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Desensitization Due to Overstimulation: A Second-Order Adaptive Network Model

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Abstract. In this paper, a second-order adaptive network model is presented for the effects of supernormal stimuli. The model describes via the underlying mechanisms in the brain how responses on stimuli occur in absence of a supernormal stimulus. Moreover, it describes how these normal responses are lost after a supernormal stimulus occurs and due to that adaptive desensitization takes place. By simulated example scenarios, it was evaluated that the model describes the expected dynamics. By stationary point analysis correctness of the implemented model with respect to its design was verified.

Keywords: Overstimulation · Desensitization · Adaptive network model

1 Introduction

Supernormal stimuli are everywhere. They can be seen on billboards, you see them on TV, and you can even find them when you take a walk in the forest. Supernormal stimuli differ from normal stimuli. A stimulus is a change in the external or internal environment to which an organism reacts. This reaction is also referred to as a response. A supernormal stimulus is an extreme version of a particular stimulus that people already have a tendency to respond to. This extreme version of an already existing stimulus creates a stronger response than the regular stimulus does. These supernormal stimuli are found both in the animal kingdom and in the human world. Cuckoo birds are a good example of this [1]. Cuckoo birds lay their eggs in other birds' nests so that these other birds feed their offspring. But why do these other birds feed the cuckoo chicks and not their own? This is due to the supernormal stimuli of the cuckoo chicks, which have a larger and redder beak than the other chicks. These supernormal stimuli of the cuckoo chick produce a stronger response of the parent birds than the stimuli of their own chicks do.

Such effects of supernormal stimuli are explored computationally in this paper. First, in Sect. 2 some related background knowledge is briefly discussed and the research question is formulated. Next, in Sect. 3 the modeling approach used is discussed. In Sect. 4 the introduced computational model is described in some detail. Section 5 illustrates the model by simulation experiments performed. In Sect. 6 verification of the implemented model with respect to its design description based on analysis of stationary points is discussed. Finally, Sect. 7 is a discussion.

2 Background: Related Work and Research Question

Superstimuli often exist in the human world and companies such as advertising agencies make grateful use of this phenomenon. People are triggered and seduced by commercials filled with astonishing landscapes, beautiful people and delicious-looking food, which are often an exaggeration of reality. Because these stimuli are often over-exaggerated, turning into supernormal stimuli, people react more strongly to this, but they will never be completely satisfied with the purchased product, and they will look for new products to satisfy their buying drive [5]. This phenomenon is known as *desensitization*.

As a general principle, after prolonged or multiple exposure to a particular stimulus or stimuli, the brain will adapt and the brain becomes less sensitive to this stimulus; for example, homeostatic excitability regulation [7, 11] refers to this principle. This principle is also reflected in the brain's dopamine release. Dopamine is a substance that is part of the brain's reward system, as a neurotransmitter. These are chemicals in the brain that support transfer of information from one nerve cell to another. Dopamine makes one feel satisfied and rewarded. Dopamine is not constantly produced, but is released during certain actions or situations such as eating, exercising, sex, or drugs. Dopamine is also released when people make purchases. In line with the principle described here, when the brain is exposed to a particular stimulus in a prolonged way, or when the brain is multiple times exposed to a particular stimulus, less dopamine is released for experiencing the same amount of stimuli. In other words, the brain desensitizes due to overstimulation. To experience the same effect, people will have to be exposed to these stimuli for longer, or they have to be exposed more often to these stimuli. Our research question derives from this:

Can mechanisms in the brain indeed explain desensitization as a result of exposure to supernormal stimuli?

3 The Network-Oriented Modeling Approach Used

In this part of the paper the modeling approach used is explained. Traditionally, modelling the world's processes often is based on isolation and separation assumptions. These generally serve as a means to reduce the complexity of the problems. For example, in physics concerning gravity, usually the force from a planet on an object is taken into account, but not all other forces in the universe acting upon this object. Sometimes these assumptions do not hold. For example, Aristotle considered that some internal processes are separated from the body [3]. Later Kim [6] and others from philosophy of mind disputed that the mind and the body could be separated.

However, the problem with modeling such processes is not a particular type of isolation or separation assumption, but with the notion of separation itself. Interaction between states in a model often need cyclic dependencies which make separation assumptions difficult to apply. In philosophy of the mind, the idea is that mental states are caused by input states and other mental states, and some mental states influence outgoing states [6]. This takes place according to a cyclic causal network of mental states within which each mental state has a causal, or functional role; e.g., see:

‘Mental events are conceived as nodes in a complex causal network that engages in causal transactions with the outside world by receiving sensory inputs and emitting behavioral outputs. (...) to explain what a given mental state is, we need to refer to other mental states, and explaining these can only be expected to require reference to further mental states, on so on – a process that can go on in an unending regress, or loop back in a circle.’ [6], pp. 104–105.

The network-oriented modeling approach described in [9] follows this dynamic perspective on causality and has been applied to obtain the network model introduced here. Within this approach, at each given time t each node (also called state) Y of such a network has a state value $Y(t)$, a real number usually in the interval $[0,1]$. The approach uses the following *network characteristics* to describe network models (called temporal-causal networks):

- **Connectivity characteristics**

Each connection from a state X to another state Y has a connection weight $\omega_{X,Y}$ which represents the strength of the connection

- **Aggregation characteristics**

Each state Y has a combination function $c_Y(\dots)$ which is used to combine the single causal impacts $\omega_{X,Y}X(t)$ from different states X on state Y ; for selection of combination functions from the available library, weights $\gamma_{i,Y}$ are used and the combination functions usually have parameters that can be indicated in general by $\pi_{i,j,Y}$

- **Timing characteristics**

Each state Y has a speed factor η_Y for the speed of change of Y defining how fast the state changes upon its causal impact.

The choice of combination functions $c_Y(\dots)$ can be problem-dependent. Within the available dedicated software environment, they can be chosen from a library for them, but can also be easily added. The combination function library, which has over 40 combination functions, also includes a facility to compose new combination functions from the available ones by (mathematical) function composition. The combination functions that have been used in this paper are basic; they are shown in Table 1.

Using these network characteristics, a network model can be simulated in a manner described in [9] as follows:

- For each time point t the aggregated impact is calculated using the combination function $c_Y(\dots)$ by

$$\mathbf{aggimpact}_Y(t) = c_Y(\omega_{X_1,Y}X_1(t), \dots, \omega_{X_k,Y}X_k(t)) \quad (1)$$

- Then for the time step from t to $t + \Delta t$ for each state Y the value is adjusted using the aggregated impact and the speed factor

$$Y(t + \Delta t) = Y(t) + \eta_Y[\mathbf{aggimpact}_Y(t) - Y(t)]\Delta t \quad (2)$$

- Hence the following differential equation is obtained:

$$\frac{dY(t)}{dt} = \eta_Y[\mathbf{aggimpact}_Y(t) - Y(t)]$$

Table 1. Basic combination functions from the library used in the presented model

	Notation	Formula	Parameters
Advanced logistic sum	alogistic $_{\sigma,\tau}(V_1, \dots, V_k)$	$\left[\frac{1}{1+e^{-\sigma(V_1+\dots+V_k-\tau)}} - \frac{1}{1+e^{\sigma\tau}} \right] (1 + e^{-\sigma\tau})$	Steepness $\sigma > 0$ Excitability threshold τ
Hebbian learning	hebb $_{\mu}(V_1, V_2, W)$	$V_1 V_2 (1 - W) + \mu W$	Persistence factor $\mu > 0$
Stepmod	stepmod $_{\rho,\delta}(V_1, \dots, V_k)$	0 if $t \bmod \rho < \delta$, else 1	Repetition ρ Duration of 0 δ

To model adaptive networks, the notion of *self-modeling network* (also called *reified network*) is applied, where the adaptive network characteristics are represented within the network by additional network states, called *self-model* states; see [10]. Shortly, adding a self-model for a temporal-causal network is done in the way that for some of the states Y of the base network and some of its related network structure characteristics for connectivity, aggregation and timing (in particular, some from $\omega_{X,Y}$, $\gamma_{i,Y}$, $\pi_{i,j,Y}$, η_Y), additional network states $\mathbf{W}_{X,Y}$, $\mathbf{C}_{i,Y}$, $\mathbf{P}_{i,j,Y}$, \mathbf{H}_Y (self-model states) are introduced:

(a) **Connectivity self-model**

- Self-model states $\mathbf{W}_{X_i,Y}$ are added representing connectivity characteristics, in particular connection weights $\omega_{X_i,Y}$

(b) **Aggregation self-model**

- Self-model states $\mathbf{C}_{j,Y}$ are added representing aggregation characteristics, in particular combination function weights $\gamma_{i,Y}$
- Self-model states $\mathbf{P}_{i,j,Y}$ are added representing aggregation characteristics, in particular combination function parameters $\pi_{i,j,Y}$

(c) **Timing self-model**

- Self-model states \mathbf{H}_Y are added representing timing characteristics, in particular speed factors η_Y

Here \mathbf{W} refers to ω , \mathbf{C} refers to γ , \mathbf{P} refers to π , and \mathbf{H} refers to η , respectively. For the processing, these self-model states define the dynamics of state Y in a canonical manner according to Eq. (2) whereby $\omega_{X,Y}$, $\gamma_{i,Y}$, $\pi_{i,j,Y}$, η_Y are replaced by the state values of $\mathbf{W}_{X,Y}$, $\mathbf{C}_{i,Y}$, $\mathbf{P}_{i,j,Y}$, \mathbf{H}_Y at time t , respectively. These special effects are defined by outgoing connections of these self-model states, which give them their specific roles. Moreover, as for any other state in the network, the incoming connections and other network characteristics relating to the self-model states (including their combination functions) give them their dynamics.

As the outcome of the addition of a self-model is also a temporal-causal network model itself, as has been shown in detail in [10], Ch 10, this construction can easily be applied iteratively to obtain multiple levels or orders of self-models.

4 The Adaptive Network Model for Desensitization Due to Overstimulation

The proposed network model is based on a couple of assumptions. Firstly, dopamine is used for the reward system for humans, but when exposed too much the human brain becomes less receptive to the stimuli [5]. The second assumption which was used is that advertisements often do try to trigger emotions such that people tend to get a positive emotion associated to the stimulus [5]. Within the introduced network model, three adaptation principles are applied to the network, two first-order adaptation principles (Hebbian learning and Excitability modulation) and one second-order adaptation principle (Exposure accelerates adaptation). They are as follows.

- **Hebbian learning**

When a cell is repetitive and persistent in activating another cell, there will be a metabolic process making that this cell’s influence on the other cell increases [4]. For example, this can increase the preparation for an action when the stimulus representation affects the preparation of an action.

- **Excitability modulation**

Depending on activation, excitability thresholds are adapted as a form of regulation of neuronal excitability, which to some extent is similar to the adaptation of neurons’ internal properties according to ‘homeostatic regulation’ to guarantee a preferred level of activation; e.g., [7, 11].

- **Exposure accelerates adaptation**

When the exposure to a stimulus becomes stronger, the adaptation speed will increase. When applied to Hebbian learning, this will make the connection change faster when exposed for some time [8].

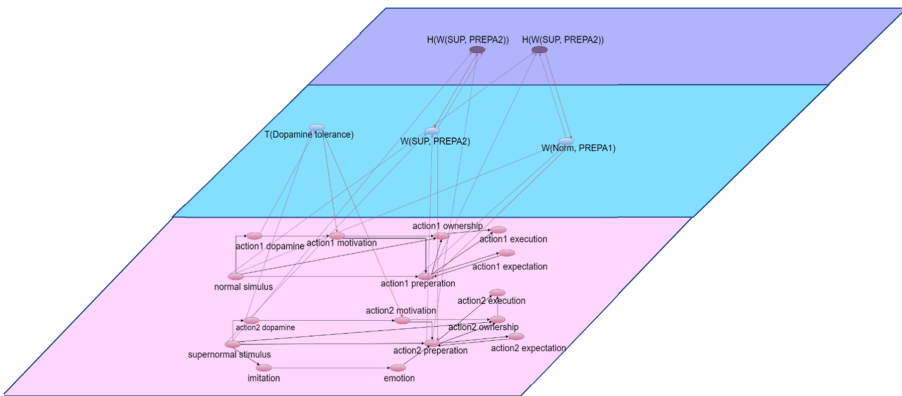


Fig. 1. The connectivity of the introduced adaptive network model

As another principle, ownership of an action makes that the action activation will be performed more frequently [2]. Using these principles as building blocks, the network model has been designed with connectivity as visualized in Fig. 1; see also the explanations of the states in Table 2. The exact dynamics of the model have been defined in the role matrices specifications shown in Figs. 2 and 3.

Table 2. Nomenclature: explanation of the states

State	Explanation
X ₁ srs _{normal}	Sensory representation state for normal stimulus
X ₂ dopamine _{a1}	Dopamine level related to action a ₁
X ₃ mot _{a1}	Motivation level related to action a ₁
X ₄ prep _{a1}	Preparation state for action a ₁
X ₅ exe _{a1}	Execution state for action a ₁
X ₆ exp _{a1}	Expectation (expected effect) for action a ₁
X ₇ own _{a1}	Ownership state for action a ₁
X ₈ srs _{supernormal}	Sensory representation state for supernormal stimulus
X ₉ dopamine _{a2}	Dopamine level related to action a ₂
X ₁₀ imitation	Imitation for supernormal stimulus
X ₁₁ emotion	Emotion for supernormal stimulus
X ₁₂ mot _{a2}	Motivation level related to action a ₂
X ₁₃ prep _{a2}	Preparation state for action a ₂
X ₁₄ exe _{a2}	Execution state for action a ₂
X ₁₅ exp _{a2}	Expectation (expected effect) for action a ₂
X ₁₆ own _{a2}	Ownership state for action a ₂
X ₁₇ T _{dopetolerance}	First-order self-model state for tolerance for dopamine
X ₁₈ W _{srs_{supernormal}, prep_{a2}}	First-order self-model state for the weight of the connection from srs _{supernormal} to prep _{a2}
X ₁₉ W _{srs_{normal}, prep_{a1}}	First-order self-model state for the weight of the connection from srs _{normal} to prep _{a1}
X ₂₀ Hw _{srs_{supernormal}, prep_{a2}}	Second-order self-model state for the adaptation speed of the weight of the connection from srs _{supernormal} to prep _{a2}
X ₂₁ Hw _{srs_{normal}, prep_{a1}}	Second-order self-model state for the adaptation speed of the weight of the connection from srs _{normal} to prep _{a1}

In role matrix **mb**, each state has its own row in which the other states that have effect on it are listed. In role matrix **mcw**, in the corresponding row the connections weights for these connections are specified. Note that for an adaptive connection weight, this is indicated by the name of the self-model state representing this connection weight (here X₁₈ and X₁₉ in the rows for the preparation states prep_{a1} and prep_{a2}).

mb		base connectivity				mcw		connection weights			
		1	2	3	4			1	2	3	4
X ₁	srs _{normal}	X ₁				X ₁	srs _{normal}	1			
X ₂	dopamine _{a1}	X ₁				X ₁	dopamine _{a1}	1			
X ₃	mot _{a1}	X ₂				X ₂	mot _{a1}	1			
X ₄	prep _{a1}	X ₁	X ₃	X ₆		X ₄	prep _{a1}	X ₁₈	0.5	0.5	
X ₅	exe _{a1}	X ₄	X ₇			X ₅	exe _{a1}	0.5	0.5		
X ₆	exp _{a1}	X ₄				X ₆	exp _{a1}	1			
X ₇	own _{a1}	X ₁	X ₃	X ₄		X ₇	own _{a1}	0.33	0.33	0.33	
X ₈	srs _{supernormal}	X ₈				X ₈	srs _{supernormal}	1			
X ₉	dopamine _{a2}	X ₈				X ₉	dopamine _{a2}	1			
X ₁₀	imitation	X ₈				X ₁₀	imitation	1			
X ₁₁	emotion	X ₁₀				X ₁₁	emotion	1			
X ₁₂	mot _{a2}	X ₉	X ₁₁			X ₁₂	mot _{a2}	1	1		
X ₁₃	prep _{a2}	X ₈	X ₁₂	X ₁₅		X ₁₃	prep _{a2}	X ₁₉	1	1	
X ₁₄	exe _{a2}	X ₁₃	X ₁₆			X ₁₄	exe _{a2}	1	1		
X ₁₅	exp _{a2}	X ₁₃				X ₁₅	exp _{a2}	1			
X ₁₆	own _{a2}	X ₈	X ₁₁	X ₁₂	X ₁₃	X ₁₆	own _{a2}	1	1	1	1
X ₁₇	T _{dopetolerance}	X ₂	X ₉	X ₁₇		X ₁₇	T _{dopetolerance}	1	1	1	
X ₁₈	W _{srs_{supernormal},prep_{a2}}	X ₈	X ₁₃	X ₁₈		X ₁₈	W _{srs_{supernormal},prep_{a2}}	1	1	1	
X ₁₉	W _{srs_{normal},prep_{a1}}	X ₁	X ₄	X ₁₉		X ₁₉	W _{srs_{normal},prep_{a1}}	1	1	1	
X ₂₀	H _{w_{srs_{supernormal},prep_{a2}}}	X ₈	X ₁₃	X ₁₈	X ₂₀	X ₂₀	H _{w_{srs_{supernormal},prep_{a2}}}	1	1	1	1
X ₂₁	H _{w_{srs_{normal},prep_{a1}}}	X ₁	X ₄	X ₁₉	X ₂₁	X ₂₁	H _{w_{srs_{normal},prep_{a1}}}	1	1	1	1

Fig. 2. Role matrices **mb** and **mcw** specifying the connectivity characteristics of the network model

In this model two actions occur, one of which is triggered by a supernormal stimulus and one by a normal stimulus. For both actions dopamine release increases the motivation of the participant to perform the action. It also increases the preparation for the respective action. The supernormal stimuli also create emotional imitation, since supernormal stimuli often play to the emotion by mimicking feeling. The emotion and the dopamine determine the motivation for performing the action relating to the supernormal stimulus. For the normal stimulus, the motivation is solely dependent on the dopamine released. At the moment the total amount of dopamine released from all actions is higher than the threshold for dopamine, the motivation will start to have a higher tolerance for dopamine. Each ownership state will be determined by the stimulus, the motivation, the preparation, and for the supernormal stimulus action it is also dependent on the emotion of the respective action. Moreover, the expectation (expected effect) of an action also influences the preparation of an action and the other way around.

mcfw		combination			ms speed factors		
function weights		1	2	3	1		
		alogistic	hebb	step-mod			
X ₁	srs _{normal}	1			X ₁	srs _{normal}	0
X ₂	dopamine _{a1}	1			X ₂	dopamine _{a1}	0.2
X ₃	mot _{a1}	1			X ₃	mot _{a1}	0.2
X ₄	prep _{a1}	1			X ₄	prep _{a1}	0.2
X ₅	exe _{a1}	1			X ₅	exe _{a1}	0.2
X ₆	exp _{a1}	1			X ₆	exp _{a1}	0.2
X ₇	own _{a1}	1			X ₇	own _{a1}	0.2
X ₈	srs _{supernormal}			1	X ₈	srs _{supernormal}	2
X ₉	dopamine _{a2}	1			X ₉	dopamine _{a2}	0.2
X ₁₀	imitation	1			X ₁₀	imitation	0.2
X ₁₁	emotion	1			X ₁₁	emotion	0.2
X ₁₂	mot _{a2}	1			X ₁₂	mot _{a2}	0.2
X ₁₃	prep _{a2}	1			X ₁₃	prep _{a2}	0.2
X ₁₄	exe _{a2}	1			X ₁₄	exe _{a2}	0.2
X ₁₅	exp _{a2}	1			X ₁₅	exp _{a2}	0.2
X ₁₆	own _{a2}	1			X ₁₆	own _{a2}	0.2
X ₁₇	T _{dopetolerance}	1			X ₁₇	T _{dopetolerance}	0.2
X ₁₈	W _{srs_{supernormal},prep_{a2}}		1		X ₁₈	W _{srs_{supernormal},prep_{a2}}	X ₂₀
X ₁₉	W _{srs_{normal},prep_{a1}}		1		X ₁₉	W _{srs_{normal},prep_{a1}}	X ₂₁
X ₂₀	Hw _{srs_{supernormal},prep_{a2}}	1			X ₂₀	Hw _{srs_{supernormal},prep_{a2}}	0.1
X ₂₁	Hw _{srs_{normal},prep_{a1}}	1			X ₂₁	Hw _{srs_{normal},prep_{a1}}	0.1

combination function		1		2		3	
mcfp		alogistic		hebb		stepmod	
parameter		1	2	1	2	1	2
		σ	τ	μ		ρ	δ
X ₁	srs _{normal}	5	0.2				
X ₂	dopamine _{a1}	5	0.2				
X ₃	mot _{a1}	5	0.2				
X ₄	prep _{a1}	5	X ₁₇				
X ₅	exe _{a1}	5	0.2				
X ₆	exp _{a1}	5	0.2				
X ₇	own _{a1}	5	0.2				
X ₈	srs _{supernormal}					100	40
X ₉	dopamine _{a2}	5	0.2				
X ₁₀	imitation	5	0.2				
X ₁₁	emotion	5	0.2				
X ₁₂	mot _{a2}	5	0.2				
X ₁₃	prep _{a2}	5	X ₁₇				
X ₁₄	exe _{a2}	5	0.2				
X ₁₅	exp _{a2}	5	0.2				
X ₁₆	own _{a2}	5	0.2				
X ₁₇	T _{dopetolerance}	5	0.6				
X ₁₈	W _{srs_{supernormal},prep_{a2}}			0.95			
X ₁₉	W _{srs_{normal},prep_{a1}}			0.95			
X ₂₀	Hw _{srs_{supernormal},prep_{a2}}	5	0.8				
X ₂₁	Hw _{srs_{normal},prep_{a1}}	5	0.8				

Fig. 3. Role matrices **mcfw**, **mcfp** and **ms** specifying the aggregation and timing characteristics of the network model

5 Simulation Results

From the simulated scenarios, the following one is presented. First (from time 0 to time 40), only a normal stimulus occurs. As a result the states related to action a_1 are activated (see Fig. 4). After time 40, a supernormal stimulus occurs. As a result the base states related to action a_2 are activated. But what also can be seen in Fig. 4, at the same time the activations of the states related to action a_1 that are triggered by the normal stimulus (which is still present) drop.

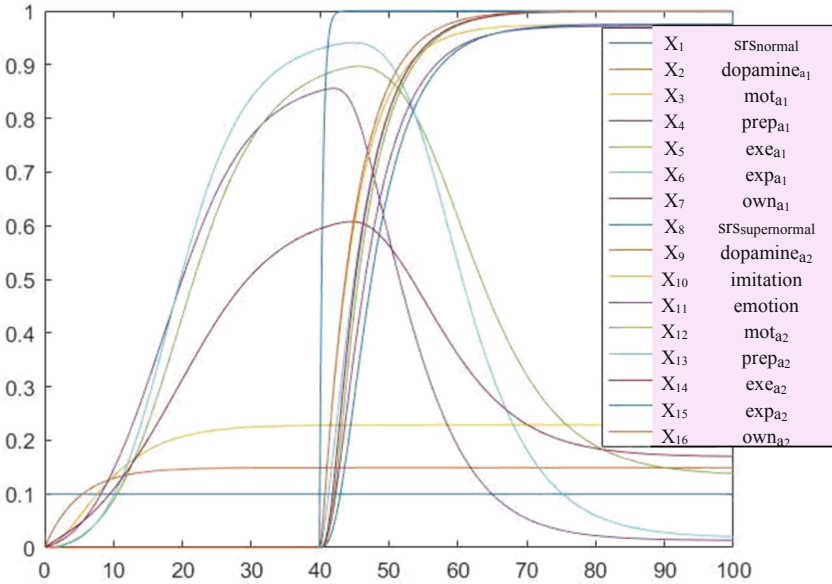


Fig. 4. Simulation results for the base states

The explanation for this drop can be found in Fig. 5, where the values of the adaptation states (the first- and second-order self-model states) have been visualized. When the supernormal stimulus arrives (in addition to the normal stimulus), the normal stimulus does not seem to have much effect anymore because the T-state for the dopamine tolerance (the blue line) goes up between time 40 and 50. This indicates desensitisation. Hence the action does not happen anymore when the stronger supernormal stimulus emerges which prevents the normal action from getting much effect.

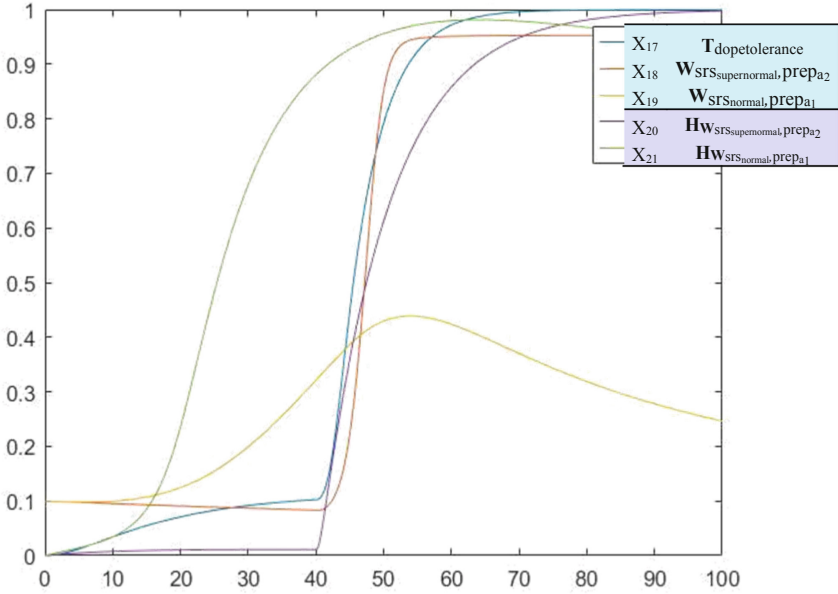


Fig. 5. Simulation results for the first- and second-order self-model states

6 Verification of the Network Model by Stationary Point Analysis

The dynamics of the implemented model were mathematically verified against its design specification by inspecting stationary points, i.e., points for some state Y where $dY(t)/dt = 0$. Based on difference Eq. (2), the following criterion is obtained for a state Y to have a stationary point at t (e.g., [9], Ch 12):

$$\begin{aligned} \eta_Y &= 0 \text{ or} \\ \mathbf{aggimpact}_Y(t) &= Y(t) \end{aligned} \quad (3)$$

where according to (1)

$$\mathbf{aggimpact}_Y(t) = \mathbf{c}_Y(\omega_{X_1,Y}X_1(t), \dots, \omega_{X_k,Y}X_k(t))$$

with X_1, \dots, X_k the states from which the state Y has incoming connections. In the example simulation, it can be observed that shortly after the individual starts being exposed to the supernormal stimulus, all of the states regarding the normal stimulus seem to reach a maximum shortly after arrival at time 40 of the supernormal stimulus. Hence the addition of the supernormal stimuli does make the action a_1 be performed less. The numerical data of the simulation do confirm this since the difference between the left hand side and right hand side of criterion (3) is less than 0.01 for all indicated states related to action a_1 within a few time units after the supernormal stimulus is added (see the upper part of Table 3).

Table 3. Mathematical analysis of stationary points and equilibrium values

State X_i	prep _{a₁}	exe _{a₁}	exp _{a₁}	own _{a₁}
Time t	42.0	45.3	44.7	44.3
$X_i(t)$	0.826204054	0.88181571	0.929487085	0.590019599
aggimpact _{$X_i(t)$}	0.825695857	0.880066117	0.928358746	0.589006296
deviation	-0.000508197	0.001749593	0.001749593	0.001013302
State X_i	exe _{a₁}		exe _{a₂}	
Time t	100		100	
$X_i(t)$	0.147271358		0.999819353	
aggimpact _{$X_i(t)$}	0.144670364		0.999829352	
deviation	0.002600994		-0.001001	

Also, at time 100 many states seem to have fairly constant values. We have checked the difference between aggregated impact for how much both actions are performed at the end of the simulation (see the lower part of Table 3). As can be seen in Table 3, the aggregated impact is always within a threshold of 0.01 from the state value. Hence, the stationary points analysis does not show any evidence that there is anything wrong with the implemented model in comparison to its design specification.

7 Discussion

In this paper an adaptive temporal-causal network model has been introduced by which the effect of overstimulation on the performance of actions is modeled. Simulation experiments have been performed in which a supernormal stimulus and/or normal stimulus was present and led to a desensitization adaptation. The model has been mathematically verified by analysis of stationary points for one of the simulation scenarios.

The proposed model has been built according to the adaptive network modeling approach based on self-modeling networks (or reified networks) described in [10]. This method of modeling makes it possible to easily design any adaptive network model, which in this case describes how by adaptation supernormal stimuli distract people from the day to day activities.

In future work, stimuli with different gradations can be explored and further validation of the model may be performed when suitable data sets are available.

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