

A Computational Agent Model for Post-Traumatic Stress Disorders

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Abstract. In this paper a computational agent model is presented that describes and mimics processing in persons with a Post-Traumatic Stress Disorder (PTSD). The model is based on insights from the neurological literature on how specific phenomena that are typical for PTSD patients can occur, such as re-experiencing the strong feeling related to the original traumatic event, dissociation (not feeling the own body), and flashbacks in the form of images. A number of simulations is presented that show how the agent model displays these phenomena of re-experiencing, dissociation and flashback episodes, triggered by a neutral stimulus. The obtained cognitive/affective agent model can be used as a basis for the design of human-like virtual agents in simulation-based training or in gaming or virtual stories.

Keywords: Post-Traumatic Stress Disorder (PTSD), computational agent model

Introduction

A Post-Traumatic Stress Disorder (PTSD) may occur when a person undergoes a traumatic event involving strong emotions and/or physical harm (for example, sexual abuse, a battlefield experience, or a car accident). Recent studies in neuroscience show that a number of mechanisms play a role in patients suffering from PTSD. The main types of patients are classified according to the symptoms they have when a stimulus occurs related to the traumatic memory. Two primary symptoms are re-experiencing (flashbacks) and dissociation. Re-experiencing happens when patients undergo a strong emotional feeling similar to the feeling experienced during the traumatic event. It is usually accompanied by visual flashbacks and physical inconvenience. Dissociative patients undergo an emotional withdrawal (due to the emotional load triggered by a stimulus) that involves loss of body perception or so-called out-of-body experience.

The computational agent model presented here is based on neurological studies of PTSD patients, among others using imagery technology, and serves to simulate the disorder's symptoms from inside (i.e., embodied perspective). In line with the recent findings, the presented model reflects the understanding of brain functions and reactions observed in reality. Indeed, the literature shows different steps to reach a reaction, for example, by representing a stimulus (which by itself may be neutral, but has some association to the traumatic event), automatic preparation of response, and possible control over the internal processes. The control process plays an important role

in inhibition of over-reacting to such stimuli. It acts over the emotional involvement triggered by the stimulus and memory of the traumatic event and the bodily response to the emotional load.

Application of such a cognitive/affective agent model can be found in the context of human-like virtual agents in simulation-based training, gaming or virtual stories. For example, a virtual patient for a simulation-based training environment for psychiatrists or psycho-therapists can be developed based on the model.

Section 1 briefly discusses the neurobiological background of the impairment. In Section 2 the detailed computational agent model is introduced. Section 3 illustrates different simulation scenarios and their outcomes. Section 4 is a discussion.

1. Neurological Background

Recent neurological studies on PTSD have focused on analysis of the default network activation and connectivity during trauma-related processes. This network is an anatomically interconnected brain system that activates when individuals focus on internal tasks such as daydreaming, envisioning the future, retrieving memories and gauging others' perspectives. It includes part of the medial temporal lobe for memory, part of the medial prefrontal cortex for theory of mind and the posterior cingulate cortex for integration, along with the adjacent precuneus and the medial, lateral and inferior parietal cortex; cf. [4]. This network undergoes developmental changes along with experiences.

Among persons experiencing PTSD, lower activation was found in the posterior cingulate gyrus compared to controls; cf. [14]. The posterior cingulate gyrus discerns emotional and self-relevant information. It interacts on one hand with the anterior cingulate gyrus, which integrates emotional information with cognition, and on the other hand with the medial prefrontal cortex, which allows for self-reflection and the regulation of emotion and arousal.

Impairment in this network appears to correlate with the experience of persons who have long-term trauma and describe feeling 'dead inside' or have a fragmented sense of self or enter dissociative states, as put forward in [1]. Higher activation levels in neural networks involved in representing body states was seen in dissociated PTSD [13].

Hyper-sensibility and hyper-vigilance are central characteristics of PTSD in terms of increased likelihood of emotional response to environmental stimuli due to altered connectivity between default network and the amygdala, hippocampus and insula. Dissociation may also involve alterations in the relation between the default network and subregions serving cognitive abilities. Thus, awareness of emotional stimulation plays a role in the strength of the symptoms. Indeed in [11, 12], it is suggested that it may engage top-down reflexive or effortful emotion regulation that, from [13], seems to be impaired in PTSD.

PTSD subjects are unable to control the intensity of their emotional reactions in the presence of stimuli that function as reminders of the traumatic experience. Studies cited in [10] have shown activation disturbances of the PCC (Posterior Cingulate Cortex) which is involved in episodic memory retrieval and pain processing.

Flashback patients (heightened autonomic and emotional reactivity) have reduced bilateral medial frontal cortex and ACC (Anterior Cingulate Cortex) activity. In contrast, dissociative patients have increased activation in frontal, superior and medial temporal gyrus, inferior frontal and parietal regions of the right hemisphere, and have a

lack of amygdala response to trauma-related neutral stimuli. This leads to the hypothesis that the heightened prefrontal activity in dissociative PTSD reflects stronger emotional regulation and inhibition of limbic emotional networks, including the amygdala. Thus in [9] it is concluded that dissociation is a strategic and controlled regulatory process invoked by extreme arousal to reduce the experience of aversive emotions. This same study shows that thalamic activity is increased in dissociation, which supports the theory that a higher sensory transmission mediates bottom-up excitatory processes. This is also claimed by Oathes et al. [17] who show that dissociative patients show faster emotion labeling.

In [13], the processes in PTSD subjects are related to Damasio (1999)'s theory on emotions and experiencing them. In the study described in [13] indeed such altered bodily perceptions and emotions were found in PTSD subjects:

'Damasio (1999) has emphasized the role of the insula and the somatosensory cortices in processing signals regarding bodily state and suggests that these signals form the basis for emotions. (...) Thus, the insula activation seen in this study might reflect this altered perception or possibly alterations in the "body map" constructed by the insula, which has been hypothesized by Damasio (1999) to contribute to emotional experiences. (...) In fact, the subjective reports of the dissociated PTSD subjects in this study suggest that they experienced alterations in both bodily perceptions and emotions during recall of the traumatic memory. It is interesting to note that patients in a dissociative state often have difficulties with perception of internal bodily states, for example recognizing pain states. (...) Moreover, patients in dissociative states often have significant difficulties experiencing feelings of emotion. In fact, all of the dissociative subjects in this study reported being "removed" from their experience of their traumatic memory.' ([13], p. 881)

As a summary, it has been shown in the recent literature that PTSD patients suffer from an impaired emotion regulation process combined with a higher sensitivity to emotional stimuli. There exists two ways of dealing with a memory recall of a traumatic event, each patient usually reacts automatically with only one of these responses. Flashback patients are over-reacting and fall into a strong re-experience of the trauma accompanied with visual recall. Dissociative patients react to traumatic emotion recalls by suppressing body and emotional affects and appraisals.

2. Description of the Computational Model

The computational agent model uses sensory representation states for external stimuli and body states, and preparation states for emotional responses and regulation actions to turn away from stimuli that lead to high, disturbing levels of arousal; for an overview, see Fig. 1. Moreover, a control state is used that detects disturbing levels of arousal, and in turn can activate suppressing or regulating processes. In line with [6] and [13], it is assumed that emotional response preparations affect sensory representations of related body states (body maps) as in [2], both by an internal as-if body loop and an external body loop. These body maps are considered the basis of feeling the emotion. Moreover, it is assumed that this feeling in turn has a strengthening effect on the emotional preparation state, so that a cyclic process occurs, in line with [8]:

'The brain has a direct means to respond to the object as feelings unfold because the object at the origin is inside the body, rather than external to it. The brain can act directly on the very object it is perceiving. It can do so by modifying the state of the object, or by altering the transmission of signals from it. The object at the origin on the one hand, and the brain map of that object on the other, can influence each other in a sort of reverberative process that is not to be found, for example, in the perception of an external object. (...) after an occasion of such feelings begins – for seconds or for

minutes – there is a dynamic engagement of the body, almost certainly in a repeated fashion, and a subsequent dynamic variation of the perception. We perceive a series of transitions. We sense an interplay, a give and take.’ ([7], pp. 91-92)

In Fig. 1, s_2 is the stimulus that caused the traumatic experience, and s_1 is a more neutral stimulus that has some association to the situation in which the trauma was caused. For example, s_2 is the visual image of a fire while being inside a burning house, while s_1 is the image of the house from outside while it is not burning. Moreover, the emotional response and feeling is assumed to relate to the preparation and sensory representation of a body state indicated by b . In Table 1 an overview is given of the states used in the model. The connections between the states have certain strengths, as indicated in Table 2. It is assumed that substantial differences exist between these connection strengths for healthy subjects and PTSD subjects. For example, the strengths of the connections ω_5 and ω_{18} from the sensory representation of stimulus s_1 are low or zero in healthy subjects: in principle s_1 is an emotion-neutral stimulus (for example, seeing a house).

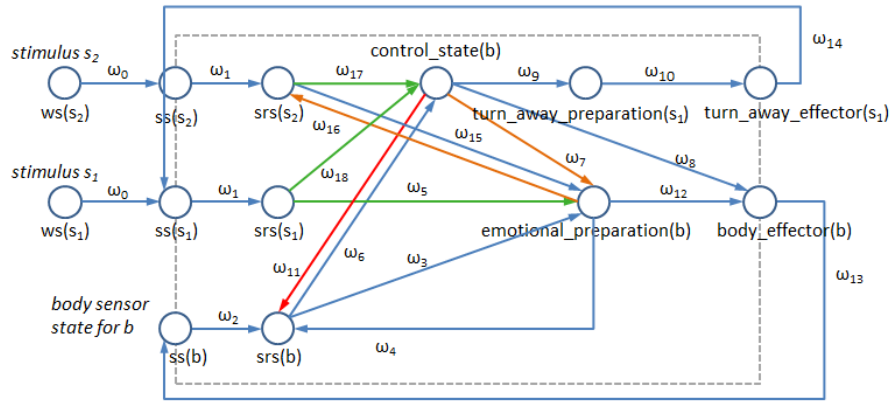


Figure 1. Overview of the computational agent model; colored links are those that differ between PTSD patients and healthy subjects (mainly, red for dissociation, orange for flashbacks and green for both); the grey dashed line represent the boundary between internal and external states. States on the dashed line are intermediate states which are seen as sensors $ss(x)$. Sensory representations $srs(x)$ are the internal representations of sensory information.

Table 1. Overview of the states used

notation	explanation
$ws(W)$	World state for W (stimulus s_1 or s_2)
$ss(W)$	Sensor state for W (stimulus s_1 , stimulus s_2 , or body state b)
$srs(W)$	Sensory representation state for W (stimulus s_1 , stimulus s_2 , or body state b)
$ep(b)$	Emotional response preparation state for b
$be(b)$	Body effector state for b
$cs(b)$	Control state for b
$tap(s_1)$	Turn away preparation state for s_1
$tae(s_1)$	Turn away effector state for s_1

However, they are assumed higher in PTSD subjects because this neutral stimulus is associated to a traumatic experience (e.g., they have experienced a fire in their house that looked similar, which had fatal repercussions). This is supported by [1] and [8]:

‘Hyper-sensibility and hyper-vigilance are central characteristics of PTSD in terms of increased likelihood of emotional response to environmental stimuli due to altered connectivity between default network and the amygdala, hippocampus and insula’ ([1], p. 192).
‘(PTSD patients) are unable to control the intensity of their emotional reactions in the presence of reminders of the traumatic experience’ ([8], p. 112).

Moreover, the strength of the connection ω_4 from emotional preparation $ep(b)$ to feeling the emotional arousal $srs(b)$ is assumed high in PTSD subjects:

‘Greater activation levels in neural networks involved in representing bodily states was seen in dissociated PTSD’ ([13], p. 873).
‘(...) there is evidence of greater activity in nonverbal and somatosensory processes in response to trauma scripts in dissociative PTSD’ ([13], p. 878).
‘Enhanced early sensory registration, somatosensory arousal and motor readiness that is consistent with this enhanced automatic arousal. (seen in PTSD)’ ([9], p. 1776).

Furthermore, for dissociative PTSD subjects ω_{11} is a strong inhibitory connection that makes the agent loose the feeling of his or her body state.

‘Neural correlates are consistent with a “super suppression” or robust inhibition of affective arousal during dissociation on the part of these individuals with PTSD’ ([10], p.114).
‘PTSD dissociative symptoms are regarded as being the consequence of an enhanced suppression of fear-induced arousal’ ([10], p. 121).
‘(...) dissociation is a strategic and controlled regulatory process invoked by extreme arousal to reduce the experience of aversive emotions’ ([9], p. 1776).

Also the strength of the connection ω_7 is assumed to be abnormal in Dissociative PTSD subjects, for example weighting -0.7 for this type of PTSD subject, instead of -0.4 in a healthy subject. This displays the lack of normal emotional control in dissociative PTSD: the connection do not inhibit $ep(b)$ in an appropriate manner as happens in healthy subjects. Finally, ω_{16} is a connection that is assumed to have some strength in PTSD patients, and is especially strong in those having visual flashbacks; it makes the person calling back the traumatic images he or she has undergone.

The agent model has been computationally formalised using the hybrid modeling language LEADSTO and its software environment; cf. [3]. Within LEADSTO a temporal causal relation or dynamic temporally local property (LP) $a \rightarrow b$ denotes that when a state property a (or conjunction thereof) occurs, then after a certain time delay, state property b will occur. This delay will be taken as a uniform time step Δt .

Table 2. Overview of connections and weights

From state	To state	Weights	LP
$ss(s_1)$	$srs(s_1)$	ω_1	LP0
$ep(b), ss(s_2)$	$srs(s_2)$	ω_{16}, ω_1	LP1
$ss(b), ep(b), cs(b)$	$srs(b)$	$\omega_2, \omega_4, \omega_{11}$	LP2
$srs(b), srs(s_2), srs(s_1)$	$cs(b)$	$\omega_6, \omega_{17}, \omega_{18}$	LP3
$srs(s_1), srs(s_2), srs(b), cs(b)$	$ep(b)$	$\omega_5, \omega_{15}, \omega_3, \omega_7$	LP4
$cs(b)$	$tap(s_1)$	ω_9	LP5
$ep(b), cs(b)$	$be(b)$	ω_{12}, ω_8	LP6
$tap(s_1)$	$tac(s_1)$	ω_{10}	LP7
$be(b)$	$ss(b)$	ω_{13}	LP8
$ws(s_1), tac(s_1)$	$ss(s_1)$	ω_0, ω_{14}	LP9
$ws(s_2)$	$ss(s_2)$	ω_0	LP10

Below, the dynamics following the connections between the states in Fig. 1 are described in more detail. This is done for each state by a dynamic local property

specifying how the activation value for this state is updated (after a time step of Δt) based on the activation values of the states connected to it (the incoming arrows in Fig. 1). In these update specifications for each node a (combination) function f is used, which in principle can be any function mapping the vector of input obtained from other nodes into the interval $[0, 1]$. In the simulations discussed in Section 3, the identity function $f(X) = X$ is used for LP8 and LP10, and the sum function $f(X, Y) = X + Y$ for LP9. For the other dynamic properties, f is defined as follows:

$$f(X_1, \dots, X_k) = th(\sigma, \tau, X_1 + \dots + X_k) \\ \text{with } th(\sigma, \tau, W) = [1 / (1 + e^{-\sigma(W - \tau)}) - 1 / (1 + e^{\sigma\tau})] / (1 + e^{-\sigma\tau})$$

a logistic threshold function, where σ is the steepness and τ is the threshold; this function f is applied to properties LP0 to LP7. Parameter γ is an update speed factor. First the generation of sensory representations for stimuli s_1 and s_2 are described by LP0 and LP1, respectively.

LP0 Sensory representation of stimulus s_1

If stimulus s_1 is sensed with level V_1
 and the sensory representation of s_1 has level V_2
 then after duration Δt the sensory representation of s_1 will have level $V_2 + \gamma[f(\omega_1 V_1) - V_2] \Delta t$.
 $ss(s_1, V_1) \& srs(s_1, V_2) \rightarrow srs(s_1, V_2 + \gamma[f(\omega_1 V_1) - V_2] \Delta t)$

LP1 Sensory representation of stimulus s_2

If the emotional preparation of B has level V_1
 and stimulus s_2 is sensed with level V_2
 and the sensory representation of s_2 has level V_3
 then after duration Δt the sensory representation of s_2
 will have level $V_3 + \gamma[f(\omega_{16} V_1, \omega_1 V_2) - V_3] \Delta t$.
 $ep(B, V_1) \& ss(s_2, V_2) \& srs(s_2, V_3) \rightarrow srs(s_2, V_3 + \gamma[f(\omega_{16} V_1, \omega_1 V_2) - V_3] \Delta t)$

In LP2 it is described how the sensory representation of a body state is maintained. Note that here the suppressing effect of the control state is also incorporated.

LP2 Sensory representation of a body state

If body state B is sensed with level V_1
 and the emotional preparation for B has level V_2
 and the control state for B has level V_3
 and the sensory representation of B has level V_4
 then after duration Δt the sensory representation of B
 will have level $V_4 + \gamma[f(\omega_2 V_1, \omega_4 V_2, \omega_{11} V_3) - V_4] \Delta t$.
 $ss(B, V_1) \& ep(B, V_2) \& cs(B, V_3) \& srs(B, V_4) \rightarrow srs(B, V_4 + \gamma[f(\omega_2 V_1, \omega_4 V_2, \omega_{11} V_3) - V_4] \Delta t)$

The control state is generated by LP3, based on the sensory representation of b (feeling the emotion); also the considered stimuli are involved.

LP3 Control state for a sensory representation of a body state

If the sensory representation of b has level V_1
 and the sensory representation of s_2 has level V_2
 and the sensory representation of s_1 has level V_3
 and the control state for b has level V_4
 then after Δt the control state for b will have level $V_4 + \gamma[f(\omega_6 V_1, \omega_{17} V_2, \omega_{18} V_3) - V_4] \Delta t$.
 $srs(b, V_1) \& srs(s_2, V_2) \& srs(s_1, V_3) \& cs(b, V_4) \rightarrow cs(b, V_4 + \gamma[f(\omega_6 V_1, \omega_{17} V_2, \omega_{18} V_3) - V_4] \Delta t)$

In LP4 it the preparation for an emotional response is described, depending on stimuli and the feeling. Here also a suppressing effect of the control state is incorporated.

LP4 Emotional preparation for a body state

If the sensory representation of b has level V_1
 and the sensory representation of s_1 has level V_2
 and the control state of b has level V_3
 and the sensory representation of s_2 has level V_4
 and the emotional preparation for b has level V_5
 then after duration Δt the emotional preparation for b
 will have level $V_5 + \gamma [f(\omega_3 V_1, \omega_5 V_2, \omega_7 V_3, \omega_{15} V_4) - V_5] \Delta t$.
 $\text{srs}(b, V_1) \& \text{srs}(s_1, V_2) \& \text{cs}(b, V_3) \& \text{srs}(s_2, V_4) \& \text{ep}(b, V_5)$
 $\rightarrow \text{ep}(b, V_5 + \gamma [f(\omega_3 V_1, \omega_5 V_2, \omega_7 V_3, \omega_{15} V_4) - V_5] \Delta t)$

Antecedent-focused regulation emotion regulation (cf. [11, 12]) has been modelled in LP5 by a ‘turn-away’ action to avoid the stimulus, based on the control state.

LP5 Turn-away preparation

If the control state for b has level V_1
 and the turn-away preparation for s_1 has level V_2
 then after Δt the turn-away preparation for s_1 will have level $V_2 + \gamma [f(\omega_9 V_1) - V_2] \Delta t$.
 $\text{cs}(b, V_1) \& \text{tap}(s_1, V_2) \rightarrow \text{tap}(s_1, V_2 + \gamma [f(\omega_9 V_1) - V_2] \Delta t)$

A body state is actually changed based on the preparation for it, possibly suppressed by the control state, as expressed in LP6. A turn-away action is described in LP7; sensing a body state is described by LP8 in a straightforward manner.

LP6 Body change

If the emotional preparation for B has level V_1
 and the control state for B has level V_2
 and the body effector for B has level V_3
 then after Δt the body effector for B will have level $V_3 + \gamma [f(\omega_{12} V_1, \omega_8 V_2) - V_3] \Delta t$.
 $\text{ep}(B, V_1) \& \text{cs}(B, V_2) \& \text{be}(B, V_3) \rightarrow \text{be}(B, V_3 + \gamma [f(\omega_{12} V_1, \omega_8 V_2) - V_3] \Delta t)$

LP7 Turn-away action

If the turn-away preparation for stimulus s_1 has level V_1
 and the turn-away effector of s_1 has level V_2
 then after duration Δt the turn-away effector for s_1 will have level $V_2 + \gamma [f(\omega_{10} V_1) - V_2] \Delta t$.
 $\text{tap}(s_1, V_1) \& \text{tae}(s_2, V_2) \rightarrow \text{tae}(s_1, V_2 + \gamma [f(\omega_{10} V_1) - V_2] \Delta t)$

LP8 Sensing a body state

If the body effector for body state B has level V_1
 and body state B is sensed with level V_2
 then after duration Δt body state B will have level $V_2 + \gamma [f(\omega_{13} V_1) - V_2] \Delta t$.
 $\text{be}(B, V_1) \& \text{ss}(B, V_2) \rightarrow \text{ss}(B, V_2 + \gamma [f(\omega_{13} V_1) - V_2] \Delta t)$

Sensing stimulus s_1 does not only depend on the actual world state, but also on whether a turn-away action has been performed; this is described in LP9. On the other hand, sensing stimulus s_2 does only depend on the actual world state; it is described in LP10.

LP9 Sensing stimulus s_1

If the turn-away effector for stimulus s_1 has level V_1
 and the world state for s_1 has level V_2
 and stimulus s_1 is sensed with level V_3
 then after duration Δt stimulus s_1 will be sensed with level $V_3 + \gamma [f(\omega_{14} V_1, \omega_0 V_2) - V_3] \Delta t$.
 $\text{tae}(s_1, V_1) \& \text{ws}(s_1, V_2) \& \text{ss}(s_1, V_3) \rightarrow \text{ss}(s_1, V_3 + \gamma [f(\omega_{14} V_1, \omega_0 V_2) - V_3] \Delta t)$

LP10 Sensing stimulus s_2

If the world state for s_2 has level V_1
 and stimulus s_2 is sensed with level V_2
 then after duration Δt stimulus s_2 will be sensed with level $V_2 + \gamma [f(\omega_0 V_1) - V_2] \Delta t$.
 $ws(s_2, V_1) \ \& \ ss(s_2, V_2) \rightarrow ss(s_2, V_2 + \gamma [f(\omega_0 V_1) - V_2] \Delta t)$

3. Simulation Experiments

In this section simulation results are discussed for a number of example scenarios, which all involve an emotional preparation triggered by some neutral but trauma-related stimulus s_1 . It is assumed that the person has experienced a traumatic event in the past, and due to this event the person has developed a configuration of connections that does not exist in a healthy person. The considered scenarios relate to phenomena in literature, as discussed in Section 1. They have been generated by the LEADSTO software environment (cf. [3]), and using the jHepWork data-analysis framework to analyse and present the simulation results (cf. [5]).

The first scenario addressed describes how a neutral stimulus affects a healthy subject, who does not react in a traumatized manner. The second and third scenario (Figures 2 and 3) concern the presentation of the stimulus s_1 to two kinds of PTSD subjects in a situation where they can avoid being exposed to it. In traces, the world state and sensor state of stimulus s_2 are not shown for the sake of clarity as they are equal to zero in the simulated scenario. In Figures 2 and 3 time is on the horizontal axis and activation levels for the different states as indicated on the vertical axis. The parameter values used (connection strenghts ω and threshold τ and steepness σ values for the logistic threshold function) can be seen in Table 3, those have been chosen according the neurological findings presented in Section 1 and tuned with intuitive sense. The step size taken is $\Delta t = 1$.

3.1. Scenario Showing a Healthy Subject

The first case describes how a healthy person senses a neutral stimulus:

- External stimulus s_1 occurs and triggers a sensory representation of this stimulus that does not trigger emotional preparation, because ω_5 is zero.
- This absence of emotional preparation makes that feeling b does not increase, and control over the situation is not performed.

The important parameter in this scenario is the low (even 0) value of the connection ω_5 , which links the neutral stimulus representation to an emotional preparation. No emotional load occurs because this neutral stimulus is not associated to any traumatic experience; the sensor state $ss(s_1)$ and its representation $srs(s_1)$ activate because the neutral stimulus occurs (world state $ws(s_1)$ is active), but no emotional preparation occurs: the emotional preparation state $ep(b)$ stays at 0.

3.2. Scenario Showing a Flashback PTSD subject

The second case considered describes a situation where the person suffers from flashback symptoms of PTSD. He or she is confronted to a stimulus which in principle

is neutral, but is related to the traumatic experience (for example, a car, when the patient's traumatic experience is a car accident).

Table 3. Parameter values for the three scenarios

	state	healthy		flashback		dissociation	
		τ	σ	τ	σ	τ	σ
LP0	sensory representation of s_1	0.2	4	0.2	4	0.2	4
LP1	sensory representation of s_2	0.2	4	0.2	4	0.2	4
LP2	sensory representation of body	0.2	4	0.2	4	0.2	4
LP3	control state	0.5	4	0.5	4	0.3	4
LP4	emotional preparation	0.6	4	0.2	4	0.2	4
LP5	turn away preparation	0.2	4	0.2	4	0.2	4
LP6	body effector	0.4	4	0.4	4	0.4	4
LP7	turn away effector	0.2	4	0.2	4	0.2	4

LP	ω	healthy	flashback	dissociation
LP10	ω_0	1	1	1
LP0	ω_1	1	1	1
LP2	ω_2	1	1	1
LP4	ω_3	0.6	0.6	0.6
LP4	ω_5	0	0.6	0.6
LP3	ω_6	0.6	0.6	0.6
LP2	ω_4	0.2	0.7	0.7
LP4	ω_7	-0.4	-0.4	-0.7
LP6	ω_8	-0.4	-0.4	-0.4
LP5	ω_9	0.8	0.8	0.8
LP7	ω_{10}	0.6	0.6	0.6
LP2	ω_{11}	0	-0.2	-0.8
LP6	ω_{12}	0.7	0.7	0.7
LP8	ω_{13}	1	1	1
LP9	ω_{14}	-1	-1	-1
LP4	ω_{15}	0.8	0.8	0.8
LP1	ω_{16}	0	0.6	0.2
LP3	ω_{17}	0.5	0.5	0.5
LP3	ω_{18}	0	0.3	0.3

In this PTSD subject, the stimulus triggers a strong emotional preparation that brings the person in a state of re-experiencing the traumatic episode. This reaction activates the control state that makes the agent turning away from the stimulus, but the emotional regulation is impaired, and although the stimulus is not present anymore, there is no way to inhibit the aversive feelings. The parameter values used are shown in Table 3, bold non-italic values are those that differ from the healthy person case. The simulation is shown in Figure 2. This situation shows the following:

- The external stimulus s_1 occurs and triggers the representation $srs(s_1)$ of this stimulus that generates a high emotional preparation $ep(b)$ through a high connection strength ω_5 and lightly triggers the control state $cs(b)$ proactively through ω_{18} .
- The emotional preparation drives the sensory representation $srs(b)$ of body state b up via ω_4 and at the same time triggers via ω_{16} sensory representation $srs(s_2)$ of the traumatic memory.
- Via ω_6 and ω_{17} these representations strongly activate the control state $cs(b)$ for the regulation.
- The control state triggers the turn-away preparation via ω_9 but fails to inhibit sufficiently the emotional load (which is too high due to the re-visualization of the traumatic past memories).
- The turn-away preparation leads to the turn-away action (effector state $t\ae(s_1)$) that takes the stimulus away from the agent (inhibition of sensor state $ss(s_1)$) via connections ω_{10} and ω_{14} .

- Although the stimulus is not present anymore or has become very weak (low $ss(s_1)$), the emotional preparation is not regulated due to the impairment of emotion regulation, and the emotional load stays high because the flashback and re-experiencing elements ($srs(s_2)$ and $srs(b)$) stay high and propagate via ω_{15} and ω_3 .

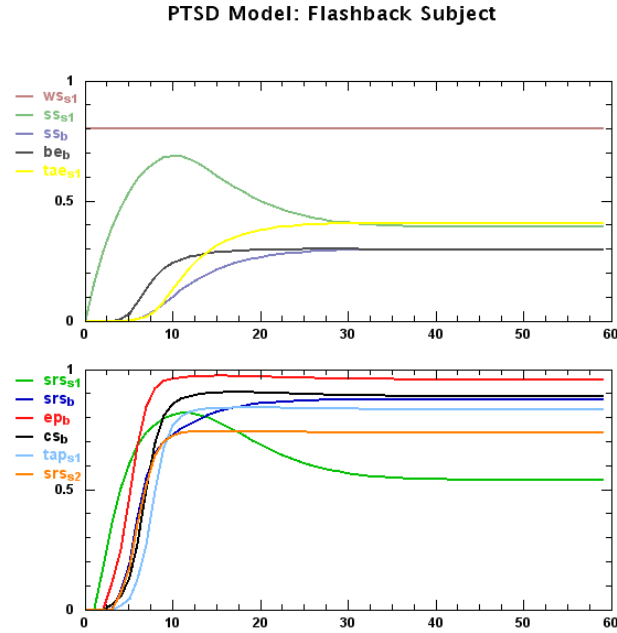


Figure 2. Simulated Flashback PTSD scenario (upper graph: sensor states, world states and body states; lower graph: internal sensory representation, preparation and control states)

In the simulation, the turn-away movement is observed by the increased level of the turn-away effector, $t\alpha e(s_1)$, in yellow, starting after time point 5. This action causes lowering of the sensed stimulus and by repercussion, its representation, but does not remove the visual representation of the trauma neither the physical discomfort ($srs(s_2)$ and $srs(b)$ in orange and blue respectively). Thus, emotional preparation ($ep(b)$, in red) is not significantly lowered, the agent is overrun by the emotion.

3.3. Scenario Showing a Dissociative PTSD subject

The third case describes a situation where the person suffers from dissociative symptoms of PTSD. He or she again is confronted with a stimulus which is associated to the traumatic experience. This kind of stimulus triggers a strong emotional preparation that puts back the patient in a state of re-experiencing the traumatic episode, but also proactively triggers the control state. This activation of the control state is then emphasized by the enhanced sensitivity due to emotional preparation. It makes the agent turn away from the situation, but also inhibits his or her emotional preparation as a defensive mechanism to avoid falling in strong reliving of the trauma.

Parameter values used are shown in Table 3; bold values are those that differ from the healthy person. The simulation can be seen in Figure 3; it shows the following:

- The external stimulus s_i occurs and triggers the representation $srs(s_i)$ of this stimulus that starts to generate an emotional preparation $ep(b)$ through ω_5 and lightly triggers the control state $cs(b)$ proactively through ω_{18} .
- The emotional preparation starts to increase the sensory representation $srs(b)$ of body state b via ω_4 , and traumatic memories can be recalled, shown in a light increase of $srs(s_2)$ via ω_{16} .
- The control state $cs(b)$ is strongly activated by both sensory representations $srs(b)$ and $srs(s_i)$ through ω_6 and ω_{18} .
- This activation of the control state results in inhibiting the sensory representation $srs(b)$ of body state b thus decreasing the re-experiencing (by ω_{11} and ω_7), and finally activates turn-away preparation $tap(s_i)$ to avoid the stimulus (through ω_9).
- The turn-away preparation propagates through ω_{10} to the turn-away effector $t\ae(s_i)$ to make the person look in another direction (by connection ω_{14}).
- The stimulus is lowered but the control state continues to act over the emotional preparation and body representation (through ω_7 and ω_{11}), which explains the weak $ep(b)$ and very low $srs(b)$, which is felt as dissociation.

PTSD Model: Dissociative Subject

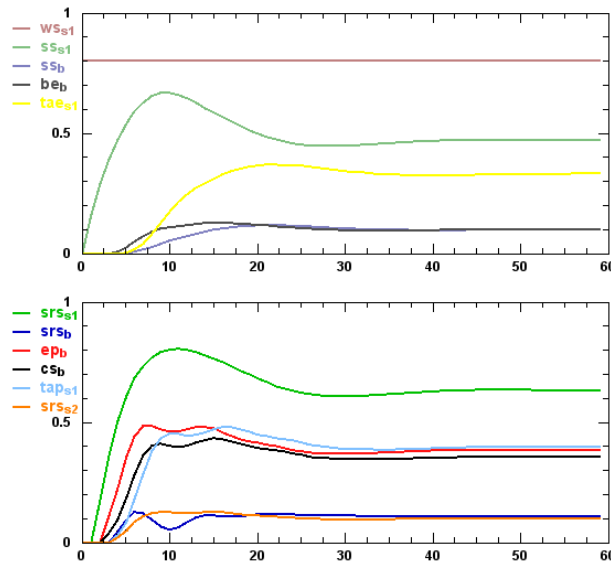


Figure 3. Simulated dissociative PTSD scenario (upper graph: sensor states, world states and body states; lower graph: internal sensory representation, preparation and control states)

In the simulation, at time point 7 the emotional preparation peak occurs while the control state activation is not yet reached (low $cs(b)$ in black before time point 7). The control activation suppresses the progression of emotional preparation and inhibits the body perception ($ep(b)$ in red stops growing and $srs(b)$ in blue get back to 0). As another control effect, the agent turns away from the stimulus ($t\ae(s_i)$, in yellow, is enhanced from time point 7). This affects the perception of the stimulus and its sensory representation (reduced $ss(s_i)$ and $srs(s_i)$).

4. Discussion

The presented computational agent model was designed using principles from the neurological literature on Post-Traumatic Stress Disorders. It was shown that by assuming deviant strengths for some connections and some deviant threshold values lead to patterns that are in accordance with patterns described in the literature. The assumption is that the traumatic experience itself has an impact on these connections and thresholds, by which they become changed for longer time periods. The presented model differs substantially from the model presented in [18] and [19], as this associative memory model focuses mainly on memory formation and does not consider the neurological modifications due to extreme emotions in addition to memory recall.

Referring to [16], the presented model falls in two categories. Firstly, it addresses at an abstract level reconstruction of the neural links and processes that underlie an organisms' emotional reactions, and therefore it falls in the category of anatomic approaches. But, secondly, it also falls in the category of appraisal-derivation approaches: emotion is assumed to arise from individual judgment concerning the relationship between events and an individual's beliefs, desires and intentions.

The obtained cognitive/affective agent model can be used as a basis for the design of human-like virtual agents in simulation-based training or in gaming or virtual stories. For the first type of application a virtual patient can be developed based on the model so that, for example, a psychiatrist or psycho-therapist (e.g., during his or her education) can gain insight in the processes in certain types of PTSD patients, or it can be used by a therapist to analyze how a certain form of therapy can have its effect on these processes. For the second type of application a system for agent-based virtual stories can be designed in which, for example, persons with PTSD play a role, that can be based on the presented model, and progressively recover from the trauma.

Modeling causal relations discussed in neurological literature in the manner as presented here does not take specific neurons into consideration but can use more abstract mental states, relating, for example, to groups of neurons. This is a way to exploit within the agent modelling area results from the large and more and more growing amount of neurological literature. This can be considered as a way of abstraction by lifting neurological knowledge to a mental (cognitive/affective) modelling level. Nevertheless, the type of agent model that results shows some technical elements that are also used at the neurological modelling level. For example, it takes states as having a certain activation level, instead of binary, for example in order to make reciprocal cognitive/affective loops and gradual adaptation possible. As a consequence, for a state causally depending on multiple other states, values for such incoming activation levels have to be combined. Therefore combination functions f are needed, such as the one based on the continuous logistic threshold function used here, or an alternative combination function f can be considered, such as:

$$\begin{aligned} f(W_1, W_2) &= 1 \text{ if } W_1 + W_2 \geq \tau, \text{ and } 0 \text{ otherwise} \\ f(W_1, W_2) &= \beta \max(W_1, W_2) + (1-\beta) \min(W_1, W_2) & (0 \leq \beta \leq 1) \\ f(W_1, W_2) &= \beta(1 - (1-W_1)(1-W_2)) + (1 - \beta)W_1W_2 & (0 \leq \beta \leq 1) \end{aligned}$$

Note that similar numerical elements play a role in the area of modelling imperfect reasoning, for example based on fuzzy or uncertain information. So, in order to model such an agent at a cognitive/affective level abstracting from neurological detail, still some machinery is needed that might be associated to a neural modelling perspective. However, in order to successfully model agents with more complex and human-like

behaviour, for example incorporating regulation processes, mutual cognitive/affective interaction loops, and/or feedback loops modelling adaptivity, the toolset for the agent modeller has to include such modelling techniques, enabling to model agents in a hybrid logical/numerical manner.

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