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Research article

A computational model to simulate development and recovery of traumatised patients

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ABSTRACT

In this paper, a computational model is presented to simulate emotional response of people after traumatising events, including their development, recovery, and the effect of group support. The model is built upon mechanisms known from cognitive and social neuroscience. Using the model, several scenarios were explored, considering both individual and groups of people. The simulation results were validated on a dataset of symptoms and recovery of patients with PTSD. The obtained model enables simulation and analysis of emotional response evolution of diverse personality types, and how extensible are effects of group therapy on patients with PTSD. Simulation results show that group therapy is positive but in the long term its effect reduces, suggesting that changing people in groups every period of time helps to keep group's atmosphere healthy, contributing to recovering of patients. The gain of group therapy also depends on the type of people present in the group which in some cases can be prejudicial to some members when dependent and toxic relations are formed between them.

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Introduction

After experiencing a traumatic event, most people recover within a few months. However, if this does not happen, a person can develop a condition, possibly diagnosed as Post Traumatic Stress Disorder (PTSD) that strongly affects one's life. During the past decades, post-trauma and PTSD patients have been extensively studied, leading to a better understanding of their symptoms; e.g., (Duvarci & Pare, 2014; Masten & Narayan, 2012; Parsons & Ressler, 2013). A patient with PTSD can suffer from different symptoms, such as repeated and unwanted re-experiencing of the event (flashbacks), hyperarousal, avoidance of stimuli or thoughts that could remind to the event, and emotional numbing involving loss of body perception (dissociation); all of these lead to unwanted emotional responses. A concept that has been known for a long time but is only recently being studied scientifically is group therapy for patients with PTSD (Litwack, Beck, & Sloan, 2015; Sloan, Feinstein, Gallagher, Beck, & Keane, 2013). Generally, no significant findings were obtained for group interventions relative to individual treatment comparison conditions, although group therapy did have superior effects relative to a wait-list comparison condition. However, some aspects of group therapy can

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http://dx.doi.org/10.1016/j.bica.2017.07.002 2212-683X/© 2017 Elsevier B.V. All rights reserved. make it a worthwhile investment. First of all, group therapy provides a possibility for often socially isolated patients to develop social relationships in a safe environment, essential for the recovery process of the patient (Foy, Eriksson, & Trice, 2001). Also, group therapy gives patients with PTSD the possibility to identify themselves with others that are in the same situation, making them feel less alone in their suffering and less frustrated about their symptoms. This identification with other patients can have advantages compared to a therapist that did not go through the same trauma. Finally, group therapy could be more cost effective in situations where staff is limited (Litwack et al., 2015). However, while the presence of other patients with PTSD can lead to a feeling of safety and connection, there is also the possibility for individuals to experience other group members as unsafe or a bad influence, which can have counterproductive effects on their recovery. Therefore, it is important to keep track of all the relations within the support group (Litwack et al., 2015). In their work, (Admon, Milad, & Hendler, 2013) review many works that show strong evidence of the relation between Amygdala, dorsal anterior cingulated cortex, ventromedial pre-frontal cortex and hippocampus. They support the idea that traumas, along life and special for children, increase the volume of amygdala, decrease the volume of anterior cingulate cortex and reduce the connection with regions of the pre-frontal cortex, making those people more leaning to other traumas or reactivate old traumas. That indicates a relation of amygdala in activating fear, reviving traumas and the pre-frontal cortex areas

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in suppressing them. Those two relations are emphasised by the works of (Admon et al., 2013) which allege that the pre-frontal cortex works on minimise traumas, and (Panksepp & Biven, 2012, chap. 1) showing strong evidence that the amygdala boost emotions in animals and human beings.

Alongside the research on patients with PTSD and group therapy, a lot of studies have been done on various cognitive emotion regulation strategies within humans. By regulating emotions, individuals can balance how they feel, helping them to maintain a form of emotional homoeostasis and have a form of control on their emotional response to certain stimuli. One example of such a strategy is cognitive reappraisal, where an individual reappraises a potentially emotion-eliciting situation in terms that decreases its emotional impact. Using fMRI a parallel was found between this reappraisal and increased activation of the lateral and medial pre-frontal regions and decreased activation of the amygdala and medial orbito-frontal cortex, which supports the hypothesis that the pre-frontal cortex is involved in constructing reappraisal strategies (Brosch & Sander, 2013; Ochsner, Bunge, Gross, & Gabrieli, 2002). Another strategy for emotion regulation is suppression of an emotional response, without taking away or modifying the triggers for this response. When an individual repeatedly suppresses an unwanted emotion caused by some stimulus, the link between the stimulus and the unwanted emotion will not strengthen much, and the suppression itself leads to a decrease in physiological aspects and experience of negative emotions (Ochsner & Gross, 2014; Webb, Miles, & Sheeran, 2012). Furthermore, (Goldenberg, Halperin, van Zomeren, & Gross, 2015) explains that emotion regulation also exists among groups. Individuals in groups attempt to regulate their emotions in line with specific collective goals, partly based on the individual's self-categorization as a group member, this way the influence of a group defines the way an individual regulates his or her emotions.

In this study, a computational model was developed based on the concepts described above. It extends a previous work (Formolo, Van Ments, & Treur, 2016), modelling diverse PTSD situations, adding more experiments, a description of the real data set used to validate it and results of the experiments. All these new elements show flexibility of the model to adapt to situations and people's profile. The model describes processes and developments that happen within a patient with PTSD, in particular, the (learning of) generation and regulation of emotional responses within that individual, and for the situation that the person participates in (group) therapy. The obtained model could help to create a better understanding of the influence of (group) therapy and other environmental influences on a patient with PTSD, and how these external factors can help the patient in the recovery process. Also, the model can be a basis for a software application that supports (group) therapy for traumatised patients, helping to overcome the challenges in group therapy that were mentioned above. Finally, with a large part of the recent wave of refugees arriving in the Western world suffering from trauma (URL1, 2016), the model could be valuable in supporting the growing need for post-traumatic therapy. In Section 'Description of the computational model' the computational model is introduced; Section 'Co mparison to empirical data' explains how empirical data were compared with outputs of model; Section 'Simulation experiments' describes various simulated scenarios; and in Section 'Discussion' is discussed the model and its results.

Description of the computational model

As discussed in Section 'Introduction', a patient with PTSD can suffer from different symptoms, which can be different for every person. Many factors define the way an individual copes with a traumatic experience: age, gender, past trauma experiences, supportive and protective factors like family and friends, cognitive skills, neurobiological protection, and others (Masten & Narayan, 2012).

Conceptual representation of the model

Patients with PTSD can respond to a traumatic event in two ways: by dissociation or by flashback. Each patient usually reacts with only one of these responses. Flashback patients are overreacting and fall into a strong re-experience of the trauma, accompanied with visual recall. Dissociative patients react to traumatic emotion recalls by strongly suppressing body and emotional effects and appraisals; e.g., (Oathes & William, 2008; Scaer, 2001). The model proposed here was designed according to the temporal-causal network modelling approach described in Treur (in press). This approach is suitable to mimic interactions among brain regions. Each state or group of states in the model plays a role. In this sense, the Control State in the model is related to Pre-frontal Cortex, while the Feeling State plays the role of amygdala. In their book, (Panksepp & Biven, 2012, chap. 1) describe how the regions in the brain interact, especially those related to emotions and sentiments and how they, in combination, respond to external stimuli. The model uses those brain mechanisms to estimate the emotional response of patients with PTSD at seconds to minutes time scale. The graphical conceptual representation of the model shown in Fig. 1 describes the states and connections of one patient with PTSD. The states inside the dashed square represent internal mental processes; the underlined connection weights are negative. In Table 1, the states and their notations are explained. Below, a more extensive description of the states and connections is given.

Both the development and recovery of a trauma can be modelled by assuming adaptability (strengthening) of the weights of a number of connections, for example, through Hebbian learning (Naze & Treur, 2012). This theory is based on the principle that connected neurones that are frequently activated simultaneously strengthen their connection. Some literature on this concept, including mathematical formulations, can be found in Gerstner and Kistler (2002). First of all, the model in Fig. 1 has several states that provide input from the external world: social support ws_{ss}, negative contagion ws_{nc}, trauma stimulus ws_{te}, trigger stimulus ws_{tr}, and environment stimulus ws_s.

These external stimuli are more extensively described below. An individual receives external input through the sensor state. In this model, the sensor states are ss_{ss} , ss_{nc} , ss_{te} , ss_{tr} and ss_{s} . The sensor states lead to sensory representations srs_{ss} , srs_{nc} , srs_{te} , srs_{tr} and srs_s within a person. These states define the intensity of external stimuli felt by the person. Each person has a different perception of external stimuli, for example, one can be more receptive to social support or more sensible to traumatic events than the other. Furthermore, there are six more states. First, as described in the introduction, the control state cs_b for emotion b monitors feelings and preparation for emotion b. If an unwanted emotion occurs, the control state suppresses this emotion *b*. Second, feeling state fs_b is affected before performing an action through the preparation state ps_b and the control state by a predictive as-if body loop (Damasio, 1999, 2003). In this paper, b is a negative emotion. Third, the preparation state ps_b is responsible for the brain mechanism of emulating situations before making a decision, according to internal simulation generating a cycle through feelings about the situation emulated. That decision activates the expressed emotional response es_b. This is the actual execution of the emotional response of *b* by the person. It expresses the level of distress, for example feeling scared. A high output of es_b means a high level of distress. Finally, the belief state about the trauma bs_{te,b} leads to expression

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