorimotor signals, leading to a breakdown in the hierarchical predictive model of reality. This disrupted SA may contribute to or be further exacerbated by peritraumatic dissociation, explaining why its intensity is a predictive factor of PTSD onset [96-99].

Currently, there is a lack of agency-based treatment options for PTSD. New treatment options to manage nonresponse to PTSD conventional treatments [7] include the promising use of ketamine [171] or psychedelic-assisted psychotherapy [172], whose dissociative effect is now reconsidered as a phenomenological therapeutic tool, as these changes of the self-experience have a subjective meaning and are transitory and necessary for coping. These drugs could help improve prefrontal function and contextual processing, modifying beliefs, refining predictions and thus the SA [173-178]. Ketamine could also induce brain-derived neurotrophic factor increase in the hippocampus [179]. Conversely, endocannabinoids modulators or drugs that reduce the glutamate response (such as D-cycloserine, a partial N-methyl-D-aspartate [NMDA] receptor agonist) could not only treat dissociative PTSD symptoms [180,181] but also enhance extinction training by preventing the hyperglutamatergic state of the stress response responsible for abnormal fear conditioning [182,183] as well as the defaults in memory processing [181]. Indeed, NMDA receptors are highly concentrated in the hippocampus and implied in long-term potentiation, a mechanism for encoding long-term (eg, episodic) memory. Neuromodulatory treatments [7] such as transcranial magnetic stimulation [184-186], transcranial direct current stimulation [187,188], deep brain stimulation [189], and vagus nerve stimulation [190,191] could increase body awareness, SO, and SA. Nevertheless, less invasive or expensive treatments, such as sensorimotor therapies [192-194], may have the potential to enhance and restore the SO and, more importantly, SA during PTSD recovery. A mounting body of evidence [165] suggests that bodily signals play an essential role in driving precision control, hinting toward the relevance of reliable body-based interventions for mental health disorders depending on the patient's life history, conditions, and symptoms. The controlled generation of artificial sensations could, therefore, lead to novel options for the diagnosis, monitoring, intervention, and treatment of disorders of emotional



and interoceptive inference. One such potential solution is “*affordance training*” with a narrative therapy map [195], which involves helping the individual to focus on action possibilities rather than action impossibilities. This can involve training patients to engage in goal-directed behavior and to seek out opportunities for action, rather than focusing solely on potential threats.

Sensorimotor technologies, such as VR, have been used for exposure therapy in immersive simulations of trauma-relevant environments [8,9]. The aim is to allow a precise control of stimulus [196]. VRET can discriminate between patients with PTSD and patients without PTSD on a measure of psychophysiological arousal such as skin conductance reactivity [197,198] as well as between patients with low symptoms of subthreshold PTSD and patients with high symptoms of subthreshold PTSD through heart rate [199].

However, various meta-analyses did not find any difference in clinical efficacy between conventional VRET and other psychotherapies [10-12], although some found moderate effects [200]. However, specific VR-graded exposure therapy seems to be beneficial [11]. All in all, VRET may be beneficial only for specific patient profiles (eg, those who are younger, with greater hyperarousal symptoms, with comorbid depression or suicidal risk, with no antidepressant medication, or who cannot engage in imaginal imagery) compared to prolonged exposure therapy [201-203]. One explanation of the limitation of VRET is that patients may become diverted by the technology, expressing doubts about its authenticity (“this isn’t real”) and using this divergence or the lack of personalization of virtual environments as a means to evade forming an emotional connection with their distressing memories [196], especially because sensorial integration prioritizes sight, as shown by the RHI [65]. Indeed, VR deprives patients of their gaze and hands due to the screen and interface, creating a disconnection between the patient’s body and its environment, a restriction of virtual objects as actionable targets, contrary to tangible real-world objects [204], compromising the transfer of visuomotor skills from virtual to real settings [205]. In addition, the disembodied effect of VR can create interoceptive and visuo-proprioceptive issues that make the experience less immersive [206,207], besides causing discomfort, headaches, nausea, or instability [205,208]. Another issue is the lack of facial or trauma-focused interaction with the therapist, which can be problematic for the therapeutic alliance, limiting a shared experience, impeding regaining a sense of social safety [209-211] as well as the possibility of group therapy, a useful method in PTSD [212]. In this context, other sensorimotor technologies letting patients connect to their environment may be of interest as an alternative or adjunctive intervention. They could provide individuals with an SA over their environment.

## The Example of GS Technologies to Treat PTSD

### Overview

GS is a technique that involves using natural body gestures to control and generate sound. It uses sensors to detect the

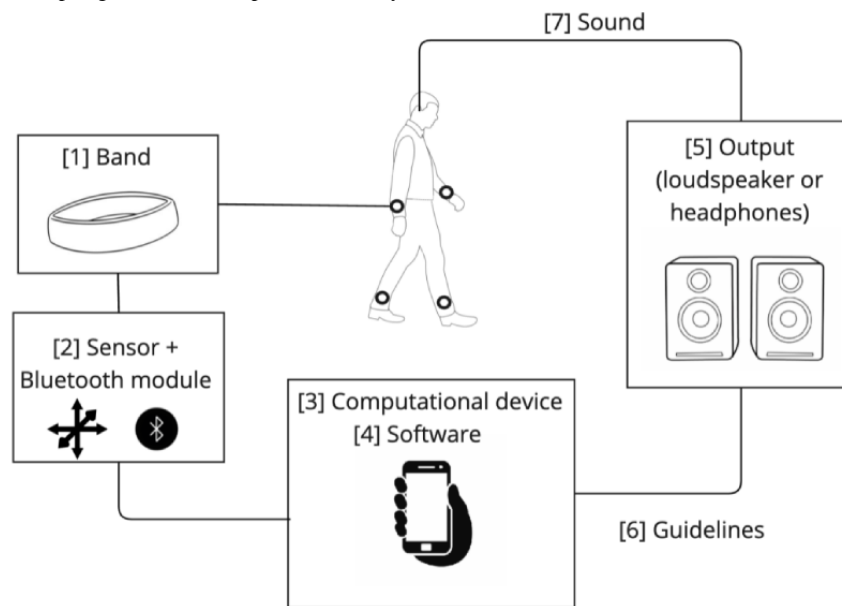
movement of an individual’s body and then translates this movement into sound (Figure 1).

GS allows overcoming VR’s limitations, as it is a screen-free alternative where only the auditory sense is augmented. It enables participant-body-environment coupling, the maintenance of contact with the therapist, and a more individualized virtual environment. It is applicable in case of language barrier, is more acceptable and user-friendly, and provides clinicians with ample opportunities for exploration. However, despite its potential benefits, some challenges might be encountered, such as acceptability. Indeed, patients may refuse to use GS; feel inhibited or startled while using it; and be exposed to various risks, such as allergies to sensors or, in case of improper use, hearing loss due to excessive volume or physical injury from uncontrolled movement. In addition, it is important to exercise caution both in the moment and in selecting sounds according to the way GS is used. For instance, some sounds that might be used for providing a sense of security in some patients could trigger intrusive symptoms in others due to specific traumatic experiences (eg, water sounds for a patient who experienced drowning).

While GS has found applications in various health contexts, such as physical rehabilitation [213] and music therapy [214], its potential in psychiatric illnesses has largely remained untapped. By combining GS with sensorimotor exercises, patients with PTSD could use their bodily movements to generate sounds and manipulate and control virtual objects and environments, creating a more immersive and engaging experience that leaves room for interaction with the therapist. GS could, therefore, emphasize the body and the sensations of movements that are often overlooked in first-line therapies such as prolonged exposure. By directing attention to the body and the physical sensations experienced, GS could assist patients in connecting with their present moment experience and developing a greater SA [193,215]. Indeed, multisensory retroaction can reinforce patients’ sense of control over their symptoms [213]. Importantly, GS can be combined with guided imagery (GI) to provide a personalized experience for patients, allowing them to manipulate, through sensorimotor exercises, the traumatic memories and facilitate their integration. GI is a behavioral mind-body intervention using appropriate scripts and imagination to manipulate representations and enable positive affective and body responses [216,217]. GI uses sensory integration to enhance affective and cognitive retrocontrol over hyperarousal through muscular relaxation and positive mental images. GI has been shown to improve depression, anxiety, and stress [218] and change the meaning of pain [219] and is also widely used in prolonged exposure as well as a self-management intervention to alleviate PTSD symptoms [220], especially hyperarousal. Nevertheless, individuals are not equal in their ability to engage in GI, and its efficacy is correlated with absorption abilities [221] and stays controversial [222]. Our team has experimented with GS and found that the technology could potentially help augment such abilities and thus mind-body interventions. We identified 5 potential applications of the system following the common course of existing therapies.



**Figure 1.** System diagram of gesture sonification illustrating (1) the integration of a wearable band (2) with sensors and a Bluetooth module, (3) which communicates with a computational device (4) running specific signal processing software. The device (5) processes the data through loudspeakers or headphones (6) following the therapist guidelines (7) to provide auditory feedback.



### PTSD Primary Prevention

Functional alterations in brain circuitry identified in patients with PTSD could already be present before the TE and constitute risk factors for developing PTSD [83]. Occasionally, they may exist before PTSD and worsen after the onset of PTSD. Indeed, PTSD has been found to be highly heritable due to epigenetic factors [83,223-226], which means that prior trauma and cumulative life adversity may induce alterations in brain circuitry responsible for vulnerability to PTSD [99,152,153].

Preventing PTSD, for instance, among at-risk populations such as women, refugees, and military and rescue workers [227] involves more than just averting TEs. It means identifying cognitive impairments predisposing for PTSD and enhancing or correcting these impairments. This includes improving cortical and hippocampus retrocontrol abilities, activating the central executive network or DMN, and regulating the SN to maintain threat detection at an optimal level. Ultimately, this entails bolstering the SA before the TE occurs, potentially averting SA failure, peritraumatic dissociation, and thus PTSD later on. GS could be used to assess vulnerability and prepare military and rescue workers before field operations.

In addition, GS could serve as an SA enhancer in the immediate and postimmediate phases following the TE. During the immediate phase, where defusing by talking [228] aims at reintegrating individuals in the present moment with the presence of the other, GS could help mitigate peritraumatic dissociation by amplifying environmental sounds and facilitating reorientation. In the postimmediate phase, during early psychological interventions or debriefing, which may not prevent the onset of PTSD but may improve subsequent adherence to necessary care [229-235], GS could help reduce stress activation by providing soothing sensorimotor environments.

### Reducing Hyperarousal and Negative Alteration of Mood: Securitization

GS could potentially help patients with PTSD to reduce their hyperarousal symptoms. Patients may indeed engage their senses to focus on the present moment. Using personalized sound environments considered secure by patients (eg, water, beach, and fireplace) can help reduce physiological arousal (eg, elevated heart rate or increased muscle tension). Furthermore, by letting patients control with their body movements the volume of the sounds produced by the device, it may help them acquire the ability to filter the external sensory signals whose input is distorted in PTSD with hypervigilance.

By strengthening proprioception and sensory modality with movement, patients can learn to tame the external world, the one that has betrayed or shattered. GS would thus help construct coherence and new congruences in multisensory processing: sorting, filtering, or hierarchization. Enhancing the patient's self with an auditory signal, a vibrational charge to movement from the environment, offers the patient the opportunity to reverse defensive modes of withdrawal: it awakens curiosity and momentarily diverts anxious rumination about the external world. The environment becomes a facilitator of action, contributing to the reshaping of mental images and the reinforcement of motor planning. The patient actively participates, seeking to recombine the missing pattern in psychological trauma, creating sensory coherence and ideas through action. This synergistic process involving the postural system and the psychomotor feedback allowed by GS nourishes past sensorimotor experiences while updating new ones. Patients are placed in a position to synchronize with the world and derive pleasure from this tuning.

### Reducing Dissociation: Reassociation of Patients With Their Body

Importantly, GS could help patients with PTSD to reduce dissociation symptoms. Sensorimotor therapy facilitates the

reconstruction of the body schema to align with functional reality. Patients are guided to inhabit their body and reshape their representations within a somatic reality. Through the pursuit of body wholeness and conscious work on muscle tone, the therapist contributes to this aim. Dance, slow movement in dynamic relaxation, and postural work (eg, Qi gong and yoga), through the antagonistic interplay of tension and relaxation, create a dynamic musculo-psychological synergy. Tonus is initially a function of dialogue with the world. Among the wide variety of sensorimotor or body-oriented therapies, GS could be combined with dance movement therapy (DMT) for instance. DMT has demonstrated its effectiveness in treating PTSD [236,237]. It calls for the awakening of supportive responses: a confident relationship with support generates a repelling force. Attention is focused on coordination and responses to orientation, alignment, and balance in rhythm. DMT is a therapeutic mediation that allows for the revisiting of early tuning connections; engages in the quality of gaze, joint attention, and gesturing; and addresses support, verticalization, and initial connections. Combined with GS, patients may be able to control with their body various elements (eg, rhythm, timbre, melody, harmony, and dynamics) of the music to which they are dancing and position themselves in a realm beneath language: the reality of music interplaying with movement that spatializes and shifts. This dual modality is a prelude to the transformation of psychological trauma through somato-psychological adjustments, when the body becomes “the thought-thinking apparatus” [238]. In essence, GS combined with sensorimotor therapies would work toward the generation of new body images, reinforcing tonic regulation, self-esteem, and primary narcissism and finally restoring not only proprioceptive feelings enabling patients to reintegrate their body, as well as a sense of contact to the ground (ie, reducing depersonalization), but also the SA over incoming sensory signals from external objects (ie, reducing derealization).

### **Reducing Intrusion Symptoms and Avoidance: Exposure to the TE**

GS involving sounds related to triggers of intrusive symptoms or to the TE itself may help patients with PTSD to engage with their environment in a more active, meaningful way than mere passive exposure, controlling intrusive symptoms and overcoming avoidance behaviors. Gradually, they would expose themselves to triggers that they are avoiding, gaining progressively the ability to control them. GS may indeed let the patient control the level of gradual exposure to TE triggers by themselves. As in prolonged exposure, the uncontrollability of intrusion symptoms or triggers would then be reduced by the gained ability to control the irruption, intensity, and end of

triggers or intrusion symptoms through body-environment interactions.

### **Social and Cognitive Rehabilitation**

Social cognition is disturbed in PTSD [143], especially social perception, affective theory of mind, affective empathy, and social interactions. Of note, social cognition is more altered if the TE is interpersonal (ie, originating from human actions) than not interpersonal [239]. Patients with PTSD exhibit an overactive SN, which has a crucial function in affective empathy, which in turn also relates to a higher sensitivity to stress [240] or the negative social impact [241]. The potential higher affective empathy may probably explain the increasing attention on social rehabilitation in PTSD treatment, such as communication training, group therapies, or community programs [242]. Indeed, social rehabilitation favors the sense of belonging, just as collective commemorations of disruptive events are perceived useful by victims and help rebuild social links [243].

Sensorimotor group therapies have recently been found to be effective in treating C-PTSD [244,245]. Just as GS would help restore localization and a sense of contact with the ground and external objects by increasing the sensorimotor afferences, it could also restore the sense of contact with other individuals and help in the process of mourning. The involvement of the body and the sensorimotor pleasures that reconstruct and reshape body schemas enhance bodily sensations of gathering or synchronization. This, in turn, boosts the effective connectivity of the mirror neuron system, which is crucial for social cognition [246].

### **Conclusions**

In this paper, we offer a perspective of PTSD in the light of the essential role of the SA in functional behavior. Recognizing the TE as a sensorimotor failure, we view psychotrauma as a profound breakdown of the SA. We offer an enactive perspective of PTSD, where symptoms represent efforts to uncover and restore the SA in response to the TE. We advocate for PTSD therapy to develop interventions fostering direct engagement with one's body and environment, gradually rebuilding the SA. We suggest that agency-based therapies could mitigate PTSD risk and enhance treatment effectiveness. GS, alongside active somatic or movement-based approaches such as sensorimotor therapy, DMT, and somatic experiencing, directs attention to the body and sensory experiences, fostering present moment connection and bolstering the SA. Overall, this paper highlights the significance of the SA in psychotrauma and PTSD, offering insights to enhance treatments and advocating for further research in this critical area.

### **Conflicts of Interest**

In the past years, FS cofounded and received compensation from BeSound SAS and Nested Minds LLC. In the past months, VA also received compensation from BeSound SAS.

### **References**

1. Karam EG, Friedman MJ, Hill ED, Kessler RC, McLaughlin KA, Petukhova M, et al. Cumulative traumas and risk thresholds: 12-month PTSD in the World Mental Health (WMH) surveys. *Depress Anxiety*. Feb 2014;31(2):130-142. [FREE Full text] [doi: [10.1002/da.22169](https://doi.org/10.1002/da.22169)] [Medline: [23983056](https://pubmed.ncbi.nlm.nih.gov/23983056/)]
2. Kessler RC, Sonnega A, Bromet E, Hughes M, Nelson CB. Posttraumatic stress disorder in the National Comorbidity Survey. *Arch Gen Psychiatry*. Dec 1995;52(12):1048-1060. [doi: [10.1001/archpsyc.1995.03950240066012](https://doi.org/10.1001/archpsyc.1995.03950240066012)] [Medline: [7492257](https://pubmed.ncbi.nlm.nih.gov/7492257/)]
3. Charney ME, Hellberg SN, Bui E, Simon NM. Evidenced-based treatment of posttraumatic stress disorder: an updated review of validated psychotherapeutic and pharmacological approaches. *Harv Rev Psychiatry*. 2018;26(3):99-115. [doi: [10.1097/HRP.000000000000186](https://doi.org/10.1097/HRP.000000000000186)] [Medline: [29734225](https://pubmed.ncbi.nlm.nih.gov/29734225/)]
4. Schnurr PP, Chard KM, Ruzek JI, Chow BK, Resick PA, Foa EB, et al. Comparison of prolonged exposure vs cognitive processing therapy for treatment of posttraumatic stress disorder among US veterans: a randomized clinical trial. *JAMA Netw Open*. Jan 04, 2022;5(1):e2136921. [FREE Full text] [doi: [10.1001/jamanetworkopen.2021.36921](https://doi.org/10.1001/jamanetworkopen.2021.36921)] [Medline: [35044471](https://pubmed.ncbi.nlm.nih.gov/35044471/)]
5. Lee AH, Brown E. Examining the effectiveness of trauma-focused cognitive behavioral therapy on children and adolescents' executive function. *Child Abuse Negl*. Apr 2022;126:105516. [doi: [10.1016/j.chiabu.2022.105516](https://doi.org/10.1016/j.chiabu.2022.105516)] [Medline: [35093801](https://pubmed.ncbi.nlm.nih.gov/35093801/)]
6. Steenkamp MM, Litz BT, Hoge CW, Marmar CR. Psychotherapy for military-related PTSD: a review of randomized clinical trials. *JAMA*. Aug 04, 2015;314(5):489-500. [doi: [10.1001/jama.2015.8370](https://doi.org/10.1001/jama.2015.8370)] [Medline: [26241600](https://pubmed.ncbi.nlm.nih.gov/26241600/)]
7. Fonzo GA, Federchenco V, Lara A. Predicting and managing treatment non-response in posttraumatic stress disorder. *Curr Treat Options Psychiatry*. Jun 23, 2020;7(2):70-87. [FREE Full text] [doi: [10.1007/s40501-020-00203-1](https://doi.org/10.1007/s40501-020-00203-1)] [Medline: [33344106](https://pubmed.ncbi.nlm.nih.gov/33344106/)]
8. Rizzo A, Reger G, Gahm G, Difede J, Rothbaum B. Virtual reality exposure therapy for combat-related PTSD. In: LeDoux JE, Keane T, Shiromani P, editors. *Post-Traumatic Stress Disorder: Basic Science and Clinical Practice*. Totowa, NJ: Humana Press; 2009:375-399.
9. Beidel DC, Frueh BC, Neer SM, Bowers CA, Trachik B, Uhde TW, et al. Trauma management therapy with virtual-reality augmented exposure therapy for combat-related PTSD: a randomized controlled trial. *J Anxiety Disord*. Jan 2019;61:64-74. [doi: [10.1016/j.janxdis.2017.08.005](https://doi.org/10.1016/j.janxdis.2017.08.005)] [Medline: [28865911](https://pubmed.ncbi.nlm.nih.gov/28865911/)]
10. Eshuis LV, van Gelderen MJ, van Zuiden M, Nijdam MJ, Vermetten E, Olff M, et al. Efficacy of immersive PTSD treatments: a systematic review of virtual and augmented reality exposure therapy and a meta-analysis of virtual reality exposure therapy. *J Psychiatr Res*. Nov 2021;143:516-527. [FREE Full text] [doi: [10.1016/j.jpsychires.2020.11.030](https://doi.org/10.1016/j.jpsychires.2020.11.030)] [Medline: [33248674](https://pubmed.ncbi.nlm.nih.gov/33248674/)]
11. Heo S, Park JH. Effects of virtual reality-based graded exposure therapy on PTSD symptoms: a systematic review and meta-analysis. *Int J Environ Res Public Health*. Nov 29, 2022;19(23):15911. [FREE Full text] [doi: [10.3390/ijerph192315911](https://doi.org/10.3390/ijerph192315911)] [Medline: [36497989](https://pubmed.ncbi.nlm.nih.gov/36497989/)]
12. Kothgassner OD, Goreis A, Kafka JX, Van Eickels RL, Plener PL, Felnhofer A. Virtual reality exposure therapy for posttraumatic stress disorder (PTSD): a meta-analysis. *Eur J Psychotraumatol*. Aug 19, 2019;10(1):1654782. [FREE Full text] [doi: [10.1080/20008198.2019.1654782](https://doi.org/10.1080/20008198.2019.1654782)] [Medline: [31489138](https://pubmed.ncbi.nlm.nih.gov/31489138/)]
13. Adrien V, Bui E, Baubet T, Galletto CP, Duhamel J, McNamara A, et al. Beyond virtual reality: towards screen-free interfaces for post-traumatic stress disorder interventions. *PsyArXiv Preprints*. Preprint posted online November 17, 2023. [doi: [10.31234/osf.io/24beh](https://doi.org/10.31234/osf.io/24beh)]
14. Rabellino D, Harricharan S, Frewen PA, Burin D, McKinnon MC, Lanius RA. "I can't tell whether it's my hand": a pilot study of the neurophenomenology of body representation during the rubber hand illusion in trauma-related disorders. *Eur J Psychotraumatol*. 2016;7:32918. [FREE Full text] [doi: [10.3402/ejpt.v7.32918](https://doi.org/10.3402/ejpt.v7.32918)] [Medline: [27876453](https://pubmed.ncbi.nlm.nih.gov/27876453/)]
15. Rabellino D, Burin D, Harricharan S, Lloyd C, Frewen PA, McKinnon MC, et al. Altered sense of body ownership and agency in posttraumatic stress disorder and its dissociative subtype: a rubber hand illusion study. *Front Hum Neurosci*. May 1, 2018;12:163. [FREE Full text] [doi: [10.3389/fnhum.2018.00163](https://doi.org/10.3389/fnhum.2018.00163)] [Medline: [29765311](https://pubmed.ncbi.nlm.nih.gov/29765311/)]
16. van der Hart O, van Dijke A, van Son M, Steele K. Somatoform dissociation in traumatized World War I combat soldiers. *J Trauma Dissociation*. Mar 28, 2001;1(4):33-66. [doi: [10.1300/J229v01n04\\_03](https://doi.org/10.1300/J229v01n04_03)]
17. Ataria Y. *Body Disownership in Complex Posttraumatic Stress Disorder*. London, United Kingdom: Palgrave Macmillan; 2018.
18. Ataria Y, Gallagher S. Somatic apathy: body disownership in the context of torture. *J Phenomenol Psychol*. Jun 10, 2015;46(1):105-122. [doi: [10.1163/15691624-12341286](https://doi.org/10.1163/15691624-12341286)]
19. Bracha HS. Freeze, flight, fight, fright, faint: adaptationist perspectives on the acute stress response spectrum. *CNS Spectr*. Sep 2004;9(9):679-685. [FREE Full text] [doi: [10.1017/s1092852900001954](https://doi.org/10.1017/s1092852900001954)] [Medline: [15337864](https://pubmed.ncbi.nlm.nih.gov/15337864/)]
20. Arieli A, Ataria Y. Helplessness: the inability to know-that you don't know-how. *Philos Psychol*. May 23, 2018;31(6):948-968. [doi: [10.1080/09515089.2018.1468559](https://doi.org/10.1080/09515089.2018.1468559)]
21. Ataria Y. Sense of ownership and sense of agency during trauma. *Phenom Cogn Sci*. Aug 30, 2013;14(1):199-212. [doi: [10.1007/s11097-013-9334-y](https://doi.org/10.1007/s11097-013-9334-y)]
22. Ataria Y. Trauma from an enactive perspective: the collapse of the knowing-how structure. *Adapt Behav*. May 27, 2015;23(3):143-154. [doi: [10.1177/1059712315578542](https://doi.org/10.1177/1059712315578542)]

23. Ataria Y. Dissociation during trauma: the ownership-agency tradeoff model. *Phenom Cogn Sci*. Oct 17, 2014;14(4):1037-1053. [doi: [10.1007/s11097-014-9392-9](https://doi.org/10.1007/s11097-014-9392-9)]
24. Linson A, Friston K. Reframing PTSD for computational psychiatry with the active inference framework. *Cogn Neuropsychiatry*. Sep 2019;24(5):347-368. [FREE Full text] [doi: [10.1080/13546805.2019.1665994](https://doi.org/10.1080/13546805.2019.1665994)] [Medline: [31564212](https://pubmed.ncbi.nlm.nih.gov/31564212/)]
25. Linson A, Parr T, Friston KJ. Active inference, stressors, and psychological trauma: a neuroethological model of (mal)adaptive explore-exploit dynamics in ecological context. *Behav Brain Res*. Feb 17, 2020;380:112421. [FREE Full text] [doi: [10.1016/j.bbr.2019.112421](https://doi.org/10.1016/j.bbr.2019.112421)] [Medline: [31830495](https://pubmed.ncbi.nlm.nih.gov/31830495/)]
26. Webb RE, Widseth JC. Traumas with and without a sense of agency. *J Aggress Maltreatment Trauma*. Jul 23, 2009;18(5):532-546. [doi: [10.1080/10926770903050993](https://doi.org/10.1080/10926770903050993)]
27. Beste JE. The vulnerable self and loss of agency: trauma theory and the challenge to a Rahnerian theology of freedom and grace. In: Beste JE, editor. *God and the Victim: Traumatic Intrusions on Grace and Freedom*. Oxford, United Kingdom. Oxford University Press; 2007.
28. Frabetti M, Gayraud F, Auxéméry Y. [Study of agency in the discourse of women suffering from post-traumatic stress disorder in the aftermath of domestic violence]. *Encephale*. Oct 2023;49(5):516-524. [doi: [10.1016/j.encep.2022.09.002](https://doi.org/10.1016/j.encep.2022.09.002)] [Medline: [36257851](https://pubmed.ncbi.nlm.nih.gov/36257851/)]
29. Tsakiris M, Schütz-Bosbach S, Gallagher S. On agency and body-ownership: phenomenological and neurocognitive reflections. *Conscious Cogn*. Sep 2007;16(3):645-660. [doi: [10.1016/j.concog.2007.05.012](https://doi.org/10.1016/j.concog.2007.05.012)] [Medline: [17616469](https://pubmed.ncbi.nlm.nih.gov/17616469/)]
30. Braun N, Debener S, Spychala N, Bongartz E, Sörös P, Müller HH, et al. The senses of agency and ownership: a review. *Front Psychol*. Apr 16, 2018;9:535. [FREE Full text] [doi: [10.3389/fpsyg.2018.00535](https://doi.org/10.3389/fpsyg.2018.00535)] [Medline: [29713301](https://pubmed.ncbi.nlm.nih.gov/29713301/)]
31. Tsakiris M, Hesse MD, Boy C, Haggard P, Fink GR. Neural signatures of body ownership: a sensory network for bodily self-consciousness. *Cereb Cortex*. Oct 2007;17(10):2235-2244. [doi: [10.1093/cercor/bhl131](https://doi.org/10.1093/cercor/bhl131)] [Medline: [17138596](https://pubmed.ncbi.nlm.nih.gov/17138596/)]
32. Tsakiris M. Looking for myself: current multisensory input alters self-face recognition. *PLoS One*. 2008;3(12):e4040. [FREE Full text] [doi: [10.1371/journal.pone.0004040](https://doi.org/10.1371/journal.pone.0004040)] [Medline: [19107208](https://pubmed.ncbi.nlm.nih.gov/19107208/)]
33. Chancel M, Ehrsson HH. Which hand is mine? Discriminating body ownership perception in a two-alternative forced-choice task. *Atten Percept Psychophys*. Nov 2020;82(8):4058-4083. [FREE Full text] [doi: [10.3758/s13414-020-02107-x](https://doi.org/10.3758/s13414-020-02107-x)] [Medline: [32856222](https://pubmed.ncbi.nlm.nih.gov/32856222/)]
34. Tsakiris M. My body in the brain: a neurocognitive model of body-ownership. *Neuropsychologia*. Feb 2010;48(3):703-712. [doi: [10.1016/j.neuropsychologia.2009.09.034](https://doi.org/10.1016/j.neuropsychologia.2009.09.034)] [Medline: [19819247](https://pubmed.ncbi.nlm.nih.gov/19819247/)]
35. Ehrsson HH, Spence C, Passingham RE. That's my hand! Activity in premotor cortex reflects feeling of ownership of a limb. *Science*. Aug 06, 2004;305(5685):875-877. [doi: [10.1126/science.1097011](https://doi.org/10.1126/science.1097011)] [Medline: [15232072](https://pubmed.ncbi.nlm.nih.gov/15232072/)]
36. Guterstam A, Larsson DE, Szczotka J, Ehrsson HH. Duplication of the bodily self: a perceptual illusion of dual full-body ownership and dual self-location. *R Soc Open Sci*. Dec 2020;7(12):201911. [FREE Full text] [doi: [10.1098/rsos.201911](https://doi.org/10.1098/rsos.201911)] [Medline: [33489299](https://pubmed.ncbi.nlm.nih.gov/33489299/)]
37. Litwin P, Zybura B, Motyka P. Tactile information counteracts the attenuation of rubber hand illusion attributable to increased visuo-proprioceptive divergence. *PLoS One*. Dec 30, 2020;15(12):e0244594. [FREE Full text] [doi: [10.1371/journal.pone.0244594](https://doi.org/10.1371/journal.pone.0244594)] [Medline: [33378385](https://pubmed.ncbi.nlm.nih.gov/33378385/)]
38. Litwin P. Extending Bayesian models of the rubber hand illusion. *Multisens Res*. Jan 08, 2020;33(2):127-160. [doi: [10.1163/22134808-20191440](https://doi.org/10.1163/22134808-20191440)] [Medline: [31648196](https://pubmed.ncbi.nlm.nih.gov/31648196/)]
39. Gallagher S. Philosophical conceptions of the self: implications for cognitive science. *Trends Cogn Sci*. Jan 2000;4(1):14-21. [doi: [10.1016/s1364-6613\(99\)01417-5](https://doi.org/10.1016/s1364-6613(99)01417-5)] [Medline: [10637618](https://pubmed.ncbi.nlm.nih.gov/10637618/)]
40. Haggard P, Chambon V. Sense of agency. *Curr Biol*. May 22, 2012;22(10):R390-R392. [FREE Full text] [doi: [10.1016/j.cub.2012.02.040](https://doi.org/10.1016/j.cub.2012.02.040)] [Medline: [22625851](https://pubmed.ncbi.nlm.nih.gov/22625851/)]
41. Blakemore SJ, Wolpert DM, Frith CD. Abnormalities in the awareness of action. *Trends Cogn Sci*. Jun 01, 2002;6(6):237-242. [doi: [10.1016/s1364-6613\(02\)01907-1](https://doi.org/10.1016/s1364-6613(02)01907-1)] [Medline: [12039604](https://pubmed.ncbi.nlm.nih.gov/12039604/)]
42. Gallagher S, Trigg D. Agency and anxiety: delusions of control and loss of control in schizophrenia and agoraphobia. *Front Hum Neurosci*. Sep 26, 2016;10:459. [FREE Full text] [doi: [10.3389/fnhum.2016.00459](https://doi.org/10.3389/fnhum.2016.00459)] [Medline: [27725796](https://pubmed.ncbi.nlm.nih.gov/27725796/)]
43. Seghezzi S, Giannini G, Zapparoli L. Neurofunctional correlates of body-ownership and sense of agency: a meta-analytical account of self-consciousness. *Cortex*. Dec 2019;121:169-178. [doi: [10.1016/j.cortex.2019.08.018](https://doi.org/10.1016/j.cortex.2019.08.018)] [Medline: [31629195](https://pubmed.ncbi.nlm.nih.gov/31629195/)]
44. Tsakiris M, Longo MR, Haggard P. Having a body versus moving your body: neural signatures of agency and body-ownership. *Neuropsychologia*. Jul 2010;48(9):2740-2749. [doi: [10.1016/j.neuropsychologia.2010.05.021](https://doi.org/10.1016/j.neuropsychologia.2010.05.021)] [Medline: [20510255](https://pubmed.ncbi.nlm.nih.gov/20510255/)]
45. Farrer C, Franck N, Paillard J, Jeannerod M. The role of proprioception in action recognition. *Conscious Cogn*. Dec 2003;12(4):609-619. [doi: [10.1016/s1053-8100\(03\)00047-3](https://doi.org/10.1016/s1053-8100(03)00047-3)] [Medline: [14656504](https://pubmed.ncbi.nlm.nih.gov/14656504/)]
46. Wegner DM. Précis of the illusion of conscious will. *Behav Brain Sci*. Oct 2004;27(5):649-59; discussion 659. [doi: [10.1017/s0140525x04000159](https://doi.org/10.1017/s0140525x04000159)] [Medline: [15895616](https://pubmed.ncbi.nlm.nih.gov/15895616/)]
47. Synofzik M, Vosgerau G, Newen A. Beyond the comparator model: a multifactorial two-step account of agency. *Conscious Cogn*. Mar 2008;17(1):219-239. [doi: [10.1016/j.concog.2007.03.010](https://doi.org/10.1016/j.concog.2007.03.010)] [Medline: [17482480](https://pubmed.ncbi.nlm.nih.gov/17482480/)]
48. Moore JW, Fletcher PC. Sense of agency in health and disease: a review of cue integration approaches. *Conscious Cogn*. Mar 2012;21(1):59-68. [FREE Full text] [doi: [10.1016/j.concog.2011.08.010](https://doi.org/10.1016/j.concog.2011.08.010)] [Medline: [21920777](https://pubmed.ncbi.nlm.nih.gov/21920777/)]



49. Caspar EA, De Beir A, Magalhaes De Saldanha Da Gama PA, Yernaux F, Cleeremans A, Vanderborcht B. New frontiers in the rubber hand experiment: when a robotic hand becomes one's own. *Behav Res Methods*. Sep 2015;47(3):744-755. [doi: [10.3758/s13428-014-0498-3](https://doi.org/10.3758/s13428-014-0498-3)] [Medline: [24942249](https://pubmed.ncbi.nlm.nih.gov/24942249/)]
50. Pyasik M, Furlanetto T, Pia L. The role of body-related afferent signals in human sense of agency. *J Exp Neurosci*. May 16, 2019;13:1179069519849907. [FREE Full text] [doi: [10.1177/1179069519849907](https://doi.org/10.1177/1179069519849907)] [Medline: [31205423](https://pubmed.ncbi.nlm.nih.gov/31205423/)]
51. Schütz-Bosbach S, Avenanti A, Aglioti SM, Haggard P. Don't do it! Cortical inhibition and self-attribution during action observation. *J Cogn Neurosci*. Jun 2009;21(6):1215-1227. [doi: [10.1162/jocn.2009.21068](https://doi.org/10.1162/jocn.2009.21068)] [Medline: [18702585](https://pubmed.ncbi.nlm.nih.gov/18702585/)]
52. Seghezzi S, Zirone E, Paulesu E, Zapparoli L. The brain in (willed) action: a meta-analytical comparison of imaging studies on motor intentionality and sense of agency. *Front Psychol*. Apr 12, 2019;10:804. [FREE Full text] [doi: [10.3389/fpsyg.2019.00804](https://doi.org/10.3389/fpsyg.2019.00804)] [Medline: [31031676](https://pubmed.ncbi.nlm.nih.gov/31031676/)]
53. Gordon EM, Chauvin RJ, Van AN, Rajesh A, Nielsen A, Newbold DJ, et al. A somato-cognitive action network alternates with effector regions in motor cortex. *Nature*. May 2023;617(7960):351-359. [FREE Full text] [doi: [10.1038/s41586-023-05964-2](https://doi.org/10.1038/s41586-023-05964-2)] [Medline: [37076628](https://pubmed.ncbi.nlm.nih.gov/37076628/)]
54. Farrer C, Frith CD. Experiencing oneself vs another person as being the cause of an action: the neural correlates of the experience of agency. *Neuroimage*. Mar 2002;15(3):596-603. [doi: [10.1006/nimg.2001.1009](https://doi.org/10.1006/nimg.2001.1009)] [Medline: [11848702](https://pubmed.ncbi.nlm.nih.gov/11848702/)]
55. Bulot V, Thomas P, Delevoye-Turrell Y. [Sense of agency: experiencing is not judging]. *Encephale*. Sep 2007;33(4 Pt 1):603-608. [doi: [10.1016/s0013-7006\(07\)92060-6](https://doi.org/10.1016/s0013-7006(07)92060-6)] [Medline: [18033150](https://pubmed.ncbi.nlm.nih.gov/18033150/)]
56. Gallagher S. Self-defense: deflecting deflationary and eliminativist critiques of the sense of ownership. *Front Psychol*. Sep 21, 2017;8:1612. [FREE Full text] [doi: [10.3389/fpsyg.2017.01612](https://doi.org/10.3389/fpsyg.2017.01612)] [Medline: [28970813](https://pubmed.ncbi.nlm.nih.gov/28970813/)]
57. Daprati E, Franck N, Georgieff N, Proust J, Pacherie E, Dalery J, et al. Looking for the agent: an investigation into consciousness of action and self-consciousness in schizophrenic patients. *Cognition*. Dec 1997;65(1):71-86. [doi: [10.1016/s0010-0277\(97\)00039-5](https://doi.org/10.1016/s0010-0277(97)00039-5)] [Medline: [9455171](https://pubmed.ncbi.nlm.nih.gov/9455171/)]
58. Franck N, Farrer C, Georgieff N, Marie-Cardine M, Daléry J, d'Amato T, et al. Defective recognition of one's own actions in patients with schizophrenia. *Am J Psychiatry*. Mar 2001;158(3):454-459. [doi: [10.1176/appi.ajp.158.3.454](https://doi.org/10.1176/appi.ajp.158.3.454)] [Medline: [11229988](https://pubmed.ncbi.nlm.nih.gov/11229988/)]
59. Stephens GL, Graham G. *When Self-Consciousness Breaks: Alien Voices and Inserted Thoughts*. Cambridge, MA: The MIT Press; 2000.
60. Haggard P, Tsakiris M. The experience of agency: feelings, judgments, and responsibility. *Curr Dir Psychol Sci*. Aug 01, 2009;18(4):242-246. [doi: [10.1111/j.1467-8721.2009.01644.x](https://doi.org/10.1111/j.1467-8721.2009.01644.x)]
61. Bermúdez JL. Bodily awareness and self-consciousness. In: Gallagher S, editor. *The Oxford Handbook of the Self*. Oxford, United Kingdom: Oxford University Press; 2011.
62. Gallagher M, Colzi C, Sedda A. Dissociation of proprioceptive drift and feelings of ownership in the somatic rubber hand illusion. *Acta Psychol (Amst)*. Jan 2021;212:103192. [FREE Full text] [doi: [10.1016/j.actpsy.2020.103192](https://doi.org/10.1016/j.actpsy.2020.103192)] [Medline: [33137614](https://pubmed.ncbi.nlm.nih.gov/33137614/)]
63. Grünbaum T. The feeling of agency hypothesis: a critique. *Synthese*. Feb 24, 2015;192(10):3313-3337. [doi: [10.1007/s11229-015-0704-6](https://doi.org/10.1007/s11229-015-0704-6)]
64. Decety J, Perani D, Jeannerod M, Bettinardi V, Tadary B, Woods R, et al. Mapping motor representations with positron emission tomography. *Nature*. Oct 13, 1994;371(6498):600-602. [doi: [10.1038/371600a0](https://doi.org/10.1038/371600a0)] [Medline: [7935791](https://pubmed.ncbi.nlm.nih.gov/7935791/)]
65. Botvinick M, Cohen J. Rubber hands 'feel' touch that eyes see. *Nature*. Feb 19, 1998;391(6669):756. [doi: [10.1038/35784](https://doi.org/10.1038/35784)] [Medline: [9486643](https://pubmed.ncbi.nlm.nih.gov/9486643/)]
66. Aarts H, Custers R, Wegner DM. On the inference of personal authorship: enhancing experienced agency by priming effect information. *Conscious Cogn*. Sep 2005;14(3):439-458. [doi: [10.1016/j.concog.2004.11.001](https://doi.org/10.1016/j.concog.2004.11.001)] [Medline: [16091264](https://pubmed.ncbi.nlm.nih.gov/16091264/)]
67. Moore JW, Obhi SS. Intentional binding and the sense of agency: a review. *Conscious Cogn*. Mar 2012;21(1):546-561. [doi: [10.1016/j.concog.2011.12.002](https://doi.org/10.1016/j.concog.2011.12.002)] [Medline: [22240158](https://pubmed.ncbi.nlm.nih.gov/22240158/)]
68. Dewey JA, Knoblich G. Do implicit and explicit measures of the sense of agency measure the same thing? *PLoS One*. Oct 16, 2014;9(10):e110118. [FREE Full text] [doi: [10.1371/journal.pone.0110118](https://doi.org/10.1371/journal.pone.0110118)] [Medline: [25330184](https://pubmed.ncbi.nlm.nih.gov/25330184/)]
69. Tapal A, Oren E, Dar R, Eitam B. The sense of agency scale: a measure of consciously perceived control over one's mind, body, and the immediate environment. *Front Psychol*. Sep 12, 2017;8:1552. [FREE Full text] [doi: [10.3389/fpsyg.2017.01552](https://doi.org/10.3389/fpsyg.2017.01552)] [Medline: [28955273](https://pubmed.ncbi.nlm.nih.gov/28955273/)]
70. Hurault JC, Broc G, Crône L, Tedesco A, Brunel L. Measuring the sense of agency: a French adaptation and validation of the sense of agency scale (F-SoAS). *Front Psychol*. Oct 8, 2020;11:584145. [FREE Full text] [doi: [10.3389/fpsyg.2020.584145](https://doi.org/10.3389/fpsyg.2020.584145)] [Medline: [33132992](https://pubmed.ncbi.nlm.nih.gov/33132992/)]
71. Kalckert A, Ehrsson HH. Moving a rubber hand that feels like your own: a dissociation of ownership and agency. *Front Hum Neurosci*. Mar 14, 2012;6:40. [FREE Full text] [doi: [10.3389/fnhum.2012.00040](https://doi.org/10.3389/fnhum.2012.00040)] [Medline: [22435056](https://pubmed.ncbi.nlm.nih.gov/22435056/)]
72. Kalckert A, Ehrsson HH. The moving rubber hand illusion revisited: comparing movements and visuotactile stimulation to induce illusory ownership. *Conscious Cogn*. May 2014;26:117-132. [FREE Full text] [doi: [10.1016/j.concog.2014.02.003](https://doi.org/10.1016/j.concog.2014.02.003)] [Medline: [24705182](https://pubmed.ncbi.nlm.nih.gov/24705182/)]
73. Gallagher S. Neurocognitive models of schizophrenia: a neurophenomenological critique. *Psychopathology*. 2004;37(1):8-19. [doi: [10.1159/000077014](https://doi.org/10.1159/000077014)] [Medline: [14988645](https://pubmed.ncbi.nlm.nih.gov/14988645/)]

74. Torregrossa LJ, Snodgrass MA, Hong SJ, Nichols HS, Glerean E, Nummenmaa L, et al. Anomalous bodily maps of emotions in schizophrenia. *Schizophr Bull*. Sep 11, 2019;45(5):1060-1067. [FREE Full text] [doi: [10.1093/schbul/sby179](https://doi.org/10.1093/schbul/sby179)] [Medline: [30551180](https://pubmed.ncbi.nlm.nih.gov/30551180/)]
75. Ardizzi M, Ambrosecchia M, Buratta L, Ferri F, Ferroni F, Palladini B, et al. The motor roots of minimal self disorders in schizophrenia. *Schizophr Res*. Apr 2020;218:302-303. [doi: [10.1016/j.schres.2020.03.007](https://doi.org/10.1016/j.schres.2020.03.007)] [Medline: [32171636](https://pubmed.ncbi.nlm.nih.gov/32171636/)]
76. Pitman RK, Rasmusson AM, Koenen KC, Shin LM, Orr SP, Gilbertson MW, et al. Biological studies of post-traumatic stress disorder. *Nat Rev Neurosci*. Nov 2012;13(11):769-787. [FREE Full text] [doi: [10.1038/nrn3339](https://doi.org/10.1038/nrn3339)] [Medline: [23047775](https://pubmed.ncbi.nlm.nih.gov/23047775/)]
77. Myers CS. *Shell Shock in France, 1914-1918: Based on a War Diary*. Cambridge, United Kingdom. Cambridge University Press; 1940.
78. Forster GL, Simons RM, Baugh LA. Revisiting the role of the amygdala in posttraumatic stress disorder. In: Ferry B, editor. *The Amygdala - Where Emotions Shape Perception, Learning and Memories*. London, United Kingdom. IntechOpen; 2017.
79. Malikowska-Racia N, Salat K. Recent advances in the neurobiology of posttraumatic stress disorder: a review of possible mechanisms underlying an effective pharmacotherapy. *Pharmacol Res*. Apr 2019;142:30-49. [doi: [10.1016/j.phrs.2019.02.001](https://doi.org/10.1016/j.phrs.2019.02.001)] [Medline: [30742899](https://pubmed.ncbi.nlm.nih.gov/30742899/)]
80. Likhtik E, Stujenske JM, Topiwala MA, Harris AZ, Gordon JA. Prefrontal entrainment of amygdala activity signals safety in learned fear and innate anxiety. *Nat Neurosci*. Jan 2014;17(1):106-113. [FREE Full text] [doi: [10.1038/nn.3582](https://doi.org/10.1038/nn.3582)] [Medline: [24241397](https://pubmed.ncbi.nlm.nih.gov/24241397/)]
81. Likhtik E, Paz R. Amygdala-prefrontal interactions in (mal)adaptive learning. *Trends Neurosci*. Mar 2015;38(3):158-166. [FREE Full text] [doi: [10.1016/j.tins.2014.12.007](https://doi.org/10.1016/j.tins.2014.12.007)] [Medline: [25583269](https://pubmed.ncbi.nlm.nih.gov/25583269/)]
82. Lopresto D, Schipper P, Homberg JR. Neural circuits and mechanisms involved in fear generalization: implications for the pathophysiology and treatment of posttraumatic stress disorder. *Neurosci Biobehav Rev*. Jan 2016;60:31-42. [doi: [10.1016/j.neubiorev.2015.10.009](https://doi.org/10.1016/j.neubiorev.2015.10.009)] [Medline: [26519776](https://pubmed.ncbi.nlm.nih.gov/26519776/)]
83. Ross DA, Arbuckle MR, Travis MJ, Dwyer JB, van Schalkwyk GI, Ressler KJ. An integrated neuroscience perspective on formulation and treatment planning for posttraumatic stress disorder: an educational review. *JAMA Psychiatry*. Apr 01, 2017;74(4):407-415. [FREE Full text] [doi: [10.1001/jamapsychiatry.2016.3325](https://doi.org/10.1001/jamapsychiatry.2016.3325)] [Medline: [28273291](https://pubmed.ncbi.nlm.nih.gov/28273291/)]
84. Liberzon I, Abelson JL. Context processing and the neurobiology of post-traumatic stress disorder. *Neuron*. Oct 05, 2016;92(1):14-30. [FREE Full text] [doi: [10.1016/j.neuron.2016.09.039](https://doi.org/10.1016/j.neuron.2016.09.039)] [Medline: [27710783](https://pubmed.ncbi.nlm.nih.gov/27710783/)]
85. Shalev A, Liberzon I, Marmar C. Post-traumatic stress disorder. *N Engl J Med*. Jun 22, 2017;376(25):2459-2469. [doi: [10.1056/NEJMra1612499](https://doi.org/10.1056/NEJMra1612499)] [Medline: [28636846](https://pubmed.ncbi.nlm.nih.gov/28636846/)]
86. Akiki TJ, Averill CL, Abdallah CG. A network-based neurobiological model of PTSD: evidence from structural and functional neuroimaging studies. *Curr Psychiatry Rep*. Sep 19, 2017;19(11):81. [FREE Full text] [doi: [10.1007/s11920-017-0840-4](https://doi.org/10.1007/s11920-017-0840-4)] [Medline: [28924828](https://pubmed.ncbi.nlm.nih.gov/28924828/)]
87. Brunet A, Weiss DS, Metzler TJ, Best SR, Neylan TC, Rogers C, et al. The Peritraumatic Distress Inventory: a proposed measure of PTSD criterion A2. *Am J Psychiatry*. Sep 2001;158(9):1480-1485. [doi: [10.1176/appi.ajp.158.9.1480](https://doi.org/10.1176/appi.ajp.158.9.1480)] [Medline: [11532735](https://pubmed.ncbi.nlm.nih.gov/11532735/)]
88. Kozłowska K, Walker P, McLean L, Carrive P. Fear and the defense cascade: clinical implications and management. *Harv Rev Psychiatry*. 2015;23(4):263-287. [FREE Full text] [doi: [10.1097/HRP.000000000000065](https://doi.org/10.1097/HRP.000000000000065)] [Medline: [26062169](https://pubmed.ncbi.nlm.nih.gov/26062169/)]
89. van der Velden PG, Wittmann L. The independent predictive value of peritraumatic dissociation for PTSD symptomatology after type I trauma: a systematic review of prospective studies. *Clin Psychol Rev*. Jul 2008;28(6):1009-1020. [FREE Full text] [doi: [10.1016/j.cpr.2008.02.006](https://doi.org/10.1016/j.cpr.2008.02.006)] [Medline: [18406027](https://pubmed.ncbi.nlm.nih.gov/18406027/)]
90. Herman JL. *Trauma and Recovery: The Aftermath of Violence--From Domestic Abuse to Political Terror*. New York, NY. Basic Books; 2015.
91. Thompson-Hollands J, Jun JJ, Sloan DM. The association between peritraumatic dissociation and PTSD symptoms: the mediating role of negative beliefs about the self. *J Trauma Stress*. Apr 2017;30(2):190-194. [FREE Full text] [doi: [10.1002/jts.22179](https://doi.org/10.1002/jts.22179)] [Medline: [28449364](https://pubmed.ncbi.nlm.nih.gov/28449364/)]
92. Spiegel D, Cardena E. Disintegrated experience: the dissociative disorders revisited. *J Abnorm Psychol*. Aug 1991;100(3):366-378. [doi: [10.1037//0021-843x.100.3.366](https://doi.org/10.1037//0021-843x.100.3.366)] [Medline: [1918616](https://pubmed.ncbi.nlm.nih.gov/1918616/)]
93. Vermetten E, Spiegel D. Trauma and dissociation: implications for borderline personality disorder. *Curr Psychiatry Rep*. Feb 2014;16(2):434. [doi: [10.1007/s11920-013-0434-8](https://doi.org/10.1007/s11920-013-0434-8)] [Medline: [24442670](https://pubmed.ncbi.nlm.nih.gov/24442670/)]
94. Seligman R, Kirmayer LJ. Dissociative experience and cultural neuroscience: narrative, metaphor and mechanism. *Cult Med Psychiatry*. Mar 2008;32(1):31-64. [FREE Full text] [doi: [10.1007/s11013-007-9077-8](https://doi.org/10.1007/s11013-007-9077-8)] [Medline: [18213511](https://pubmed.ncbi.nlm.nih.gov/18213511/)]
95. Schauer M, Elbert T. Dissociation following traumatic stress. *J Psychol*. Jan 2010;218(2). [doi: [10.1027/0044-3409/a000018](https://doi.org/10.1027/0044-3409/a000018)]
96. Marmar CR, Weiss DS, Metzler T. Peritraumatic dissociation and posttraumatic stress disorder. In: Bremner JD, Marmar CR, editors. *Trauma, Memory, and Dissociation*. Washington, DC. American Psychiatric Association; 1998.
97. Marshall GN, Schell TL. Reappraising the link between peritraumatic dissociation and PTSD symptom severity: evidence from a longitudinal study of community violence survivors. *J Abnorm Psychol*. Nov 2002;111(4):626-636. [doi: [10.1037//0021-843x.111.4.626](https://doi.org/10.1037//0021-843x.111.4.626)] [Medline: [12428776](https://pubmed.ncbi.nlm.nih.gov/12428776/)]

98. Birmes P, Brunet A, Carreras D, Ducassé JL, Charlet JP, Lauque D, et al. The predictive power of peritraumatic dissociation and acute stress symptoms for posttraumatic stress symptoms: a three-month prospective study. *Am J Psychiatry*. Jul 2003;160(7):1337-1339. [doi: [10.1176/appi.ajp.160.7.1337](https://doi.org/10.1176/appi.ajp.160.7.1337)] [Medline: [12832251](https://pubmed.ncbi.nlm.nih.gov/12832251/)]
99. Ozer EJ, Best SR, Lipsey TL, Weiss DS. Predictors of posttraumatic stress disorder and symptoms in adults: a meta-analysis. *Psychol Bull*. Jan 2003;129(1):52-73. [doi: [10.1037/0033-2909.129.1.52](https://doi.org/10.1037/0033-2909.129.1.52)] [Medline: [12555794](https://pubmed.ncbi.nlm.nih.gov/12555794/)]
100. Samuelson KW, Neylan TC, Lenoci M, Metzler TJ, Cardenas V, Weiner MW, et al. Longitudinal effects of PTSD on memory functioning. *J Int Neuropsychol Soc*. Nov 2009;15(6):853-861. [doi: [10.1017/S1355617709990282](https://doi.org/10.1017/S1355617709990282)] [Medline: [19703319](https://pubmed.ncbi.nlm.nih.gov/19703319/)]
101. Tulving E. Episodic and semantic memory. In: Tulving E, Donaldson W, editors. *Organization of Memory*. Cambridge, MA: Academic Press; 1972:381-403.
102. Samuelson KW. Post-traumatic stress disorder and declarative memory functioning: a review. *Dialogues Clin Neurosci*. 2011;13(3):346-351. [FREE Full text] [doi: [10.31887/DCNS.2011.13.2/ksamuelson](https://doi.org/10.31887/DCNS.2011.13.2/ksamuelson)] [Medline: [22033732](https://pubmed.ncbi.nlm.nih.gov/22033732/)]
103. van der Hart O, Nijenhuis ER, Steele K. *The Haunted Self: Structural Dissociation and the Treatment of Chronic Traumatization (Norton Series on Interpersonal Neurobiology)*. New York, NY: W. W. Norton & Company; 2006.
104. Nijenhuis E, van der Hart O, Steele K. Trauma-related structural dissociation of the personality. *Act Nerv Super*. Feb 23, 2017;52:1-23. [doi: [10.1007/bf03379560](https://doi.org/10.1007/bf03379560)]
105. Tulving E. Memory and consciousness. *Can Psychol*. 1985;26(1):1-12. [doi: [10.1037/h0080017](https://doi.org/10.1037/h0080017)]
106. Wheeler MA, Stuss DT, Tulving E. Toward a theory of episodic memory: the frontal lobes and autonoetic consciousness. *Psychol Bull*. May 1997;121(3):331-354. [doi: [10.1037/0033-2909.121.3.331](https://doi.org/10.1037/0033-2909.121.3.331)] [Medline: [9136640](https://pubmed.ncbi.nlm.nih.gov/9136640/)]
107. Woodward SH, Kaloupek DG, Grande LJ, Stegman WK, Kutter CJ, Leskin L, et al. Hippocampal volume and declarative memory function in combat-related PTSD. *J Int Neuropsychol Soc*. Nov 2009;15(6):830-839. [doi: [10.1017/S1355617709990476](https://doi.org/10.1017/S1355617709990476)] [Medline: [19703322](https://pubmed.ncbi.nlm.nih.gov/19703322/)]
108. Acheson DT, Gresack JE, Risbrough VB. Hippocampal dysfunction effects on context memory: possible etiology for posttraumatic stress disorder. *Neuropharmacology*. Feb 2012;62(2):674-685. [FREE Full text] [doi: [10.1016/j.neuropharm.2011.04.029](https://doi.org/10.1016/j.neuropharm.2011.04.029)] [Medline: [21596050](https://pubmed.ncbi.nlm.nih.gov/21596050/)]
109. Rougemont-Bücking A, Linnman C, Zeffiro TA, Zeidan MA, Lebron-Milad K, Rodriguez-Romaguera J, et al. Altered processing of contextual information during fear extinction in PTSD: an fMRI study. *CNS Neurosci Ther*. Aug 2011;17(4):227-236. [doi: [10.1111/j.1755-5949.2010.00152.x](https://doi.org/10.1111/j.1755-5949.2010.00152.x)] [Medline: [20406268](https://pubmed.ncbi.nlm.nih.gov/20406268/)]
110. Sheynin J, Liberzon I. Circuit dysregulation and circuit-based treatments in posttraumatic stress disorder. *Neurosci Lett*. May 10, 2017;649:133-138. [FREE Full text] [doi: [10.1016/j.neulet.2016.11.014](https://doi.org/10.1016/j.neulet.2016.11.014)] [Medline: [27845239](https://pubmed.ncbi.nlm.nih.gov/27845239/)]
111. Vitriol V, Cancino A, Weil K, Salgado C, Asenjo MA, Potthoff S. Depression and psychological trauma: an overview integrating current research and specific evidence of studies in the treatment of depression in public mental health services in Chile. *Depress Res Treat*. 2014;2014:608671. [FREE Full text] [doi: [10.1155/2014/608671](https://doi.org/10.1155/2014/608671)] [Medline: [24695633](https://pubmed.ncbi.nlm.nih.gov/24695633/)]
112. Kascakova N, Furstova J, Hasto J, Madarasova Geckova A, Tavel P. The unholy trinity: childhood trauma, adulthood anxiety, and long-term pain. *Int J Environ Res Public Health*. Jan 08, 2020;17(2):414. [FREE Full text] [doi: [10.3390/ijerph17020414](https://doi.org/10.3390/ijerph17020414)] [Medline: [31936285](https://pubmed.ncbi.nlm.nih.gov/31936285/)]
113. Semiz UB, Inanc L, Bezgin CH. Are trauma and dissociation related to treatment resistance in patients with obsessive-compulsive disorder? *Soc Psychiatry Psychiatr Epidemiol*. Aug 2014;49(8):1287-1296. [doi: [10.1007/s00127-013-0787-7](https://doi.org/10.1007/s00127-013-0787-7)] [Medline: [24213522](https://pubmed.ncbi.nlm.nih.gov/24213522/)]
114. Yehuda R, Hoge CW, McFarlane AC, Vermetten E, Lanius RA, Nievergelt CM, et al. Post-traumatic stress disorder. *Nat Rev Dis Primers*. Oct 08, 2015;1:15057. [doi: [10.1038/nrdp.2015.57](https://doi.org/10.1038/nrdp.2015.57)] [Medline: [27189040](https://pubmed.ncbi.nlm.nih.gov/27189040/)]
115. *Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition*. Washington, DC: American Psychiatric Association; 2013.
116. Weathers FW, Bovin MJ, Lee DJ, Sloan DM, Schnurr PP, Kaloupek DG, et al. The clinician-administered PTSD scale for DSM-5 (CAPS-5): development and initial psychometric evaluation in military veterans. *Psychol Assess*. Mar 2018;30(3):383-395. [FREE Full text] [doi: [10.1037/pas0000486](https://doi.org/10.1037/pas0000486)] [Medline: [28493729](https://pubmed.ncbi.nlm.nih.gov/28493729/)]
117. Wolf EJ, Hawn SE, Sullivan DR, Miller MW, Sanborn V, Brown E, et al. Neurobiological and genetic correlates of the dissociative subtype of posttraumatic stress disorder. *J Psychopathol Clin Sci*. May 2023;132(4):409-427. [doi: [10.1037/abn0000795](https://doi.org/10.1037/abn0000795)] [Medline: [37023279](https://pubmed.ncbi.nlm.nih.gov/37023279/)]
118. Harrison JE, Weber S, Jakob R, Chute CG. ICD-11: an international classification of diseases for the twenty-first century. *BMC Med Inform Decis Mak*. Nov 09, 2021;21(Suppl 6):206. [FREE Full text] [doi: [10.1186/s12911-021-01534-6](https://doi.org/10.1186/s12911-021-01534-6)] [Medline: [34753471](https://pubmed.ncbi.nlm.nih.gov/34753471/)]
119. Brewin CR, Cloitre M, Hyland P, Shevlin M, Maercker A, Bryant RA, et al. A review of current evidence regarding the ICD-11 proposals for diagnosing PTSD and complex PTSD. *Clin Psychol Rev*. Dec 2017;58:1-15. [doi: [10.1016/j.cpr.2017.09.001](https://doi.org/10.1016/j.cpr.2017.09.001)] [Medline: [29029837](https://pubmed.ncbi.nlm.nih.gov/29029837/)]
120. *International statistical classification of diseases and related health problems (ICD)*. World Health Organization. URL: <https://www.who.int/standards/classifications/classification-of-diseases> [accessed 2024-06-05]
121. Herman JL. Complex PTSD: a syndrome in survivors of prolonged and repeated trauma. *J Trauma Stress*. Jul 1992;5(3):377-391. [FREE Full text] [doi: [10.1002/jts.2490050305](https://doi.org/10.1002/jts.2490050305)]



122. Maercker A, Cloitre M, Bachem R, Schlumpf YR, Khoury B, Hitchcock C, et al. Complex post-traumatic stress disorder. *Lancet*. Jul 02, 2022;400(10345):60-72. [doi: [10.1016/S0140-6736\(22\)00821-2](https://doi.org/10.1016/S0140-6736(22)00821-2)] [Medline: [35780794](https://pubmed.ncbi.nlm.nih.gov/35780794/)]
123. Sachser C, Keller F, Goldbeck L. Complex PTSD as proposed for ICD-11: validation of a new disorder in children and adolescents and their response to trauma-focused cognitive behavioral therapy. *J Child Psychol Psychiatry*. Feb 2017;58(2):160-168. [doi: [10.1111/jcpp.12640](https://doi.org/10.1111/jcpp.12640)] [Medline: [27677771](https://pubmed.ncbi.nlm.nih.gov/27677771/)]
124. Murray H, Grey N, Warnock-Parkes E, Kerr A, Wild J, Clark DM, et al. Ten misconceptions about trauma-focused CBT for PTSD. *Cogn Behav Therap*. Jul 22, 2022;15:s1754470x22000307. [FREE Full text] [doi: [10.1017/S1754470X22000307](https://doi.org/10.1017/S1754470X22000307)] [Medline: [36247408](https://pubmed.ncbi.nlm.nih.gov/36247408/)]
125. van Minnen A, Harned MS, Zoellner L, Mills K. Examining potential contraindications for prolonged exposure therapy for PTSD. *Eur J Psychotraumatol*. 2012;3. [FREE Full text] [doi: [10.3402/ejpt.v3i0.18805](https://doi.org/10.3402/ejpt.v3i0.18805)] [Medline: [22893847](https://pubmed.ncbi.nlm.nih.gov/22893847/)]
126. Halvorsen JØ, Stenmark H, Neuner F, Nordahl HM. Does dissociation moderate treatment outcomes of narrative exposure therapy for PTSD? A secondary analysis from a randomized controlled clinical trial. *Behav Res Ther*. Jun 2014;57:21-28. [doi: [10.1016/j.brat.2014.03.010](https://doi.org/10.1016/j.brat.2014.03.010)] [Medline: [24762779](https://pubmed.ncbi.nlm.nih.gov/24762779/)]
127. Hoebner CM, De Kleine RA, Molendijk ML, Schoorl M, Oprel DA, Mouthaan J, et al. Impact of dissociation on the effectiveness of psychotherapy for post-traumatic stress disorder: meta-analysis. *BJPsych Open*. May 19, 2020;6(3):e53. [FREE Full text] [doi: [10.1192/bjo.2020.30](https://doi.org/10.1192/bjo.2020.30)] [Medline: [32423501](https://pubmed.ncbi.nlm.nih.gov/32423501/)]
128. Di Paolo EA, Buhmann T, Barandiaran XE. The sense of agency. In: *Sensorimotor: Life An Enactive Proposal*. Oxford, United Kingdom. Oxford University Press; 2017.
129. Bomyea J, Lang AJ. Accounting for intrusive thoughts in PTSD: contributions of cognitive control and deliberate regulation strategies. *J Affect Disord*. Mar 01, 2016;192:184-190. [FREE Full text] [doi: [10.1016/j.jad.2015.12.021](https://doi.org/10.1016/j.jad.2015.12.021)] [Medline: [26741045](https://pubmed.ncbi.nlm.nih.gov/26741045/)]
130. Falsetti SA, Monnier J, Davis JL, Resnick HS. Intrusive thoughts in posttraumatic stress disorder. *J Cogn Psychother*. Jun 2002;16(2):127-143. [doi: [10.1891/jcop.16.2.127.63993](https://doi.org/10.1891/jcop.16.2.127.63993)]
131. Shipherd JC, Salters-Pedneault K. Attention, memory, intrusive thoughts, and acceptance in PTSD: an update on the empirical literature for clinicians. *Cogn Behav Pract*. Nov 2008;15(4):349-363. [FREE Full text] [doi: [10.1016/j.cbpra.2008.01.003](https://doi.org/10.1016/j.cbpra.2008.01.003)]
132. Bryant RA, Sackville T, Dang ST, Moulds M, Guthrie R. Treating acute stress disorder: an evaluation of cognitive behavior therapy and supportive counseling techniques. *Am J Psychiatry*. Nov 1999;156(11):1780-1786. [doi: [10.1176/ajp.156.11.1780](https://doi.org/10.1176/ajp.156.11.1780)] [Medline: [10553743](https://pubmed.ncbi.nlm.nih.gov/10553743/)]
133. Kube T, Berg M, Kleim B, Herzog P. Rethinking post-traumatic stress disorder - a predictive processing perspective. *Neurosci Biobehav Rev*. Jun 2020;113:448-460. [doi: [10.1016/j.neubiorev.2020.04.014](https://doi.org/10.1016/j.neubiorev.2020.04.014)] [Medline: [32315695](https://pubmed.ncbi.nlm.nih.gov/32315695/)]
134. Shipherd JC, Beck JG. The effects of suppressing trauma-related thoughts on women with rape-related posttraumatic stress disorder. *Behav Res Ther*. Feb 1999;37(2):99-112. [doi: [10.1016/s0005-7967\(98\)00136-3](https://doi.org/10.1016/s0005-7967(98)00136-3)] [Medline: [9990742](https://pubmed.ncbi.nlm.nih.gov/9990742/)]
135. Tull MT, Trotman A, Duplinsky MS, Reynolds EK, Daughters SB, Potenza MN, et al. The effect of posttraumatic stress disorder on risk-taking propensity among crack/cocaine users in residential substance abuse treatment. *Depress Anxiety*. 2009;26(12):1158-1164. [FREE Full text] [doi: [10.1002/da.20637](https://doi.org/10.1002/da.20637)] [Medline: [19957281](https://pubmed.ncbi.nlm.nih.gov/19957281/)]
136. Tyler KA. Social and emotional outcomes of childhood sexual abuse: a review of recent research. *Aggress Violent Behav*. 2002;7(6):567-589. [doi: [10.1016/s1359-1789\(01\)00047-7](https://doi.org/10.1016/s1359-1789(01)00047-7)]
137. Terr LC. Childhood traumas: an outline and overview. *Am J Psychiatry*. Jan 1991;148(1):10-20. [doi: [10.1176/ajp.148.1.10](https://doi.org/10.1176/ajp.148.1.10)] [Medline: [1824611](https://pubmed.ncbi.nlm.nih.gov/1824611/)]
138. Freud S, Strachey J. *Beyond the Pleasure Principle*. Scotts Valley, CA. CreateSpace Independent Publishing Platform; 2010.
139. Winnicott DW. Transitional objects and transitional phenomena. In: Caldwell L, Taylor Robinson H, editors. *The Collected Works of D. W. Winnicott: Volume 3*. Oxford, United Kingdom. Oxford Academic Press; 2016.
140. Thompson BL, Waltz J. Mindfulness and experiential avoidance as predictors of posttraumatic stress disorder avoidance symptom severity. *J Anxiety Disord*. May 2010;24(4):409-415. [doi: [10.1016/j.janxdis.2010.02.005](https://doi.org/10.1016/j.janxdis.2010.02.005)] [Medline: [20304602](https://pubmed.ncbi.nlm.nih.gov/20304602/)]
141. Schneider A, Gudiño OG. Predicting avoidance symptoms in U.S. Latino youth exposed to community violence: the role of cultural values and behavioral inhibition. *J Trauma Stress*. Aug 2018;31(4):509-517. [doi: [10.1002/jts.22313](https://doi.org/10.1002/jts.22313)] [Medline: [30058738](https://pubmed.ncbi.nlm.nih.gov/30058738/)]
142. Măirean C. Fear and avoidance of driving among drivers involved in a road traffic crash. The role of traumatic fear and driving cognitions. *Transp Res Part F Traffic Psychol Behav*. Oct 2020;74:322-329. [doi: [10.1016/j.trf.2020.08.026](https://doi.org/10.1016/j.trf.2020.08.026)]
143. Couette M, Mouchabac S, Bourla A, Nuss P, Ferreri F. Social cognition in post-traumatic stress disorder: a systematic review. *Br J Clin Psychol*. Jun 2020;59(2):117-138. [doi: [10.1111/bjc.12238](https://doi.org/10.1111/bjc.12238)] [Medline: [31696974](https://pubmed.ncbi.nlm.nih.gov/31696974/)]
144. Galea S, Nandi A, Vlahov D. The epidemiology of post-traumatic stress disorder after disasters. *Epidemiol Rev*. 2005;27:78-91. [doi: [10.1093/epirev/mxi003](https://doi.org/10.1093/epirev/mxi003)] [Medline: [15958429](https://pubmed.ncbi.nlm.nih.gov/15958429/)]
145. Neria Y, Olfson M, Gameroff MJ, Wickramaratne P, Pilowsky D, Verdelli H, et al. Trauma exposure and posttraumatic stress disorder among primary care patients with bipolar spectrum disorder. *Bipolar Disord*. Jun 2008;10(4):503-510. [FREE Full text] [doi: [10.1111/j.1399-5618.2008.00589.x](https://doi.org/10.1111/j.1399-5618.2008.00589.x)] [Medline: [18452446](https://pubmed.ncbi.nlm.nih.gov/18452446/)]



146. Flynn AJ, Navarro GY, Basehore HK. PTSD avoidance symptoms associated with alcohol craving in treatment-seeking veteran population. *J Dual Diagn.* 2022;18(3):135-143. [doi: [10.1080/15504263.2022.2089799](https://doi.org/10.1080/15504263.2022.2089799)] [Medline: [35761472](https://pubmed.ncbi.nlm.nih.gov/35761472/)]
147. Basedow LA, Kuitunen-Paul S, Wiedmann MF, Roessner V, Golub Y. Self-reported PTSD is associated with increased use of MDMA in adolescents with substance use disorders. *Eur J Psychotraumatol.* Sep 28, 2021;12(1):1968140. [FREE Full text] [doi: [10.1080/20008198.2021.1968140](https://doi.org/10.1080/20008198.2021.1968140)] [Medline: [34603636](https://pubmed.ncbi.nlm.nih.gov/34603636/)]
148. Müller M, Vandeleur C, Rodgers S, Rössler W, Castelao E, Preisig M, et al. Posttraumatic stress avoidance symptoms as mediators in the development of alcohol use disorders after exposure to childhood sexual abuse in a Swiss community sample. *Child Abuse Negl.* Aug 2015;46:8-15. [FREE Full text] [doi: [10.1016/j.chiabu.2015.03.006](https://doi.org/10.1016/j.chiabu.2015.03.006)] [Medline: [25828861](https://pubmed.ncbi.nlm.nih.gov/25828861/)]
149. Ciccone A, Ferrant A. *Honte, Culpabilité Et Traumatisme*. Paris, France. Dunod; 2015.
150. Freud S. *Inhibitions, Symptoms and Anxiety*. Tempe, AZ. Norton; 1926.
151. Klein M. Notes on some schizoid mechanisms. *J Psychother Pract Res.* 1996;5(2):160-179. [FREE Full text] [Medline: [22700275](https://pubmed.ncbi.nlm.nih.gov/22700275/)]
152. Keane TM, Marshall AD, Taft CT. Posttraumatic stress disorder: etiology, epidemiology, and treatment outcome. *Annu Rev Clin Psychol.* 2006;2:161-197. [doi: [10.1146/annurev.clinpsy.2.022305.095305](https://doi.org/10.1146/annurev.clinpsy.2.022305.095305)] [Medline: [17716068](https://pubmed.ncbi.nlm.nih.gov/17716068/)]
153. Brewin CR, Andrews B, Valentine JD. Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *J Consult Clin Psychol.* Oct 2000;68(5):748-766. [doi: [10.1037//0022-006x.68.5.748](https://doi.org/10.1037//0022-006x.68.5.748)] [Medline: [11068961](https://pubmed.ncbi.nlm.nih.gov/11068961/)]
154. Cameron AY, Mamon D. Towards a better understanding of hypervigilance in combat veterans. *Mil Behav Health.* Jan 07, 2019;7(2):206-217. [doi: [10.1080/21635781.2018.1526144](https://doi.org/10.1080/21635781.2018.1526144)]
155. Kimble M, Boxwala M, Bean W, Maletsky K, Halper J, Spollen K, et al. The impact of hypervigilance: evidence for a forward feedback loop. *J Anxiety Disord.* Mar 2014;28(2):241-245. [FREE Full text] [doi: [10.1016/j.janxdis.2013.12.006](https://doi.org/10.1016/j.janxdis.2013.12.006)] [Medline: [24507631](https://pubmed.ncbi.nlm.nih.gov/24507631/)]
156. Shalev AY, Sahar T, Freedman S, Peri T, Glick N, Brandes D, et al. A prospective study of heart rate response following trauma and the subsequent development of posttraumatic stress disorder. *Arch Gen Psychiatry.* Jun 1998;55(6):553-559. [doi: [10.1001/archpsyc.55.6.553](https://doi.org/10.1001/archpsyc.55.6.553)] [Medline: [9633675](https://pubmed.ncbi.nlm.nih.gov/9633675/)]
157. Bryant RA, Salmon K, Sinclair E, Davidson P. Heart rate as a predictor of posttraumatic stress disorder in children. *Gen Hosp Psychiatry.* 2007;29(1):66-68. [doi: [10.1016/j.genhosppsy.2006.10.002](https://doi.org/10.1016/j.genhosppsy.2006.10.002)] [Medline: [17189749](https://pubmed.ncbi.nlm.nih.gov/17189749/)]
158. Seligowski AV, Misganaw B, Duffy LA, Ressler KJ, Guffanti G. Leveraging large-scale genetics of PTSD and cardiovascular disease to demonstrate robust shared risk and improve risk prediction accuracy. *Am J Psychiatry.* Nov 01, 2022;179(11):814-823. [FREE Full text] [doi: [10.1176/appi.ajp.21111113](https://doi.org/10.1176/appi.ajp.21111113)] [Medline: [36069022](https://pubmed.ncbi.nlm.nih.gov/36069022/)]
159. Wood J, Mathews A, Dalgleish T. Anxiety and cognitive inhibition. *Emotion.* Jun 2001;1(2):166-181. [doi: [10.1037/1528-3542.1.2.166](https://doi.org/10.1037/1528-3542.1.2.166)] [Medline: [12899195](https://pubmed.ncbi.nlm.nih.gov/12899195/)]
160. Menon V, Uddin LQ. Saliency, switching, attention and control: a network model of insula function. *Brain Struct Funct.* Jun 2010;214(5-6):655-667. [FREE Full text] [doi: [10.1007/s00429-010-0262-0](https://doi.org/10.1007/s00429-010-0262-0)] [Medline: [20512370](https://pubmed.ncbi.nlm.nih.gov/20512370/)]
161. Stewart L, Brewin CR, Muggleton NG, Javardi AH, Tcheang L. Hypervigilance without threat: eye-movements reveal vigilant behaviours. In: *Proceedings of the International Society for Traumatic Stress Studies 29th Annual Meeting*. 2013. Presented at: ISTSS 2013; November 7-9, 2013; Philadelphia, PA.
162. Coll SY, Eustache F, Doidy F, Fraisse F, Peschanski D, Dayan J, et al. Avoidance behaviour generalizes to eye processing in posttraumatic stress disorder. *Eur J Psychotraumatol.* 2022;13(1):2044661. [FREE Full text] [doi: [10.1080/20008198.2022.2044661](https://doi.org/10.1080/20008198.2022.2044661)]
163. Critchley HD, Harrison NA. Visceral influences on brain and behavior. *Neuron.* Feb 20, 2013;77(4):624-638. [FREE Full text] [doi: [10.1016/j.neuron.2013.02.008](https://doi.org/10.1016/j.neuron.2013.02.008)] [Medline: [23439117](https://pubmed.ncbi.nlm.nih.gov/23439117/)]
164. Schoeller F, Haar AJ, Jain A, Maes P. Enhancing human emotions with interoceptive technologies. *Phys Life Rev.* Dec 2019;31:310-319. [doi: [10.1016/j.plrev.2019.10.008](https://doi.org/10.1016/j.plrev.2019.10.008)] [Medline: [31757602](https://pubmed.ncbi.nlm.nih.gov/31757602/)]
165. Schoeller F, Horowitz AH, Jain A, Maes P, Reggente N, Christov-Moore L, et al. Interoceptive technologies for psychiatric interventions: from diagnosis to clinical applications. *Neurosci Biobehav Rev.* Jan 2024;156:105478. [doi: [10.1016/j.neubiorev.2023.105478](https://doi.org/10.1016/j.neubiorev.2023.105478)] [Medline: [38007168](https://pubmed.ncbi.nlm.nih.gov/38007168/)]
166. Anzieu D. *The Skin Ego*. London, United Kingdom. Yale University Press; 1989.
167. Schoeller F. Primary states of consciousness: a review of historical and contemporary developments. *Conscious Cogn.* Aug 2023;113:103536. [doi: [10.1016/j.concog.2023.103536](https://doi.org/10.1016/j.concog.2023.103536)] [Medline: [37321024](https://pubmed.ncbi.nlm.nih.gov/37321024/)]
168. Butler LD. Normative dissociation. *Psychiatr Clin North Am.* Mar 2006;29(1):45-62, viii. [doi: [10.1016/j.psc.2005.10.004](https://doi.org/10.1016/j.psc.2005.10.004)] [Medline: [16530586](https://pubmed.ncbi.nlm.nih.gov/16530586/)]
169. Taylor MK, Morgan CA. Spontaneous and deliberate dissociative states in military personnel: relationships to objective performance under stress. *Mil Med.* Sep 2014;179(9):955-958. [doi: [10.7205/milmed-d-14-00081](https://doi.org/10.7205/milmed-d-14-00081)]
170. Morgan CA3, Taylor MK. Spontaneous and deliberate dissociative states in military personnel: are such states helpful? *J Trauma Stress.* Aug 2013;26(4):492-497. [doi: [10.1002/jts.21834](https://doi.org/10.1002/jts.21834)] [Medline: [23893559](https://pubmed.ncbi.nlm.nih.gov/23893559/)]
171. Liriano F, Hatten C, Schwartz TL. Ketamine as treatment for post-traumatic stress disorder: a review. *Drugs Context.* Apr 08, 2019;8:212305. [FREE Full text] [doi: [10.7573/dic.212305](https://doi.org/10.7573/dic.212305)] [Medline: [31007698](https://pubmed.ncbi.nlm.nih.gov/31007698/)]

172. Elsouiri KN, Kalhori S, Colunge D, Grabarczyk G, Hanna G, Carrasco C, et al. Psychoactive drugs in the management of post traumatic stress disorder: a promising new horizon. *Cureus*. May 23, 2022;14(5):e25235. [FREE Full text] [doi: [10.7759/cureus.25235](https://doi.org/10.7759/cureus.25235)] [Medline: [35747039](https://pubmed.ncbi.nlm.nih.gov/35747039/)]
173. Feder A, Parides MK, Murrough JW, Perez AM, Morgan JE, Saxena S, et al. Efficacy of intravenous ketamine for treatment of chronic posttraumatic stress disorder: a randomized clinical trial. *JAMA Psychiatry*. Jun 2014;71(6):681-688. [doi: [10.1001/jamapsychiatry.2014.62](https://doi.org/10.1001/jamapsychiatry.2014.62)] [Medline: [24740528](https://pubmed.ncbi.nlm.nih.gov/24740528/)]
174. Yarnell S. The use of medicinal marijuana for posttraumatic stress disorder: a review of the current literature. *Prim Care Companion CNS Disord*. May 07, 2015;17(3):10.4088/PCC.15r01786. [FREE Full text] [doi: [10.4088/PCC.15r01786](https://doi.org/10.4088/PCC.15r01786)] [Medline: [26644963](https://pubmed.ncbi.nlm.nih.gov/26644963/)]
175. Niciu MJ, Shovestul BJ, Jaso BA, Farmer C, Luckenbaugh DA, Brutsche NE, et al. Features of dissociation differentially predict antidepressant response to ketamine in treatment-resistant depression. *J Affect Disord*. May 2018;232:310-315. [FREE Full text] [doi: [10.1016/j.jad.2018.02.049](https://doi.org/10.1016/j.jad.2018.02.049)] [Medline: [29501990](https://pubmed.ncbi.nlm.nih.gov/29501990/)]
176. Brecksema JJ, Niemeijer A, Kuin B, Veraart J, Vermetten E, Kamphuis J, et al. Phenomenology and therapeutic potential of patient experiences during oral esketamine treatment for treatment-resistant depression: an interpretative phenomenological study. *Psychopharmacology (Berl)*. Jul 2023;240(7):1547-1560. [FREE Full text] [doi: [10.1007/s00213-023-06388-6](https://doi.org/10.1007/s00213-023-06388-6)] [Medline: [37222753](https://pubmed.ncbi.nlm.nih.gov/37222753/)]
177. Mathai DS, Nayak SM, Yaden DB, Garcia-Romeu A. Reconsidering "dissociation" as a predictor of antidepressant efficacy for esketamine. *Psychopharmacology (Berl)*. Apr 2023;240(4):827-836. [doi: [10.1007/s00213-023-06324-8](https://doi.org/10.1007/s00213-023-06324-8)] [Medline: [36729145](https://pubmed.ncbi.nlm.nih.gov/36729145/)]
178. Barrett FS, Carbonaro TM, Hurwitz E, Johnson MW, Griffiths RR. Double-blind comparison of the two hallucinogens psilocybin and dextromethorphan: effects on cognition. *Psychopharmacology (Berl)*. Oct 2018;235(10):2915-2927. [FREE Full text] [doi: [10.1007/s00213-018-4981-x](https://doi.org/10.1007/s00213-018-4981-x)] [Medline: [30062577](https://pubmed.ncbi.nlm.nih.gov/30062577/)]
179. Garcia LS, Comim CM, Valvassori SS, Réus GZ, Barbosa LM, Andreazza AC, et al. Acute administration of ketamine induces antidepressant-like effects in the forced swimming test and increases BDNF levels in the rat hippocampus. *Prog Neuropsychopharmacol Biol Psychiatry*. Jan 01, 2008;32(1):140-144. [doi: [10.1016/j.pnpbp.2007.07.027](https://doi.org/10.1016/j.pnpbp.2007.07.027)] [Medline: [17884272](https://pubmed.ncbi.nlm.nih.gov/17884272/)]
180. Laruelle M, Abi-Dargham A, van Dyck CH, Gil R, D'Souza CD, Erdos J, et al. Single photon emission computerized tomography imaging of amphetamine-induced dopamine release in drug-free schizophrenic subjects. *Proc Natl Acad Sci U S A*. Aug 20, 1996;93(17):9235-9240. [FREE Full text] [doi: [10.1073/pnas.93.17.9235](https://doi.org/10.1073/pnas.93.17.9235)] [Medline: [8799184](https://pubmed.ncbi.nlm.nih.gov/8799184/)]
181. Chambers RA, Bremner JD, Moghaddam B, Southwick SM, Charney DS, Krystal JH. Glutamate and post-traumatic stress disorder: toward a psychobiology of dissociation. *Semin Clin Neuropsychiatry*. Oct 1999;4(4):274-281. [Medline: [10553033](https://pubmed.ncbi.nlm.nih.gov/10553033/)]
182. Maren S. Long-term potentiation in the amygdala: a mechanism for emotional learning and memory. *Trends Neurosci*. Dec 1999;22(12):561-567. [FREE Full text] [doi: [10.1016/s0166-2236\(99\)01465-4](https://doi.org/10.1016/s0166-2236(99)01465-4)] [Medline: [10542437](https://pubmed.ncbi.nlm.nih.gov/10542437/)]
183. Ori R, Amos T, Bergman H, Soares-Weiser K, Ipser JC, Stein DJ. Augmentation of cognitive and behavioural therapies (CBT) with d-cycloserine for anxiety and related disorders. *Cochrane Database Syst Rev*. May 10, 2015;2015(5):CD007803. [FREE Full text] [doi: [10.1002/14651858.CD007803.pub2](https://doi.org/10.1002/14651858.CD007803.pub2)] [Medline: [25957940](https://pubmed.ncbi.nlm.nih.gov/25957940/)]
184. Furukawa TA, Cipriani A, Cowen PJ, Leucht S, Egger M, Salanti G. Optimal dose of selective serotonin reuptake inhibitors, venlafaxine, and mirtazapine in major depression: a systematic review and dose-response meta-analysis. *Lancet Psychiatry*. Jul 2019;6(7):601-609. [FREE Full text] [doi: [10.1016/S2215-0366\(19\)30217-2](https://doi.org/10.1016/S2215-0366(19)30217-2)] [Medline: [31178367](https://pubmed.ncbi.nlm.nih.gov/31178367/)]
185. Thierrée S, Raulin-Briot M, Legrand M, Le Gouge A, Vancappel A, Tudorache AC, et al. Combining trauma script exposure with rTMS to reduce symptoms of post-traumatic stress disorder: randomized controlled trial. *Neuromodulation*. Jun 2022;25(4):549-557. [doi: [10.1111/ner.13505](https://doi.org/10.1111/ner.13505)] [Medline: [35667770](https://pubmed.ncbi.nlm.nih.gov/35667770/)]
186. Ferreri F, Mouchabac S, Sylvestre V, Millet B, El Hage W, Adrien V, et al. Repetitive transcranial magnetic stimulation (rTMS) in post-traumatic stress disorder: study protocol of a nationwide randomized controlled clinical trial of neuro-enhanced psychotherapy "TraumaStim". *Brain Sci*. Aug 31, 2023;13(9):1274. [FREE Full text] [doi: [10.3390/brainsci13091274](https://doi.org/10.3390/brainsci13091274)] [Medline: [37759875](https://pubmed.ncbi.nlm.nih.gov/37759875/)]
187. van 't Wout-Frank M, Shea MT, Larson VC, Greenberg BD, Philip NS. Combined transcranial direct current stimulation with virtual reality exposure for posttraumatic stress disorder: feasibility and pilot results. *Brain Stimul*. 2019;12(1):41-43. [FREE Full text] [doi: [10.1016/j.brs.2018.09.011](https://doi.org/10.1016/j.brs.2018.09.011)] [Medline: [30266416](https://pubmed.ncbi.nlm.nih.gov/30266416/)]
188. Han J, Choi KM, Yang C, Kim HS, Park SS, Lee SH. Treatment efficacy of tDCS and predictors of treatment response in patients with post-traumatic stress disorder. *J Affect Disord*. Dec 01, 2022;318:357-363. [doi: [10.1016/j.jad.2022.08.111](https://doi.org/10.1016/j.jad.2022.08.111)] [Medline: [36055537](https://pubmed.ncbi.nlm.nih.gov/36055537/)]
189. Abelson JL, Curtis GC, Sagher O, Albucher RC, Harrigan M, Taylor SF, et al. Deep brain stimulation for refractory obsessive-compulsive disorder. *Biol Psychiatry*. Mar 01, 2005;57(5):510-516. [doi: [10.1016/j.biopsych.2004.11.042](https://doi.org/10.1016/j.biopsych.2004.11.042)] [Medline: [15737666](https://pubmed.ncbi.nlm.nih.gov/15737666/)]
190. Noble LJ, Souza RR, McIntyre CK. Vagus nerve stimulation as a tool for enhancing extinction in exposure-based therapies. *Psychopharmacology (Berl)*. Jan 2019;236(1):355-367. [FREE Full text] [doi: [10.1007/s00213-018-4994-5](https://doi.org/10.1007/s00213-018-4994-5)] [Medline: [30091004](https://pubmed.ncbi.nlm.nih.gov/30091004/)]

191. Souza RR, Robertson NM, Pruitt DT, Gonzales PA, Hays SA, Rennaker RL, et al. Vagus nerve stimulation reverses the extinction impairments in a model of PTSD with prolonged and repeated trauma. *Stress*. Jul 2019;22(4):509-520. [doi: [10.1080/10253890.2019.1602604](https://doi.org/10.1080/10253890.2019.1602604)] [Medline: [31010369](https://pubmed.ncbi.nlm.nih.gov/31010369/)]
192. Ogden P, Pain C, Fisher J. A sensorimotor approach to the treatment of trauma and dissociation. *Psychiatr Clin North Am*. Mar 2006;29(1):263-79, xi. [doi: [10.1016/j.psc.2005.10.012](https://doi.org/10.1016/j.psc.2005.10.012)] [Medline: [16530597](https://pubmed.ncbi.nlm.nih.gov/16530597/)]
193. Ogden P, Fisher J. *Sensorimotor Psychotherapy: Interventions For Trauma And Attachment*. New York, NY. W. W. Norton & Company; Apr 27, 2015.
194. van Gelderen MJ, Nijdam MJ, Vermetten E. An innovative framework for delivering psychotherapy to patients with treatment-resistant posttraumatic stress disorder: rationale for interactive motion-assisted therapy. *Front Psychiatry*. May 04, 2018;9:176. [FREE Full text] [doi: [10.3389/fpsy.2018.00176](https://doi.org/10.3389/fpsy.2018.00176)] [Medline: [29780334](https://pubmed.ncbi.nlm.nih.gov/29780334/)]
195. Beaudoin MN. Agency and choice in the face of trauma: a narrative therapy map. *J Syst Ther*. Dec 2005;24(4):32-50. [FREE Full text] [doi: [10.1521/jsyt.2005.24.4.32](https://doi.org/10.1521/jsyt.2005.24.4.32)]
196. Rothbaum BO, Rizzo AS, Difede J. Virtual reality exposure therapy for combat-related posttraumatic stress disorder. *Ann N Y Acad Sci*. Oct 2010;1208:126-132. [doi: [10.1111/j.1749-6632.2010.05691.x](https://doi.org/10.1111/j.1749-6632.2010.05691.x)] [Medline: [20955334](https://pubmed.ncbi.nlm.nih.gov/20955334/)]
197. Webb AK, Vincent AL, Jin AB, Pollack MH. Physiological reactivity to nonideographic virtual reality stimuli in veterans with and without PTSD. *Brain Behav*. Feb 2015;5(2):e00304. [FREE Full text] [doi: [10.1002/brb3.304](https://doi.org/10.1002/brb3.304)] [Medline: [25642387](https://pubmed.ncbi.nlm.nih.gov/25642387/)]
198. van 't Wout M, Spofford CM, Unger WS, Sevin EB, Shea MT. Skin conductance reactivity to standardized virtual reality combat scenes in veterans with PTSD. *Appl Psychophysiol Biofeedback*. Sep 2017;42(3):209-221. [doi: [10.1007/s10484-017-9366-0](https://doi.org/10.1007/s10484-017-9366-0)] [Medline: [28646388](https://pubmed.ncbi.nlm.nih.gov/28646388/)]
199. Roy MJ, Costanzo ME, Jovanovic T, Leaman S, Taylor P, Norrholm SD, et al. Heart rate response to fear conditioning and virtual reality in subthreshold PTSD. *Stud Health Technol Inform*. 2013;191:115-119. [Medline: [23792855](https://pubmed.ncbi.nlm.nih.gov/23792855/)]
200. Deng W, Hu D, Xu S, Liu X, Zhao J, Chen Q, et al. The efficacy of virtual reality exposure therapy for PTSD symptoms: a systematic review and meta-analysis. *J Affect Disord*. Oct 01, 2019;257:698-709. [doi: [10.1016/j.jad.2019.07.086](https://doi.org/10.1016/j.jad.2019.07.086)] [Medline: [31382122](https://pubmed.ncbi.nlm.nih.gov/31382122/)]
201. Difede J, Cukor J, Jayasinghe N, Patt I, Jedel S, Spielman L, et al. Virtual reality exposure therapy for the treatment of posttraumatic stress disorder following September 11, 2001. *J Clin Psychiatry*. Nov 2007;68(11):1639-1647. [Medline: [18052556](https://pubmed.ncbi.nlm.nih.gov/18052556/)]
202. Norr AM, Smolenski DJ, Katz AC, Rizzo AA, Rothbaum BO, Difede J, et al. Virtual reality exposure versus prolonged exposure for PTSD: which treatment for whom? *Depress Anxiety*. Jun 2018;35(6):523-529. [doi: [10.1002/da.22751](https://doi.org/10.1002/da.22751)] [Medline: [29734488](https://pubmed.ncbi.nlm.nih.gov/29734488/)]
203. Difede J, Rothbaum BO, Rizzo AA, Wyka K, Spielman L, Reist C, et al. Enhancing exposure therapy for posttraumatic stress disorder (PTSD): a randomized clinical trial of virtual reality and imaginal exposure with a cognitive enhancer. *Transl Psychiatry*. Jul 27, 2022;12(1):299. [FREE Full text] [doi: [10.1038/s41398-022-02066-x](https://doi.org/10.1038/s41398-022-02066-x)] [Medline: [35896533](https://pubmed.ncbi.nlm.nih.gov/35896533/)]
204. Gomez MA, Skiba RM, Snow JC. Graspable objects grab attention more than images do. *Psychol Sci*. Feb 2018;29(2):206-218. [FREE Full text] [doi: [10.1177/0956797617730599](https://doi.org/10.1177/0956797617730599)] [Medline: [29215960](https://pubmed.ncbi.nlm.nih.gov/29215960/)]
205. Harris DJ, Buckingham G, Wilson MR, Vine SJ. Virtually the same? How impaired sensory information in virtual reality may disrupt vision for action. *Exp Brain Res*. Nov 2019;237(11):2761-2766. [FREE Full text] [doi: [10.1007/s00221-019-05642-8](https://doi.org/10.1007/s00221-019-05642-8)] [Medline: [31485708](https://pubmed.ncbi.nlm.nih.gov/31485708/)]
206. El Ali A, Ney R, van Berlo ZM, Cesar P. Is that my heartbeat? Measuring and understanding modality-dependent cardiac interoception in virtual reality. *IEEE Trans Vis Comput Graph*. Nov 2023;29(11):4805-4815. [doi: [10.1109/TVCG.2023.3320228](https://doi.org/10.1109/TVCG.2023.3320228)] [Medline: [37782606](https://pubmed.ncbi.nlm.nih.gov/37782606/)]
207. Kilteni K, Bergstrom I, Slater M. Drumming in immersive virtual reality: the body shapes the way we play. *IEEE Trans Vis Comput Graph*. Apr 2013;19(4):597-605. [FREE Full text] [doi: [10.1109/TVCG.2013.29](https://doi.org/10.1109/TVCG.2013.29)] [Medline: [23428444](https://pubmed.ncbi.nlm.nih.gov/23428444/)]
208. Davis S, Nesbitt K, Nalivaiko E. Comparing the onset of cybersickness using the Oculus Rift and two virtual roller coasters. In: *Proceedings of the 11th Australasian Conference on Interactive Entertainment*. 2015. Presented at: IE 2015; January 27-30, 2015; Sydney, Australia.
209. Coulter S. Creating safety for trauma survivors: what can therapists do? *Child Care Pract*. Jan 17, 2008;7(1):45-56. [doi: [10.1080/13575270108413233](https://doi.org/10.1080/13575270108413233)]
210. Ross N, Brown C, Johnstone M. Beyond medicalized approaches to violence and trauma: empowering social work practice. *J Soc Work*. Jan 09, 2023;23(3):567-585. [doi: [10.1177/14680173221144557](https://doi.org/10.1177/14680173221144557)]
211. Botella C, Serrano B, Baños RM, Garcia-Palacios A. Virtual reality exposure-based therapy for the treatment of post-traumatic stress disorder: a review of its efficacy, the adequacy of the treatment protocol, and its acceptability. *Neuropsychiatr Dis Treat*. Oct 3, 2015;11:2533-2545. [FREE Full text] [doi: [10.2147/NDT.S89542](https://doi.org/10.2147/NDT.S89542)] [Medline: [26491332](https://pubmed.ncbi.nlm.nih.gov/26491332/)]
212. Schleyer W, Zona K, Quigley D, Spottswood M. Group therapy in primary care settings for the treatment of posttraumatic stress disorder: a systematic literature review. *Gen Hosp Psychiatry*. 2022;77:1-10. [doi: [10.1016/j.genhosppsy.2022.03.010](https://doi.org/10.1016/j.genhosppsy.2022.03.010)] [Medline: [35390567](https://pubmed.ncbi.nlm.nih.gov/35390567/)]
213. Hunt A, Wanderley MM, Paradis M. The importance of parameter mapping in electronic instrument design. *J New Music Res*. Dec 1, 2003;32(4):429-440. [doi: [10.1076/jnmr.32.4.429.18853](https://doi.org/10.1076/jnmr.32.4.429.18853)]



214. Thompson WF, Schlaug G. The healing power of music. *Sci Am Mind*. Mar 2015;26(2):32-41. [doi: [10.1038/scientificamericanmind0315-32](https://doi.org/10.1038/scientificamericanmind0315-32)]
215. Koch SC, Riege RF, Tisborn K, Biondo J, Martin L, Beelmann A. Effects of dance movement therapy and dance on health-related psychological outcomes. A meta-analysis update. *Front Psychol*. Aug 20, 2019;10:1806. [FREE Full text] [doi: [10.3389/fpsyg.2019.01806](https://doi.org/10.3389/fpsyg.2019.01806)] [Medline: [31481910](https://pubmed.ncbi.nlm.nih.gov/31481910/)]
216. Rossman ML. *Guided Imagery for Self-Healing: An Essential Resource for Anyone Seeking Wellness*. Tiburon, CA. HJ Kramer Incorporated; 2000.
217. Özü Ö. Guided imagery as a psychotherapeutic mind-body intervention in health psychology: a brief review of efficacy research. *Eur J Psychol*. Nov 29, 2010;6(4). [doi: [10.5964/ejop.v6i4.232](https://doi.org/10.5964/ejop.v6i4.232)]
218. Apóstolo JL, Kolcaba K. The effects of guided imagery on comfort, depression, anxiety, and stress of psychiatric inpatients with depressive disorders. *Arch Psychiatr Nurs*. Dec 2009;23(6):403-411. [doi: [10.1016/j.apnu.2008.12.003](https://doi.org/10.1016/j.apnu.2008.12.003)] [Medline: [19926022](https://pubmed.ncbi.nlm.nih.gov/19926022/)]
219. Lewandowski W, Good M, Draucker CB. Changes in the meaning of pain with the use of guided imagery. *Pain Manag Nurs*. Jun 2005;6(2):58-67. [doi: [10.1016/j.pmn.2005.01.002](https://doi.org/10.1016/j.pmn.2005.01.002)] [Medline: [15970919](https://pubmed.ncbi.nlm.nih.gov/15970919/)]
220. Strauss J, Calhoun P, Marx C. Guided imagery as a therapeutic tool in post-traumatic stress disorder. In: LeDoux J, Keane T, Shiromani P, editors. *Post-Traumatic Stress Disorder*. Totowa, NJ. Humana Press; 2009.
221. Kwekkeboom K, Huseby-Moore K, Ward S. Imaging ability and effective use of guided imagery. *Res Nurs Health*. Jun 1998;21(3):189-198. [doi: [10.1002/\(sici\)1098-240x\(199806\)21:3<189::aid-nur2>3.0.co;2-d](https://doi.org/10.1002/(sici)1098-240x(199806)21:3<189::aid-nur2>3.0.co;2-d)] [Medline: [9609504](https://pubmed.ncbi.nlm.nih.gov/9609504/)]
222. Neuner F. Safety first? Trauma exposure in PTSD. In: Neudeck P, Wittchen HU, editors. *Exposure Therapy*. New York, NY. Springer; 2012.
223. Yehuda R, Bierer LM. The relevance of epigenetics to PTSD: implications for the DSM-V. *J Trauma Stress*. Oct 2009;22(5):427-434. [FREE Full text] [doi: [10.1002/jts.20448](https://doi.org/10.1002/jts.20448)] [Medline: [19813242](https://pubmed.ncbi.nlm.nih.gov/19813242/)]
224. Yehuda R, Daskalakis NP, Lehrner A, Desarnaud F, Bader HN, Makotkine I, et al. Influences of maternal and paternal PTSD on epigenetic regulation of the glucocorticoid receptor gene in Holocaust survivor offspring. *Am J Psychiatry*. Aug 2014;171(8):872-880. [FREE Full text] [doi: [10.1176/appi.ajp.2014.13121571](https://doi.org/10.1176/appi.ajp.2014.13121571)] [Medline: [24832930](https://pubmed.ncbi.nlm.nih.gov/24832930/)]
225. Yehuda R, Daskalakis NP, Bierer LM, Bader HN, Klengel T, Holsboer F, et al. Holocaust exposure induced intergenerational effects on FKBP5 methylation. *Biol Psychiatry*. Sep 01, 2016;80(5):372-380. [doi: [10.1016/j.biopsych.2015.08.005](https://doi.org/10.1016/j.biopsych.2015.08.005)] [Medline: [26410355](https://pubmed.ncbi.nlm.nih.gov/26410355/)]
226. Yehuda R, Lehrner A. Intergenerational transmission of trauma effects: putative role of epigenetic mechanisms. *World Psychiatry*. Oct 2018;17(3):243-257. [doi: [10.1002/wps.20568](https://doi.org/10.1002/wps.20568)] [Medline: [30192087](https://pubmed.ncbi.nlm.nih.gov/30192087/)]
227. Javidi H, Yadollahie M. Post-traumatic stress disorder. *Int J Occup Environ Med*. Jan 2012;3(1):2-9. [Medline: [23022845](https://pubmed.ncbi.nlm.nih.gov/23022845/)]
228. Fillion JS, Clements PT, Averill JB, Vigil GJ. Talking as a primary method of peer defusing for military personnel exposed to combat trauma. *J Psychosoc Nurs Ment Health Serv*. Aug 2002;40(8):40-49. [doi: [10.3928/0279-3695-20020801-15](https://doi.org/10.3928/0279-3695-20020801-15)] [Medline: [12174514](https://pubmed.ncbi.nlm.nih.gov/12174514/)]
229. Stileman HM, Jones CA. Revisiting the debriefing debate: does psychological debriefing reduce PTSD symptomology following work-related trauma? A meta-analysis. *Front Psychol*. Dec 21, 2023;14:1248924. [FREE Full text] [doi: [10.3389/fpsyg.2023.1248924](https://doi.org/10.3389/fpsyg.2023.1248924)] [Medline: [38204890](https://pubmed.ncbi.nlm.nih.gov/38204890/)]
230. Wessely S, Rose S, Bisson J. Brief psychological interventions ("debriefing") for trauma-related symptoms and the prevention of post traumatic stress disorder. *Cochrane Database Syst Rev*. 2000;2002(2):CD000560. [FREE Full text] [doi: [10.1002/14651858.CD000560](https://doi.org/10.1002/14651858.CD000560)] [Medline: [10796724](https://pubmed.ncbi.nlm.nih.gov/10796724/)]
231. Bolwig TG. Debriefing after psychological trauma. *Acta Psychiatr Scand*. Sep 1998;98(3):169-170. [doi: [10.1111/j.1600-0447.1998.tb10062.x](https://doi.org/10.1111/j.1600-0447.1998.tb10062.x)] [Medline: [9761401](https://pubmed.ncbi.nlm.nih.gov/9761401/)]
232. Sensky T. The utility of systematic reviews: the case of psychological debriefing after trauma. *Psychother Psychosom*. 2003;72(4):171-175. [doi: [10.1159/000070780](https://doi.org/10.1159/000070780)] [Medline: [12817571](https://pubmed.ncbi.nlm.nih.gov/12817571/)]
233. Rose S, Bisson J, Wessely S. A systematic review of single-session psychological interventions ('debriefing') following trauma. *Psychother Psychosom*. 2003;72(4):176-184. [doi: [10.1159/000070781](https://doi.org/10.1159/000070781)] [Medline: [12792122](https://pubmed.ncbi.nlm.nih.gov/12792122/)]
234. van Emmerik AA, Kamphuis JH, Hulsbosch AM, Emmelkamp PM. Single session debriefing after psychological trauma: a meta-analysis. *Lancet*. Sep 07, 2002;360(9335):766-771. [doi: [10.1016/S0140-6736\(02\)09897-5](https://doi.org/10.1016/S0140-6736(02)09897-5)] [Medline: [12241834](https://pubmed.ncbi.nlm.nih.gov/12241834/)]
235. Richins MT, Gauntlett L, Tehrani N, Hesketh I, Weston D, Carter H, et al. Early post-trauma interventions in organizations: a scoping review. *Front Psychol*. Jun 25, 2020;11:1176. [FREE Full text] [doi: [10.3389/fpsyg.2020.01176](https://doi.org/10.3389/fpsyg.2020.01176)] [Medline: [32670143](https://pubmed.ncbi.nlm.nih.gov/32670143/)]
236. Levine B, Land HM. A meta-synthesis of qualitative findings about dance/movement therapy for individuals with trauma. *Qual Health Res*. Feb 2016;26(3):330-344. [doi: [10.1177/1049732315589920](https://doi.org/10.1177/1049732315589920)] [Medline: [26063604](https://pubmed.ncbi.nlm.nih.gov/26063604/)]
237. Grasser LR, Al-Saghir H, Wanna C, Spinei J, Javanbakht A. Moving through the trauma: dance/movement therapy as a somatic-based intervention for addressing trauma and stress among Syrian refugee children. *J Am Acad Child Adolesc Psychiatry*. Nov 2019;58(11):1124-1126. [doi: [10.1016/j.jaac.2019.07.007](https://doi.org/10.1016/j.jaac.2019.07.007)] [Medline: [31348987](https://pubmed.ncbi.nlm.nih.gov/31348987/)]
238. Bion WR. A theory of thinking. In: Spillius EB, editor. *Melanie Klein Today: Developments in Theory and Practice, Volume 1*. Milton Park, United Kingdom. Taylor & Francis Group; 1988:178-186.



239. Stevens JS, Jovanovic T. Role of social cognition in post-traumatic stress disorder: a review and meta-analysis. *Genes Brain Behav.* Jan 2019;18(1):e12518. [FREE Full text] [doi: [10.1111/gbb.12518](https://doi.org/10.1111/gbb.12518)] [Medline: [30221467](https://pubmed.ncbi.nlm.nih.gov/30221467/)]
240. Tibi-Elhanany Y, Shamay-Tsoory SG. Social cognition in social anxiety: first evidence for increased empathic abilities. *Isr J Psychiatry Relat Sci.* 2011;48(2):98-106. [FREE Full text] [Medline: [22120444](https://pubmed.ncbi.nlm.nih.gov/22120444/)]
241. Elmi LM, Clapp JD. Interpersonal functioning and trauma: the role of empathy in moderating the association of PTSD and interpersonal functioning. *Behav Ther.* Sep 2021;52(5):1251-1264. [doi: [10.1016/j.beth.2021.02.004](https://doi.org/10.1016/j.beth.2021.02.004)] [Medline: [34452677](https://pubmed.ncbi.nlm.nih.gov/34452677/)]
242. Maercker A, Horn AB. A socio-interpersonal perspective on PTSD: the case for environments and interpersonal processes. *Clin Psychol Psychother.* 2013;20(6):465-481. [doi: [10.1002/cpp.1805](https://doi.org/10.1002/cpp.1805)] [Medline: [22730216](https://pubmed.ncbi.nlm.nih.gov/22730216/)]
243. Mitima-Verloop HB, Boelen PA, Mooren TT. Commemoration of disruptive events: a scoping review about posttraumatic stress reactions and related factors. *Eur J Psychotraumatol.* Jan 13, 2020;11(1):1701226. [FREE Full text] [doi: [10.1080/20008198.2019.1701226](https://doi.org/10.1080/20008198.2019.1701226)] [Medline: [32082507](https://pubmed.ncbi.nlm.nih.gov/32082507/)]
244. Gene-Cos N, Fisher J, Ogden P, Cantrell A. Sensorimotor psychotherapy group therapy in the treatment of complex PTSD. *Ann Psychiatr Ment Health.* 2016;4(6):1080. [FREE Full text]
245. Classen CC, Hughes L, Clark C, Hill Mohammed B, Woods P, Beckett B. A pilot RCT of a body-oriented group therapy for complex trauma survivors: an adaptation of sensorimotor psychotherapy. *J Trauma Dissociation.* 2021;22(1):52-68. [doi: [10.1080/15299732.2020.1760173](https://doi.org/10.1080/15299732.2020.1760173)] [Medline: [32419670](https://pubmed.ncbi.nlm.nih.gov/32419670/)]
246. Sadeghi S, Schmidt SN, Mier D, Hass J. Effective connectivity of the human mirror neuron system during social cognition. *Soc Cogn Affect Neurosci.* Aug 01, 2022;17(8):732-743. [FREE Full text] [doi: [10.1093/scan/nsab138](https://doi.org/10.1093/scan/nsab138)] [Medline: [35086135](https://pubmed.ncbi.nlm.nih.gov/35086135/)]

## Abbreviations

- ACC:** anterior cingulate cortex  
**CBT:** cognitive behavioral therapy  
**C-PTSD:** complex posttraumatic stress disorder  
**DMN:** default mode network  
**DMT:** dance movement therapy  
**D-PTSD:** dissociative posttraumatic stress disorder  
**GI:** guided imagery  
**GS:** gesture sonification  
**MDMA:** 3,4-Methylenedioxymethamphetamine  
**NMDA:** N-methyl-D-aspartate  
**PFC:** prefrontal cortex  
**PTSD:** posttraumatic stress disorder  
**RHI:** rubber hand illusion  
**SA:** sense of agency  
**SN:** salience network  
**SO:** sense of body ownership  
**TE:** traumatic event  
**VR:** virtual reality  
**VRET:** virtual reality exposure therapy

*Edited by A Mavragani; submitted 14.03.24; peer-reviewed by S Gallagher, J Mistry; comments to author 03.04.24; revised version received 17.04.24; accepted 02.05.24; published 01.07.24*

### *Please cite as:*

*Adrien V, Bosc N, Peccia Galletto C, Diot T, Claverie D, Reggente N, Trousselard M, Bui E, Baubet T, Schoeller F  
Enhancing Agency in Posttraumatic Stress Disorder Therapies Through Sensorimotor Technologies*

*J Med Internet Res* 2024;26:e58390

URL: <https://www.jmir.org/2024/1/e58390>

doi: [10.2196/58390](https://doi.org/10.2196/58390)

PMID: [38742989](https://pubmed.ncbi.nlm.nih.gov/38742989/)

©Vladimir Adrien, Nicolas Bosc, Claire Peccia Galletto, Thomas Diot, Damien Claverie, Nicco Reggente, Marion Trousselard, Eric Bui, Thierry Baubet, Félix Schoeller. Originally published in the Journal of Medical Internet Research (<https://www.jmir.org>), 01.07.2024. This is an open-access article distributed under the terms of the Creative Commons Attribution License (<https://creativecommons.org/licenses/by/4.0/>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work, first published in the Journal of Medical Internet Research (ISSN 1438-8871), is properly cited. The

complete bibliographic information, a link to the original publication on <https://www.jmir.org/>, as well as this copyright and license information must be included.