Chapter 5
Disturbed by Flashbacks: A Controlled Adaptive Network Model Addressing Mental Models for Flashbacks from PTSD

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Abstract In this chapter, 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 · Flashback movie

5.1 Introduction

A Post Traumatic Stress Disorder (PTSD) is usually developed after experiencing an event (often consisting of a sequence of steps) that triggers strong negative emotions like fear; e.g., (Duvarci and Pare 2014; Parsons and Ressler 2013). One of the symptoms is a recurring re-experiencing of the event sequence 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. The occurrence of such flashbacks can be described as a mental model that was learned during the traumatic event sequence and that is replayed by internal (mental) simulation.

In the literature such as (Admon et al. 2013; Akiki et al. 2017; Holmes et al. 2018; Zandvakili et al. 2020) strong evidence can be found for relations to amygdala,

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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., (Admon et al. 2013; Panksepp and Biven 2012). 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., (Fitzgerald et al. 2018; Ochsner and Gross 2014; Webb et al. 2012).

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 event sequence leading to the trauma. This is a form of observational learning; e.g., (Benbassat 2014; Van Gog et al. 2009). It is this learnt mental model that is the basis of the flashback symptoms. Moreover, during development also learning 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., (Brogden 1947; Hall 1996). 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., (Ochsner and Gross 2014; Webb et al. 2012). 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., (Garcia 2002). Metaplasticity (Abraham and Bear 1996) 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 chapter 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 Sect. 5.2 some background knowledge is discussed for the different types of adaptation. Section 5.3 introduces the second-order adaptive network model to address these forms of adaptation. In Sect. 5.4 some example simulations for this network model are discussed. Finally, Sect. 5.5 is a discussion.

5.2 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.

5.2.1 First-Order Adaptation Principle: Hebbian Learning

In neuroscientific literature such as (Chandra and Barkai 2018), 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 (Hebb 1949). It can be explained by:

> 'When an axon of cell A is near enough to excite B and repeatedly or persistently (5.1)
> 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.'
> (Hebb 1949), p. 62

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

> 'What fires together, wires together' (Keysers and Gazzola 2014; Shatz 1992) (5.2)

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 event sequence based on sensory preconditioning (Brogden 1947; Hall 1996).
  - Learning the connections in the mental model of the traumatic event sequence based on observational learning, also using sensory preconditioning (Benbassat 2014; Van Gog et al. 2009).
- Recovery from the trauma:
  - Strengthening emotion regulation for recovery by learning the connections to the prefrontal cortex areas (Ochsner and Gross 2014; Webb et al. 2012).

5.2.2 Second-Order Adaptation Principle: Stress Reduces Adaptation Speed

In (Garcia 2002) 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 (2002) 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.' (Garcia 2002), 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.' (Garcia 2002), 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.' (Garcia 2002), 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 (Garcia 2002)
- 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 (Garcia 2002)

In Sect. 5.3 it will be discussed how these have been modeled by using a so-called self-modeling network model.

5.3 The Second-Order Adaptive 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 (Treur 2016) and further developed to cover higher-order adaptive networks in (Treur 2020a; b), where also the supporting dedicated software environment is presented.

5.3.1 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.

5.3.2 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. 5.1):

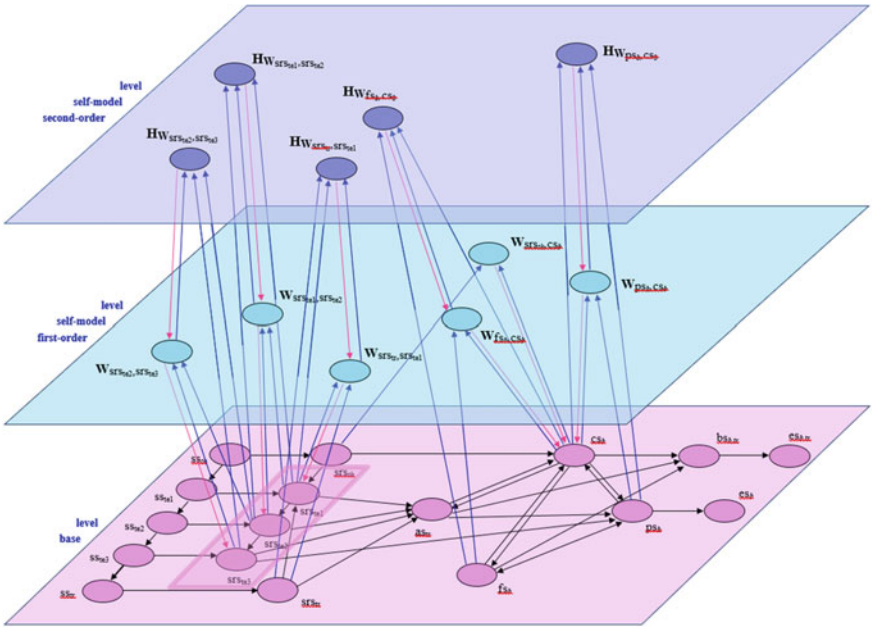

Fig. 5.1 Connectivity of the introduced second-order adaptive network model with the developed mental model for the traumatic course of events highlighted

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. 5.1; Table 5.1 shows the states and brief explanations of them. Within the network model, the first-order adaptation

Table 5.1 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: observation of action te2
X_3 ss_{te3}	Sensor state for traumatic event phase 3: observation of effect te3
X_4 ss_{tr}	Sensor state for trigger tr for the traumatic event sequence te
X_5 ss_{th}	Sensor state for input from therapy *th*
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 event sequence
X_{10} srs_{th}	Sensory representation state for therapy *th*
X_{11} as_{te}	Awareness state for the traumatic event 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 traumatic event te
X_{18} $\mathbf{W}_{srs_{te1},srs_{te2}}$	Representation state for weight of the connection from srs_{te1} to srs_{te2} for imprinting traumatic sequence
X_{19} $\mathbf{W}_{srs_{te2},srs_{te3}}$	Representation state for weight of the connection from srs_{te2} to srs_{te3} for imprinting traumatic sequence
X_{20} $\mathbf{W}_{srs_{tr},srs_{te1}}$	Representation state for weight of the connection from srs_{tr} to srs_{te1} for sensory preconditioning to link trigger tr to the traumatic sequence
X_{21} $\mathbf{W}_{ps_b,cs_b}$	Representation state for weight of the 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 the connection from fs_b to cs_b for learning of emotion regulation
X_{23} $\mathbf{W}_{th,cs_b}$	Representation state for weight of the 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

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 Sect. 5.2, for the self-model's connectivity, incoming connections from X and Y to $\mathbf{W}_{X,Y}$ are used; see Fig. 5.1 (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. 5.1 (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 5.4.

The sensing of an example of a traumatic event te in the form of a sequence of steps is modeled by the sensor states $\mathrm{ss}_{\mathrm{te1}}$, $\mathrm{ss}_{\mathrm{te2}}$, $\mathrm{ss}_{\mathrm{te3}}$. For example, te1 (or traumatic event phase 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 $\mathrm{srs}_{\mathrm{te1}}$, $\mathrm{srs}_{\mathrm{te2}}$, $\mathrm{srs}_{\mathrm{te3}}$ are activated for these phases 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 event is formed and represented by base states $\mathrm{srs}_{\mathrm{te1}}$, $\mathrm{srs}_{\mathrm{te2}}$, $\mathrm{srs}_{\mathrm{te3}}$ and their connections (see the small pink parallelogram within the base plane in Fig. 5.1) with first-order self-model states $\mathbf{W}_{\mathrm{srs}_{\mathrm{te1}},\mathrm{srs}_{\mathrm{te2}}}$ and $\mathbf{W}_{\mathrm{srs}_{\mathrm{te2}},\mathrm{srs}_{\mathrm{te3}}}$. Similarly, the connection between the sensory representations of the trigger tr and the traumatic event sequence is learnt based on sensory preconditioning, represented by $\mathbf{W}_{\mathrm{srs}_{\mathrm{tr}},\mathrm{srs}_{\mathrm{te1}}}$. These newly formed connections activate the mental model as a form of internal mental simulation, every time the trigger is sensed. For the traumatized person this shows as an internal flashback movie of the traumatic sequence. In turn, this flashback movie activates the related negative emotions experienced at the original traumatic event.

In contrast to what was believed earlier, such learnt connections usually do not show any form of natural extinction; e.g., (Levin and Nielsen 2007), 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 $\mathbf{H}_{\mathbf{W}_{\mathrm{ps}_b,\mathrm{cs}_b}}$ and $\mathbf{H}_{\mathbf{W}_{\mathrm{fs}_b,\mathrm{cs}_b}}$ are very low. Therefore, without any additional help the situation will stay as it is. But, following (Garcia 2002) the therapy *th* is able to temporarily reduce the level of negative emotions, so that $\mathbf{H}_{\mathbf{W}_{\mathrm{ps}_b,\mathrm{cs}_b}}$ and $\mathbf{H}_{\mathbf{W}_{\mathrm{fs}_b,\mathrm{cs}_b}}$ get higher values. Due to this, learning of the connections to the control state takes place: $\mathbf{W}_{\mathrm{ps}_b,\mathrm{cs}_b}$ and $\mathbf{W}_{\mathrm{fs}_b,\mathrm{cs}_b}$ get higher values.

5.3.3 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 5.2 and 5.3.

Table 5.2 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 $X \rightarrow Y$
$\mathbf{H}_{\mathbf{W}_{X,Y}}$	Learning control state for the connection weight state for connection $X \rightarrow Y$

Table 5.3 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
$\mathbf{W}_{X,Y} \rightarrow \mathbf{H}_{\mathbf{W}_{X,Y}}$ $X \rightarrow \mathbf{H}_{\mathbf{W}_{X,Y}}$ $Y \rightarrow \mathbf{H}_{\mathbf{W}_{X,Y}}$	Connections from connection adaptation state $\mathbf{W}_{X,Y}$ and base level states X and Y to learning control state $\mathbf{H}_{\mathbf{W}_{X,Y}}$	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
$\mathbf{H}_{\mathbf{W}_{X,Y}} \rightarrow \mathbf{W}_{X,Y}$	Connection from learning control state $\mathbf{H}_{\mathbf{W}_{X,Y}}$ to adaptive connection adaptation state $\mathbf{W}_{X,Y}$ to effectuate learning control	Interlevel connection, downward from the third level to the second level

Table 5.4 The combination functions used from the library

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

The model with connectivity shown in Fig. 5.1 was then specified by tables in role matrix format: Connectivity characteristics (1), aggregation characteristics (2) and timing characteristics (3); see Sect. 5.6. Four different combination functions from the library are used that each serve a different purpose; see Table 5.4.

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 σ and threshold τ. 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).

5.4 Example Simulations

The role matrices listed in the Appendix Sect. 5.6 can easily be transferred to the dedicated software 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 $\Delta t = 0.5$) and provides as output a simulation graph for the model. In Fig. 5.2 the development of PTSD is shown based on traumatic event phases 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. 5.3 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 Figs. 5.2 and 5.3 in the time period from 100 to 200 the traumatic event sequence te1 to te3 in the world are sensed (via sensor states ss_{te1}, ss_{te2}, ss_{te3}) of which internal representations srs_{te1}, srs_{te2}, srs_{te3} are made. Due to sensory preconditioning (first-order adaptation based on Hebbian learning), the connections between them are developed (thus forming a mental model of the traumatic event sequence) and also a connection from the

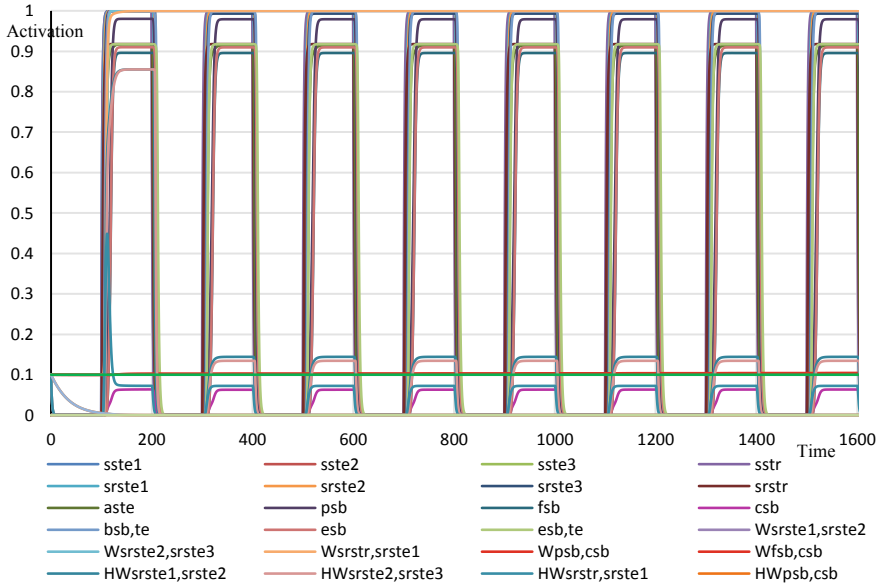

Fig. 5.2 Development of PTSD without using therapy. The trauma develops from time 100 to 200. The trigger also occurs from 100 to 200 and after that regularly recurs from 300 to 400, from 500 to 600, et cetera. No recovery from PTSD takes place

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_{te2}, srs_{te3}, the flasback movie is replayed (as a form of internal simulation of the mental model) and because of that the negative emotion and feeling are activated to high values again.

In Fig. 5.3 it is shown how the therapy temporarily (from time 400 to 800) reduces the negative emotion and feeling due to which the adaptation speeds (represented by the **H**-states) for the connections to control state cs_b increase again (second-order adaptation for metaplasticity) and therefore the learning of these connections to cs_b takes place. This finally results in much lower activations of the emotion and feeling states due to the strengthened emotion regulation.

5.5 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

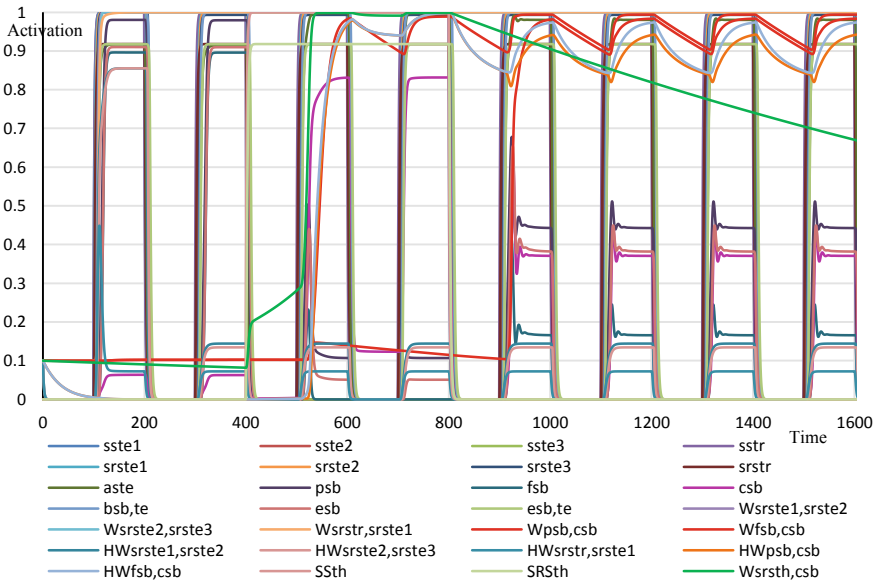

Fig. 5.3 Development of PTSD and recovery using therapy. Again, the trauma develops from time 100 to 200 and the trigger also occurs from 100 to 200 and after that regularly recurs from 300 to 400, from 500 to 600, et cetera. In this case therapy takes place from time 400 to 800 which leads to recovery

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. Most of the material was adopted from (Van Ments and Treur 2021). The computational model was developed following the approach described in (Treur 2020b), 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.

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 (Formolo et al. 2017; Naze and Treur 2011; Naze and Treur 2012). However, none of these previous works allowed for the adaptation of the learnt connections of the mental model and therapy. In addition, in (Naze

and Treur 2011,2012) 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 development of a trauma is the learning for the mental model of the traumatic course of affairs. In another comparison, (Formolo et al. 2017) addresses social support instead of the type of therapy suggested by Garcia (2002) and used in the current chapter. Moreover, the underlying second-order adaptation process as explained extensively by (Garcia 2002) is fully addressed here while it is ignored in (Formolo et al. 2017; Naze and Treur 2011; Naze and Treur 2012). Finally, in the current chapter 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 (Formolo et al. 2017; Naze and Treur 2011; Naze and Treur 2012) 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 chapter 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.

5.6 Appendix: Full Specification of the Adaptive Network Model

In this section, the full specification of the model is provided in terms of role matrices, which is the standard format used for design of a model and which can also be used as input for the software environment. Each role matrix has 3 color blocks that match the colors in the graphical representation used earlier: pink for the base level, blue for the first-order self-model states and purple for the second-order self-model states.

Connectivity characteristics role matrices.

The first role matrix **mb** (see Fig. 5.4, left hand side) represents the base connections between all the states, as presented in the graphical representation in Fig. 5.1. For example, row 6 with state srs_{te1} (also indicated by X_6) has two incoming connection: one from ss_{te1} and one from srs_{tr}. Role matrix **mcw** (see Fig. 5.4, right hand side) shows the weights $\boldsymbol{\omega}$ of the connections presented in role matrix **mb**. There is a difference in nonadaptive (green) and adaptive connections (peach-red). For example, again row 6 with state srs_{te1} with two incoming connections: one non adaptive from ss_{te1} with connection weight 1 and one adaptive connection from srs_{tr}, presented by state X_{20}, i.e. $\mathbf{W}_{srs_{tr},srs_{te1}}$ (a first-order self-model state).

mb base connectivity		1	2	3	4	5
X_1	ss_{te1}	X_1				
X_2	ss_{te2}	X_1				
X_3	ss_{te3}	X_2				
X_4	ss_{tr}	X_4				
X_5	ss_{th}	X_5				
X_6	srs_{te1}	X_1	X_9			
X_7	srs_{te2}	X_2	X_6			
X_8	srs_{te3}	X_3	X_7			
X_9	srs_{tr}	X_4				
X_{10}	srs_{th}	X_5				
X_{11}	as_{te}	X_6	X_7	X_8	X_9	X_{14}
X_{12}	ps_b	X_8	X_{11}	X_{13}	X_{14}	
X_{13}	fs_b	X_{12}	X_{14}			
X_{14}	cs_b	X_8	X_{11}	**X_{10}**	**X_{12}**	**X_{13}**
X_{15}	$bs_{b,te}$	X_8	X_{11}	X_{13}		
X_{16}	es_b	X_{12}				
X_{17}	$es_{b,te}$	X_{15}				
X_{18}	$\mathbf{W}_{srs_{te1},srs_{te2}}$	X_6	X_7	X_{18}		
X_{19}	$\mathbf{W}_{srs_{te2},srs_{te3}}$	X_7	X_8	X_{19}		
X_{20}	$\mathbf{W}_{srs_{tr},srs_{te1}}$	X_9	X_6	X_{20}		
X_{21}	$\mathbf{W}_{ps_b,cs_b}$	X_{12}	X_{14}	X_{21}		
X_{22}	$\mathbf{W}_{fs_b,cs_b}$	X_{13}	X_{14}	X_{22}		
X_{23}	$\mathbf{W}_{th,cs_b}$	X_{15}	X_{14}	X_{23}		
X_{24}	$\mathbf{Hw}_{srs_{te1},srs_{te2}}$	X_7	X_8	X_{18}	X_{24}	
X_{25}	$\mathbf{Hw}_{srs_{te2},srs_{te3}}$	X_6	X_7	X_{19}	X_{25}	
X_{26}	$\mathbf{Hw}_{srs_{tr},srs_{te1}}$	X_9	X_6	X_{20}	X_{26}	
X_{27}	$\mathbf{Hw}_{ps_b,cs_b}$	X_{12}	X_{14}	X_{21}	X_{27}	
X_{28}	$\mathbf{Hw}_{fs_b,cs_b}$	X_{13}	X_{14}	X_{22}	X_{28}	

mcw connection weights		1	2	3	4	5
X_1	ss_{te1}	1				
X_2	ss_{te2}	1				
X_3	ss_{te3}	1				
X_4	ss_{tr}	1				
X_5	ss_{th}	1				
X_6	srs_{te1}	1	X_{20}			
X_7	srs_{te2}	1	X_{18}			
X_8	srs_{te3}	1	X_{19}			
X_9	srs_{tr}	1				
X_{10}	srs_{th}	1				
X_{11}	as_{te}	1	1	1	1	1
X_{12}	ps_b	1	1	-0.5	1	
X_{13}	fs_b	1	-0.5			
X_{14}	cs_b	0.3	0.3	X_{23}	X_{21}	X_{22}
X_{15}	$bs_{b,te}$	1	1			
X_{16}	es_b	1				
X_{17}	$es_{b,te}$	1				
X_{18}	$\mathbf{W}_{srs_{te1},srs_{te2}}$	1	1	1		
X_{19}	$\mathbf{W}_{srs_{te2},srs_{te3}}$	1	1	1		
X_{20}	$\mathbf{W}_{srs_{tr},srs_{te1}}$	1	1	1		
X_{21}	$\mathbf{W}_{ps_b,cs_b}$	1	1	1		
X_{22}	$\mathbf{W}_{fs_b,cs_b}$	1	1	1		
X_{23}	$\mathbf{W}_{th,cs_b}$	1	1	1		
X_{24}	$\mathbf{Hw}_{srs_{te1},srs_{te2}}$	1	1	-0.5	1	
X_{25}	$\mathbf{Hw}_{srs_{te2},srs_{te3}}$	1	1	-0.5	1	
X_{26}	$\mathbf{Hw}_{srs_{tr},srs_{te1}}$	1	1	-0.5	1	
X_{27}	$\mathbf{Hw}_{ps_b,cs_b}$	-0.5	1	0	1	
X_{28}	$\mathbf{Hw}_{fs_b,cs_b}$	-0.5	1	0	1	

Fig. 5.4 Connectivity characteristics: role matrices **mb** and **mcw**

Aggregation characteristics role matrices.

In order to transform the presented graphical model with states and connections into a numerical model each state needs a combination function $\mathbf{c}_Y(..)$ to aggregate the impacts of other states on state Y. Role matrix **mcfw** (see Fig. 5.5) specifies which combination function is used for each state. For example, state ss_{te1} uses the steponce function, so this is indicated with a combination function weight $\boldsymbol{\gamma}$ of 1 in the fourth column.

Role matrix **mcfp** (see Fig. 5.6) defines the exact parameters used for each state and function. The parameters used depend on the combination function.

Timing characteristics role matrix and initial values.

The speed factors $\boldsymbol{\eta}_Y$ are specified in role matrix **ms;** see Fig. 5.7. Concerning the initial values, the simulated scenario starts with most state values 0. The learning states have initial value >0, chosen at 0.1.

mcfw combination function weights		1 alogistic	2 hebb	3 stepmod	4 steponce
X_1	ss_{te1}				1
X_2	ss_{te2}	1			
X_3	ss_{te3}	1			
X_4	ss_{tr}			1	
X_5	ss_{th}				1
X_6	srs_{te1}	1			
X_7	srs_{te2}	1			
X_8	srs_{te3}	1			
X_9	srs_{th}	1			
X_{10}	srs_{tr}	1			
X_{11}	as_{te}	1			
X_{12}	ps_b	1			
X_{13}	fs_b	1			
X_{14}	cs_b	1			
X_{15}	$bs_{b,te}$	1			
X_{16}	es_b	1			
X_{17}	$es_{b,te}$	1			
X_{18}	$\mathbf{W}_{srs_{te1},srs_{te2}}$		1		
X_{19}	$\mathbf{W}_{srs_{te2},srs_{te3}}$		1		
X_{20}	$\mathbf{W}_{srs_{tr},srs_{te1}}$		1		
X_{21}	$\mathbf{W}_{ps_b,cs_b}$		1		
X_{22}	$\mathbf{W}_{fs_b,cs_b}$		1		
X_{23}	$\mathbf{W}_{th,cs_b}$		1		
X_{24}	$\mathbf{Hw}_{srste1,srste2}$	1			
X_{25}	$\mathbf{Hw}_{srste2,srste3}$	1			
X_{26}	$\mathbf{Hw}_{srstr,srste1}$	1			
X_{27}	$\mathbf{Hw}_{psb,csb}$	1			
X_{28}	$\mathbf{Hw}_{fsb,csb}$	1			

Fig. 5.5 Aggregation characteristics: role matrix **mcfw**

mcfp	combination function parameters	1 alogistic σ	τ	2 hebb μ	3 stepmod ρ	δ	4 steponce α	β
X_1	ss_{te1}						100	200
X_2	ss_{te2}	20	0.5					
X_3	ss_{te3}	20	0.5					
X_4	ss_{tr}				200	100		
X_5	ss_{th}	5	0.5				400	1600
X_6	srs_{te1}	20	0.5					
X_7	srs_{te2}	20	0.5					
X_8	srs_{te3}	10	0.5					
X_9	srs_{tr}	5	0.5					
X_{10}	srs_{th}	5	0.5					
X_{11}	as_{te}	5	0.5					
X_{12}	ps_b	5	0.5					
X_{13}	fs_b	5	0.5					
X_{14}	cs_b	5	0.5					
X_{15}	$bs_{b,te}$	5	0.5					
X_{16}	es_b	5	0.5					
X_{17}	$es_{b,te}$	5	0.5					
X_{18}	$\mathbf{W}_{srs_{te1},srs_{te2}}$			1				
X_{19}	$\mathbf{W}_{srs_{te2},srs_{te3}}$			1				
X_{20}	$\mathbf{W}_{srs_{tr},srs_{te1}}$			1				
X_{21}	$\mathbf{W}_{ps_b,cs_b}$			0.999				
X_{22}	$\mathbf{W}_{fs_b,cs_b}$			0.999				
X_{23}	$\mathbf{W}_{th,cs_b}$			0.909				
X_{24}	$\mathbf{H}_{\mathbf{W}srste1,srste2}$	5	2					
X_{25}	$\mathbf{H}_{\mathbf{W}srste2,srste3}$	5	2					
X_{26}	$\mathbf{H}_{\mathbf{W}srstr,srste1}$	5	2					
X_{27}	$\mathbf{H}_{\mathbf{W}psb,csb}$	5	2					
X_{28}	$\mathbf{H}_{\mathbf{W}fsb,csb}$	5	2					

Fig. 5.6 Aggregation characteristics: role matrix **mcfp**

ms speed factors		1	initial values		
X_1	ss_{te1}	0.5	X_1	ss_{te1}	0
X_2	ss_{te2}	0.5	X_2	ss_{te2}	0
X_3	ss_{te3}	0.5	X_3	ss_{te3}	0
X_4	ss_{tr}	0.5	X_4	ss_{tr}	0
X_5	ss_{th}	0.5	X_5	ss_{th}	0
X_6	srs_{te1}	0.5	X_6	srs_{te1}	0
X_7	srs_{te2}	0.5	X_7	srs_{te2}	0
X_8	srs_{te3}	0.5	X_8	srs_{te3}	0
X_9	srs_{tr}	0.5	X_9	srs_{tr}	0
X_{10}	srs_{th}	0.5	X_{10}	srs_{th}	0
X_{11}	as_{te}	0.5	X_{11}	as_{te}	0
X_{12}	ps_b	0.5	X_{12}	ps_b	0
X_{13}	fs_b	0.5	X_{13}	fs_b	0
X_{14}	cs_b	0.5	X_{14}	cs_b	0
X_{15}	$bs_{b,te}$	0.5	X_{15}	$bs_{b,te}$	0
X_{16}	es_b	0.5	X_{16}	es_b	0
X_{17}	$es_{b,te}$	0.5	X_{17}	$es_{b,te}$	0
X_{18}	$\mathbf{W}_{srs_{te1},srs_{te2}}$	X_{24}	X_{18}	$\mathbf{W}_{srs_{te1},srs_{te2}}$	0.1
X_{19}	$\mathbf{W}_{srs_{te2},srs_{te3}}$	X_{25}	X_{19}	$\mathbf{W}_{srs_{te2},srs_{te3}}$	0.1
X_{20}	$\mathbf{W}_{srs_{tr},srs_{te1}}$	X_{26}	X_{20}	$\mathbf{W}_{srs_{tr},srs_{te1}}$	0.1
X_{21}	$\mathbf{W}_{ps_b,cs_b}$	X_{27}	X_{21}	$\mathbf{W}_{ps_b,cs_b}$	0.1
X_{22}	$\mathbf{W}_{fs_b,cs_b}$	X_{28}	X_{22}	$\mathbf{W}_{fs_b,cs_b}$	0.1
X_{23}	$\mathbf{W}_{th_b,cs_b}$	0.5	X_{23}	$\mathbf{W}_{th_b,cs_b}$	0.1
X_{24}	$\mathbf{Hw}_{srste1,srste2}$	0.5	X_{24}	$\mathbf{Hw}_{srste1,srste2}$	0.1
X_{25}	$\mathbf{Hw}_{srste2,srste3}$	0.5	X_{25}	$\mathbf{Hw}_{srste2,srste3}$	0.1
X_{26}	$\mathbf{Hw}_{srstr,srste1}$	0.5	X_{26}	$\mathbf{Hw}_{srstr,srste1}$	0.1
X_{27}	$\mathbf{Hw}_{psb,csb}$	0.05	X_{27}	$\mathbf{Hw}_{psb,csb}$	0.1
X_{28}	$\mathbf{Hw}_{fsb,csb}$	0.05	X_{28}	$\mathbf{Hw}_{fsb,csb}$	0.1

Fig. 5.7 Timing characteristics: role matrix **ms** and initial values

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