A Higher-Order Adaptive Network Model to Simulate Development of and Recovery from PTSD

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Abstract. In this paper, a second-order adaptive network model is introduced for a number of phenomena that occur in the context of PTSD. First of all the model covers simulation of the formation of a mental model of a traumatic course of events and its emotional responses that make replay of flashback movies happen. Secondly, it addresses learning processes of how a stimulus can become a trigger to activate this acquired mental model. Furthermore, the influence of therapy on the ability of an individual to learn to control the emotional responses to the traumatic mental model was modeled. Finally, a form of second-order adaptation was covered to unblock and activate this learning ability.

Keywords: PTSD, higher-order adaptive, mental model, flasback movie

1 Introduction

A Post Traumatic Stress Disorder (PTSD) is usually developed after experiencing one or a course of events that trigger strong negative emotions like fear; e.g., [7, 21]. One of the symptoms is a recurring re-experiencing of the course of events that led to the trauma and that are played again and again in the mind as a kind of flashback movie and thereby trigger the strong negative emotions again. In the literature such as ([2-3, 13, 28] strong evidence can be found for relations to amygdala, dorsal anterior cingulated cortex, ventromedial prefrontal cortex and hippocampus. One of the reported issues here is a reduction of the connections to regions of the prefrontal cortex, which makes it difficult to apply emotion regulation. The role of the amygdala in activating fear and of the relation between amygdala and the pre-frontal cortex areas in suppressing fear was found to be crucial; e.g., [2, 20]. If the emotion regulation strategy based on suppression is strengthened, this leads to a decrease in physiological and experiential effects of negative emotions; e.g., [9, 19, 27].

Multiple forms of adaptivity play a crucial role in both the development of PTSD and therapies to recover from it. During the development, an important role is played by the learning of a form of mental model of the course of events leading to the trauma. This is a form of observational learning; e.g., [4, 26]. It is this learnt mental model that is the basis of the flashback symptoms. Moreover, during development also learning

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takes place to connect different stimuli (by themselves irrelevant but just co-occurring with the traumatic events) to the traumatic stimuli which makes them triggers for the flashbacks; this is a form of sensory preconditioning; e.g., [5, 11]. To recover from PTSD another form of learning is required: learning to strengthen the connections to the relevant prefrontal cortex areas to improve emotion regulation; e.g., [19, 27]. However, this learning capability is impaired by the stress itself, which prevents the learning from taking place in a natural manner. This effect is called metaplasticity; e.g., [10]. Metaplasticity [1] is a form of second-order adaptation, as it exerts a form of control over adaptation. In contrast, the other forms of adaptation mentioned above are called first-order adaptation.

The focus in the current paper is to introduce a computational network model addressing all these forms of adaptivity pointed out above. This leads to a second-order adaptive network model in which during development of PTSD a mental model for the flashbacks is learnt and also an association of a trigger to the traumatic events (both first-order adaptation). As an additional effect of the development phase, a negative effect of metaplasticity occurs that impairs the plasticity of the emotion regulation (second-order adaptation). For recovery, a therapy is applied to resolve the impairment of the plasticity of the emotion regulation which is a positive effect of metaplasticity (second-order adaptation). After this, the learning to strengthen the emotion regulation takes place which then leads to recovery (first-order adaptation).

In Section 2 some background knowledge is discussed for the different types of adaptation. Section 3 introduces the second-order adaptive network model to address these forms of adaptation. In Section 4 some example simulations for this network model are discussed. Finally, Section 5 is a discussion.

2 Background Knowledge on Adaptation Principles Used

As discussed above, different forms of adaptation play a role in development of and recovery from traumas. The more specific adaptation principles for these forms of adaptation are discussed in this section.

2.1 First-order adaptation principle: Hebbian learning

In neuroscientific literature such as [6], two types of first-order adaptation principles are discussed: synaptic and non-synaptic. An example of the latter type is intrinsic excitability adaptation, which will not be used here. Hebbian learning is a well-known first-order adaptation principle of the first type; it addresses adaptive connectivity [12]. It can be explained by:

> 'When an axon of cell A is near enough to excite B and repeatedly or persistently takes part in firing it, some growth process or metabolic change takes place in one or both cells such that A's efficiency, as one of the cells firing B, is increased.' [12], p. 62 (1)

This is sometimes simplified (neglecting the phrase 'one of the cells firing B') to:

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‘What fires together, wires together’ [14, 22] (2)

This first-order adaptation principle will be used to model adaptation for the following.

- Development of the trauma:
  - Learning of a connection of a trigger stimulus to the traumatic course of events based on sensory preconditioning [5, 11]
  - Learning the connections in the mental model of the traumatic course of events based on observational learning, also using sensory preconditioning [4, 26]
- Recovery from the trauma:
  - Strengthening emotion regulation for recovery by learning the connections to the prefrontal cortex areas [19, 27]

2.2 Second-order adaptation principle: Stress reduces adaptation speed

In [10] the focus is on the role of stress in reducing or blocking plasticity. Many mental and physical disorders are stress-related, and are hard to overcome due to poor or even blocked plasticity that comes with the stress. Garcia [10] describes the negative role of stress-related metaplasticity for this, which often leans to a situation that a patient is locked in his or her disorder by that negative pattern. However, he also shows that by some form of therapy this negative cycle might be broken:

> ‘At the cellular level, evidence has emerged indicating neuronal atrophy and cell loss in response to stress and in depression. At the molecular level, it has been suggested that these cellular deficiencies, mostly detected in the hippocampus, result from a decrease in the expression of brain-derived neurotrophic factor (BDNF) associated with elevation of glucocorticoids.’ [10], p. 629
>
> ‘…modifications in the threshold for synaptic plasticity that enhances cognitive function is referred here to as ‘positive’ metaplasticity. In contrast, changes in the threshold for synaptic plasticity that yield impairment of cognitive functions, for example (..) in response to stress (..), is referred to as ‘negative’ metaplasticity.’ [10], pp. 630-631
>
> ‘In summary, depressive-like behavior in animals and human depression are associated with high plasma levels of glucocorticoids that produce ‘negative’ metaplasticity in limbic structures (…). This stress-related metaplasticity impairs performance on certain hippocampal-dependent tasks. Antidepressant treatments act by increasing expression of BDNF in the hippocampus. This antidepressant effect can trigger, in turn, the suppression of stress-related metaplasticity in hippocampal-hypothalamic pathways thus restoring physiological levels of glucocorticoids.’ [10], p. 634

This second-order adaptation principle will be used to model adaptation for the following.

- Development of the trauma:
  - Reducing the adaptation speed for the learning of the emotion regulation connections to the prefrontal cortex areas due to the high stress levels [10]

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- Recovery from the trauma:
    - Increasing the adaptation speed for the learning of the emotion regulation connections to the prefrontal cortex areas due to a therapy that (temporarily) reduces the stress levels [10]

In Section 3 it will be discussed how these have been modeled by using a so-called self-modeling network model.

3 The Second-Order Network Model

In this section, a detailed overview is presented of the designed second-order adaptive network model for modeling the learning of PTSD trauma and the influence of therapy on recovery. For the modeling, we use the Network-Oriented Modeling approach introduced in [23] and further developed to cover higher-order adaptive networks in [24, 25], where also the supporting dedicated software environment is presented.

3.1 The general format

This approach can be broken down in the following steps:

- Translating the domain into a conceptual causal network model in terms of network characteristics
- Transcribing the conceptual causal network model into a standard table format called *role matrix format*. These role matrices break down the network characteristics for all the different types of causal influences on a state in the model
- The network characteristics are grouped into the following types:

    1. **Connectivity characteristics**
    What *states* X, Y and *connections* $X \rightarrow Y$ are there in the model and what are the *weights* $\boldsymbol{\omega}_{X,Y}$ of the connections? These are specified in role matrix **mb** (for the states and their connections) and **mcw** (for the connection weights $\boldsymbol{\omega}_{X,Y}$)
    2. **Aggregation characteristics**
    How are different impacts from other states on a state Y aggregated by a *combination function* $\mathbf{c}_Y(..)$ and what are the values of the *parameters* for these combination functions? The combination functions are chosen from a library by assigning weights $\boldsymbol{\gamma}_{i,Y}$ to them and values for the parameters $\boldsymbol{\pi}_{i,j,Y}$ are set. These characteristics are specified in role matrix **mcfw** (for combination function weights $\boldsymbol{\gamma}_{i,Y}$) and **mcfp** (for the combination function parameters $\boldsymbol{\pi}_{i,j,Y}$)
    3. **Timing characteristics**
    How fast do the states Y change upon the received impact, due to their *speed factor* $\boldsymbol{\eta}_Y$? These speed factors $\boldsymbol{\eta}_Y$ are specified in role matrix **ms**.

- Providing the above network characteristics as tables in role matrix format as input for the available dedicated software environment. Based on these received tables, the software environment runs simulations.

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3.2 Translating the domain knowledge into a conceptual causal model

Based on a domain study, the first step towards building a computational model is translating the processes and brain mechanisms discussed in the literature into a conceptual causal network model. To accommodate for the forms of adaptation of different orders order for the model, the conceptual model uses so-called *self-modeling networks* that include self-models, in this case leading to three levels (see Fig. 1):

1. **The Base Level**
   This level includes all *basic* (non-adaptive/non-learning) *processes* of the conceptual model.
2. **The First-Order Self-Model Level (or First Reification Level)**
   On this level, states are added that represent (adaptive) network characteristics of the base level. For example, a *self-model state* $\mathbf{W}_{X,Y}$ can be added to represent an adaptive connection weight $\boldsymbol{\omega}_{X,Y}$, or a *self-model state* $\mathbf{H}_Y$ can be added to represent a speed factor $\boldsymbol{\eta}_Y$. In the model in this way the learning of several connections in the base level takes place through Hebbian learning. These learning connections are represented by the dynamics of the **W**-states in the blue middle plane. This first-order self-model enables adaptation of the connections of the mental model in the base level.
3. **The Second-Order Self-Model Level (or Second Reification Level)**
   Because the learning itself is adaptive as well, another level is added on top of the first-order self-model level: the second-order self-model level. This level allows to *control the learning speed* of the states $\mathbf{W}_{X,Y}$ for the learning connections by adding state $\mathbf{H}\mathbf{w}_{X,Y}$ here representing the speed factor of $\mathbf{W}_{X,Y}$.

See for the connectivity of the network model Fig. 1; Table 1 shows the states and brief explanations of them. Within the network model, the first-order adaptation based on the Hebbian learning principle has been modeled by using a *connectivity self-model* (in the blue plane) based on self-model states $\mathbf{W}_{X,Y}$ representing connection weights $\boldsymbol{\omega}_{X,Y}$. These self-model states need incoming and outgoing connections to let them function within the network. To incorporate the 'firing together' part of (2) from Section 2, for the self-model's connectivity, incoming connections from X and Y to $\mathbf{W}_{X,Y}$ are used; see Fig. 1 (upward arrows in blue). These upward connections have weight 1. Also a connection from $\mathbf{W}_{X,Y}$ to itself with weight 1 is used to model persistence of the learnt effect; in pictures they are usually left out. In addition, an outgoing connection from $\mathbf{W}_{X,Y}$ to state Y is used to indicate where this self-model state $\mathbf{W}_{X,Y}$ has its effect; again see Fig. 1 (pink downward arrow). The downward connection indicates that the value of $\mathbf{W}_{X,Y}$ is actually used for the connection weight of the connection from X to Y. For the *aggregation characteristics* of the first-order self-model, the Hebbian learning rule is defined by the combination function $\mathbf{hebb}_{\boldsymbol{\mu}}(V_1, V_2, W)$ for self-model state $\mathbf{W}_{X,Y}$ from Table 4.

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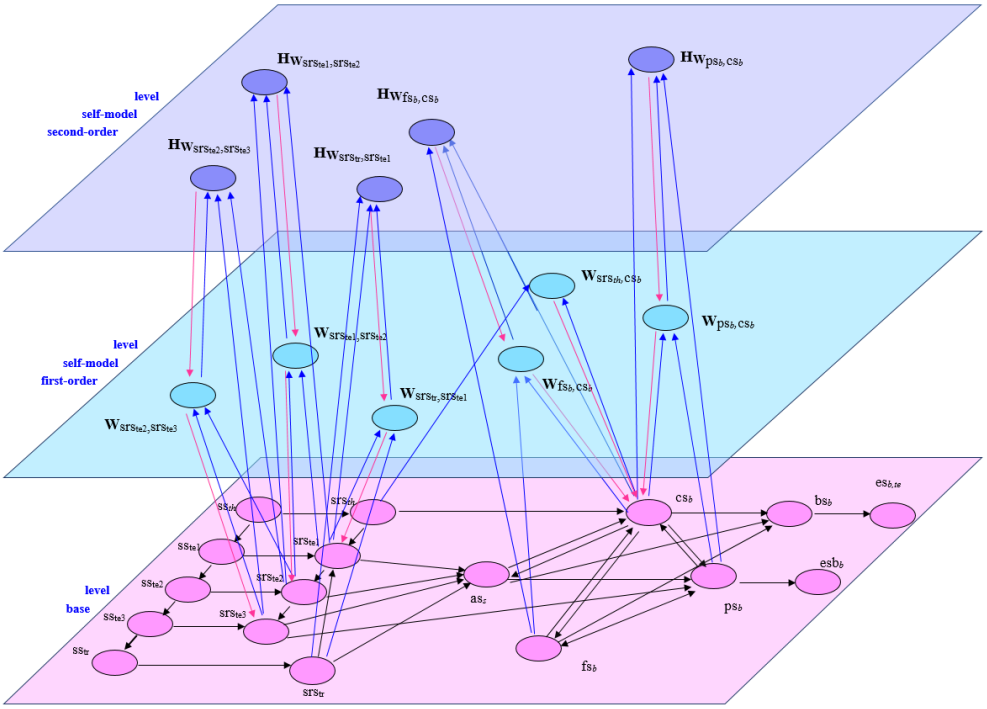

Fig. 1. Connectivity of the introduced second-order adaptive network model

The sensing of an example of a traumatic course of events is modeled by the sensor states ss_{te1}, ss_{te2}, ss_{te3}. For example, te1 or traumatic event 1, is a potentially dangerous situation for a child you observe , te2 is an action from your side with the intention to save the child from that situation and te3 is an unfortunate failure of your action such that the child actually gets hurt. During this traumatic course of affairs, sensory representations srs_{te1}, srs_{te2}, srs_{te3} are activated for these events te1, te2 and te3, and by sensory preconditioning the connections between these sensory representations are learned. By this observational learning process, the mental model of the traumatic course of events is formed and represented by first-order self-model states $\mathbf{W}_{srs_{te1},srs_{te2}}$ and $\mathbf{W}_{srs_{te2},srs_{te3}}$. Similarly, the connection between the sensory representations of the trigger tr and the traumatic events is learnt based on sensory preconditioning, represented by $\mathbf{W}_{srs_{tr},srs_{te1}}$. These newly formed connections activate the mental model every time the trigger is sensed. For the traumatized person this shows as an internal flashback movie of the traumatic course of events. In turn, this flashback movie activates the related negative emotions experienced at the original traumatic events.

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Table 1. The states in the network model and their explanation

state		explanation
X_1	ss_{te1}	Sensor state for traumatic event phase 1: observation te1
X_2	ss_{te2}	Sensor state for traumatic event phase 2: action te2
X_3	ss_{te3}	Sensor state for traumatic event phase 3: effect te3
X_4	ss_{tr}	Sensor state for trigger tr for traumatic sequence of events
X_5	ss_{th}	Sensor state for trigger th for therapy input
X_6	srs_{te1}	Sensory representation state for traumatic event phase 1: observation te1
X_7	srs_{te2}	Sensory representation state for traumatic event phase 2: action te2
X_8	srs_{te3}	Sensory representation state for traumatic event phase 3: effect te3
X_9	srs_{tr}	Sensory representation state for trigger tr for traumatic sequence of events
X_{10}	srs_{th}	Sensory representation state for therapy th from therapy
X_{11}	as_{te}	Awareness state for traumatic sequence of events te
X_{12}	ps_b	Preparation state for emotional response b
X_{13}	fs_b	Feeling state for emotional response b
X_{14}	cs_b	Control state for emotional response b
X_{15}	$bs_{b,te}$	Belief that emotional response b is from traumatic event te
X_{16}	es_b	Bodily expressed emotional response b
X_{17}	$es_{b,te}$	Expressing that emotional response b is from te
X_{18}	$\mathbf{W}_{srs_{te1},srs_{te2}}$	Representation state for weight of connection from srs_{te1} to srs_{te2} for imprinting traumatic sequence of events
X_{19}	$\mathbf{W}_{srs_{te2},srs_{te3}}$	Representation state for weight of connection from srs_{te2} to srs_{te3} for imprinting traumatic sequence of events
X_{20}	$\mathbf{W}_{srs_{tr},srs_{te1}}$	Representation state for weight of connection from srs_{tr} to srs_{te1} for sensory preconditioning to link trigger tr to the traumatic sequence of events
X_{21}	$\mathbf{W}_{ps_b,cs_b}$	Representation state for weight of connection from ps_b to cs_b for learning of emotion regulation
X_{22}	$\mathbf{W}_{fs_b,cs_b}$	Representation state for weight of connection from fs_b to cs_b for learning of emotion regulation
X_{23}	$\mathbf{W}_{th,cs_b}$	Representation state for weight of connection from th to cs_b for learning of emotion regulation from therapy
X_{24}	$\mathbf{Hw}_{srs_{te1},srs_{te2}}$	Control state for adaptation speed for weight of connection from srs_{te1} to srs_{te2}
X_{25}	$\mathbf{Hw}_{srs_{te2},srs_{te3}}$	Control state for adaptation speed for weight of connection from srs_{te2} to srs_{te3}
X_{26}	$\mathbf{Hw}_{srs_{tr},srs_{te1}}$	Control state for adaptation speed for weight of connection from srs_{tr} to srs_{te1}
X_{27}	$\mathbf{Hw}_{ps_b,cs_b}$	Control state for adaptation speed for weight of connection from ps_b to cs_b
X_{28}	$\mathbf{Hw}_{fs_b,cs_b}$	Control state for adaptation speed for weight of connection from fs_b to cs_b

In contrast to what was believed earlier, such learnt connections usually do not show any form of natural extinction; e.g., [15], p. 507. Therefore, to make their effect more bearable, the only option is to suppress the emotional consequences related to the trauma by activating the emotion regulation control state cs_b. However, due to the high negative emotion levels the learning process for the activation of cs_b is impaired: learning speeds $\mathbf{H}_{\mathbf{W}_{ps_b,cs_b}}$ and $\mathbf{H}_{\mathbf{W}_{fs_b,cs_b}}$ are very low. Therefore, without any additional help the situation will stay as it is. But, following [10] the therapy *th* is able to temporarily

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reduce the level of negative emotions, so that $\mathbf{H}_{\mathbf{W}ps_b,cs_b}$ and $\mathbf{H}_{\mathbf{W}fs_b,cs_b}$ get higher values. Due to this, learning of the connections to the control state takes place: $\mathbf{W}_{ps_b,cs_b}$ and $\mathbf{W}_{fs_b,cs_b}$ get higher values.

3.3 Transcribing the conceptual model into role matrices

To allow for easy formalization of the conceptual model into role matrices and an executable computational model, we use generic ways to describe the states, intra-level connections and interlevel connections. See an abstracted overview of all types of states and connections used in the model in Tables 2 and 3.

Table 2. Overview of types of states

State Name	Representation
ss_y	Sensor state for state y in the world
srs_y	Sensory representation state for y
as_s	Awareness state for s
fs_b	Feeling state for feeling b
ps_b	Preparation state for feeling b
cs_b	Control state for feeling b
bs_b	Belief state for feeling b
es_b	Execution state for feeling b
$\mathbf{W}_{X,Y}$	Connection weight representation state for connection $X \rightarrow Y$
$\mathbf{Hw}_{X,Y}$	Learning control state for the connection weight state for connection $X \rightarrow Y$

Table 3. Overview of types of connections

Connection	Representation	Connection Type
$X \rightarrow Y$	Connection between base states X and Y	Intra-level (horizontal) connection
$X \rightarrow \mathbf{W}_{X,Y}$ $Y \rightarrow \mathbf{W}_{X,Y}$	Connections from base level states X and Y to connection adaptation state $\mathbf{W}_{X,Y}$ to support the Hebbian learning formation	Interlevel connection, upward from the base level to the first-order self-model level
$\mathbf{W}_{X,Y} \rightarrow Y$	Connection from connection adaptation state $\mathbf{W}_{X,Y}$ to base state Y; these connections effectuate the learnt connection	Interlevel connection, downward from the first-order self-model level to the base level
$\mathbf{W}_{X,Y} \rightarrow \mathbf{Hw}_{X,Y}$ $X \rightarrow \mathbf{Hw}_{X,Y}$ $Y \rightarrow \mathbf{Hw}_{X,Y}$	Connections from connection adaptation state $\mathbf{W}_{X,Y}$ and base level states X and Y to learning control state $\mathbf{Hw}_{X,Y}$	Interlevel connections, upward from the base level to the second-order self-model level, and upward from the first-order self-model level to the second-order self-model level
$\mathbf{Hw}_{X,Y} \rightarrow \mathbf{W}_{X,Y}$	Connection from learning control state $\mathbf{Hw}_{X,Y}$ to adaptive connection adaptation state $\mathbf{W}_{X,Y}$ to effectuate learning control	Interlevel connection, downward from the third level to the second level

The model with connectivity shown in Fig. 1 was then specified by tables in role matrix format: Connectivity characteristics (1), aggregation characteristics (2) and timing characteristics (3). See the Appendix at URL https://www.researchgate.net/publication/350159052. Four different combination functions from the library are used that each serve a different purpose; see Table 4.

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Table 4. The combination functions used from the library

Combination function	Notation	Formula	Parameters
Advanced logistic sum	$\mathbf{alogistic}_{\boldsymbol{\sigma},\boldsymbol{\tau}}(V_1, \ldots, V_k)$	$[\frac{1}{1+e^{-\boldsymbol{\sigma}(V_1+\cdots+V_k-\boldsymbol{\tau})}} - \frac{1}{1+e^{\boldsymbol{\sigma}\boldsymbol{\tau}}}](1+e^{-\boldsymbol{\sigma}\boldsymbol{\tau}})$	Steepness $\boldsymbol{\sigma}>0$ Excitability threshold $\boldsymbol{\tau}$
Hebbian learning	$\mathbf{hebb}_{\boldsymbol{\mu}}(V_1, V_2, W)$	$V_1 V_2 (1 - W) + \boldsymbol{\mu} W$	Persistence factor $\boldsymbol{\mu}\geq 0$
Steponce	$\mathbf{steponce}(V)$	1 if $\boldsymbol{\alpha} \leq t \leq \boldsymbol{\beta}$, else 0	$\boldsymbol{\alpha}\geq 0$ begin, $\boldsymbol{\beta}\geq\boldsymbol{\alpha}$ end time
Stepmod	$\mathbf{stepmod}_{\boldsymbol{\rho},\boldsymbol{\delta}}(V_1, \ldots, V_k)$	0 if t mod $\boldsymbol{\rho} < \boldsymbol{\delta}$, else 1	Repetition $\boldsymbol{\rho}\geq 0$ Duration $\boldsymbol{\delta}\geq 0$

The advanced logistic sum combination function combines influences of multiple states by adding them but makes sure they stay between 0 and 1, with parameters steepness $\boldsymbol{\sigma}$ and threshold $\boldsymbol{\tau}$. The Hebbian learning combination function is used for learning of a connection weight. The stepmod function allows for an activation of states with a predefined length and frequency (here, that is used for the recurring trigger state). The steponce function allows for the activation of states with predefined length and start time (here, that is used for the therapy and trauma states).

The following standard generic difference equation is used for simulation purposes and also for analysis. It incorporates the network characteristics $\boldsymbol{\omega}_{X,Y}$, $\mathbf{c}_Y(..)$, $\boldsymbol{\eta}_Y$ in a numerical difference equation format:

$$Y(t + \Delta t) = Y(t) + \boldsymbol{\eta}_Y[\mathbf{c}_Y(\boldsymbol{\omega}_{X_1,Y} X_1(t), \cdots, \boldsymbol{\omega}_{X_k,Y} X_k(t)) - Y(t)]\,\Delta t \quad (1)$$

for any state Y and where X_1 to X_k are the states from which Y gets its incoming connections. Based on the role matrices as input, his generic difference equation is automatically applied to all network states (including the self-model states) within the dedicated software environment used to perform simulation experiments.

4 Example Simulations

The role matrices can easily be transferred to the dedicated softare environment for simulations. Running the software loops over a chosen time period (in this case a time interval from 0 to 1400 with step size Δt = 0.5) and provides as output a simulation graph for the model. In Fig. 2 the development of PTSD is shown based on traumatic events te1 to te3 in time period from 100 to 200 without applying therapy. The trigger also occurs from 100 to 200 and after that regularly recurs from 300 to 400, from 500 to 600, et cetera. In Fig. 3 the same is shown but this time therapy is taking place from time 400 to time 800 where the therapy leads to recovery. In both Fig. 2 and 3 in the time period from 100 to 200 the sequence of traumatic events te1 to te3 in the world are sensed (via sensor states ss_{te1}, .., ss_{te3}) of which internal representations srs_{te1}, .., srs_{te3} are made. Due to sensory preconditioning (first-order adaptation based on

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Hebbian learning), the connections between them are developed (thus forming a mental model of the traumatic course of events) and also a connection from the trigger representation srs_{tr} to srs_{te1}. Moreover, they trigger the negative emotional response preparation ps_b and feeling state fs_b, and these in turn reduce the adaptation speed (represented by the **H**-states) of the learning of the connections to the control state cs_b (second-order adaptation for metaplasticity). Therefore, no strengthening of the emotion regulation takes place, what would be needed to get rid of the negative feelings. Every time period that the trigger recurs, due to the connection from srs_{tr} to srs_{te1} and the connections between srs_{te1}, .., srs_{te3}, the flasback movie is replayed (as a form of internal simulation) and because of that the negative emotion and feeling are activated to high values again.

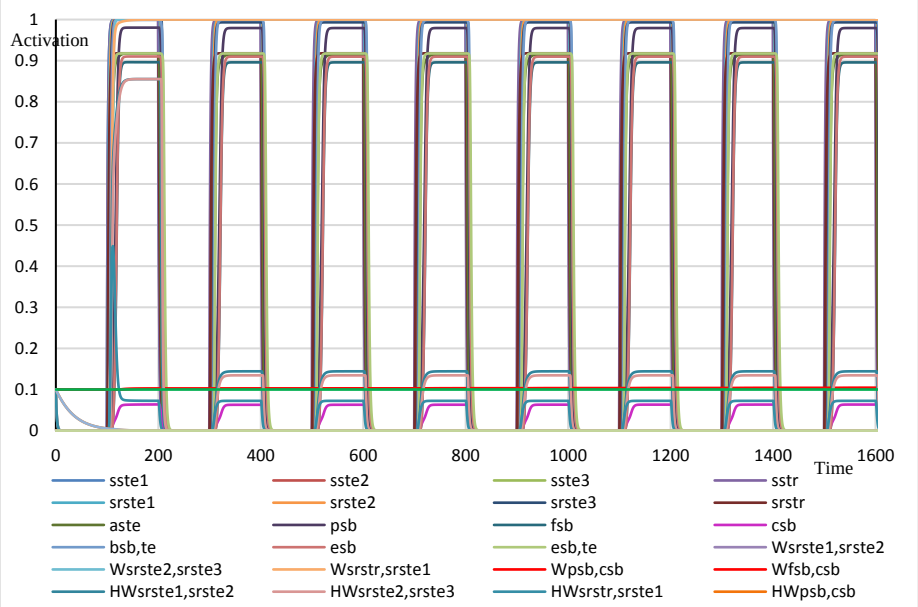

Fig. 2. Development of PTSD without using therapy. The trauma develops from time 100 to 200. The trigger also occurs from 100 to 200 and after that regularly recurs from 300 to 400, from 500 to 600, et cetera. No recovery from PTSD takes place.

5 Discussion

In this work, a second-order adaptive model was developed to allow for simulation of the formation of a mental model of a trauma that is built up over time and its emotional responses, and neurological processes of how a stimulus can become a trigger to activate this mental model. Furthermore, the influence of therapy on the ability of an individual to control the emotional response to the trauma mental model was explored. The

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computational model was developed following the approach described in [25], using the following steps:

- A conceptual causal network model was designed based on literature on patients with PTSD and existing theories and models about PTSD and emotion regulation
- The conceptual causal network model was translated into role matrices format
- The role matrices were used in the dedicated software environment to obtain simulations; this software environment is available at https://www.researchgate.net/project/Network-Oriented-Modeling-Software.

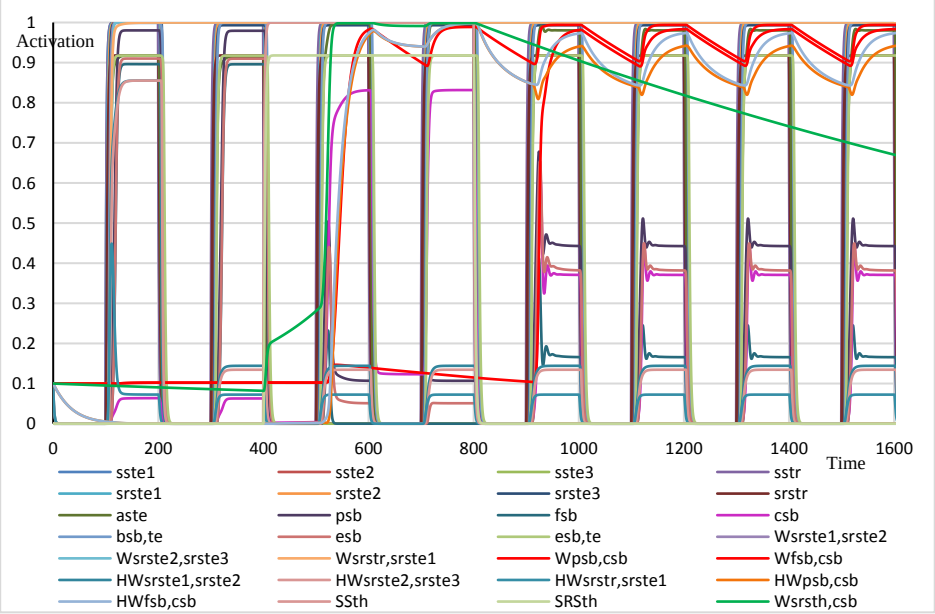

Fig. 3. Development of PTSD and recovery using therapy. Again, the trauma develops from time 100 to 200 and the trigger also occurs from 100 to 200 and after that regularly recurs from 300 to 400, from 500 to 600, et cetera. In this case therapy takes place from time 400 to 800 which leads to recovery.

Different simulation experiments were done, for individuals developing a trigger response, individuals not developing a trigger response, and individuals receiving therapy.

Other work addressing computational modelling for trauma development and recovery can be found in [8, 16-17]. However, none of these previous works allowed for the adaptation of the learnt connections of the mental model and therapy. In addition, in [16-17] it is assumed that already built-in upward connections for the emotion regulation exist and are static, while in the model presented here an important part of the

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development of a trauma is the learning for the mental model of the traumatic course of affairs. In another comparison, [8] addresses social support instead of the type of therapy suggested by Garcia [10] and used in the current paper. Moreover, the underlying second-order adaptation process as explained extensively by [10] is fully addressed here while it is ignored in [8, 16-17]. Finally, in the current paper the source of the trauma can be a process taking place over a longer time period with a successive course of events over time, and modeled in the form of an internal mental model that can be replayed as a flashback movie, while in [8, 16-17] only one traumatic state at one time point is assumed where a flashback is only one static image, which is not quite realistic.

The second-order adaptive model described in this paper can be used as a basis for development of integrated computing applications to support PTSD therapy or to develop virtual characters illustrating the processes involved in patients with PTSD. In such contexts, also possibilities may be exploited for further validation of the model.

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