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Huijzer, Willem; Treur, Jan

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An Adaptive Network Model for Sleep Paralysis: The Risk Factors and Working Mechanisms

Willem Huijzer and Jan Treur(✉)

Social AI Group, Department of Computer Science, Vrije Universiteit, Amsterdam, The Netherlands
w.d.huijzer@student.vu.nl, j.treur@vu.nl

Abstract. In this paper, an adaptive network model is presented for working mechanisms and risk factors of sleep paralysis with and without hallucinations. Sleep paralysis is a mysterious phenomenon where a person is awake but not able to move. Correspondingly, people can experience hallucinations and if so, often in a horrifying manner. The introduced network model covers the potential role of serotonin, temporoparietal junction, amygdala, 5-HT2AR receptor and covers the influence of risk factors as sleep deprivation, alcohol use, and the HT2AR gene.

Keywords: Adaptive network model · Sleep paralysis · Hallucinations · Alcohol · Sleep deprivation · Serotonin · 5-HT2AR receptor · Pimavanserin

1 Introduction

Many functions and working mechanisms of sleep have been addressed by researchers, although some phenomena of sleep are still a bit mysterious (Frank 2006). Sleep paralysis is one of those mysterious phenomena. During this state a person is awake, but can't move his or her muscles (Sateia 2014). Some people correspondingly experience hallucinations and if so, often in a horrifying manner. Not being able to move and having terrifying hallucinations can cause a lot of fear and distress for an individual. It has been hypothesized by researchers that sleep paralysis could be an explanation for people experiencing extraordinary events throughout history. In culturally specific contexts an occurrence of sleep paralysis could lead to the interpretation of a spiritual or supernatural event (Hufford 2005). Sleep paralysis normally occurs during the transition between sleep and wakefulness. In a systematic review paper of Sharpless and Barber (2011), the life time prevalence of experiencing at least one episode of sleep paralysis has been estimated to be 7.6% for the general population. Students and psychiatric patients are estimated to be more vulnerable, having a prevalence of respectively 28.3% and 34.6%. Researching the causes and working mechanisms of sleep paralysis could contribute to the understanding of sleep and might provide ways of coping for people that are troubled by sleep paralysis.

In the study reported here, the Network-Oriented Modeling approach for adaptive networks (Treur 2020) was applied to model possible working mechanisms and risk

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factors of sleep paralysis. The proposed adaptive network model based on prior biological and neurological literature covers the potential role of serotonin level, risk factors, genes, medicine and more. Four different scenarios have been simulated to evaluate the model for: a normal sleep-wake cycle, the occurrence of sleep paralysis without hallucinations, sleep paralysis with hallucinations due to gene vulnerability and again without hallucinations due to medicine intake against the gene vulnerability.

2 Neurological Background

In this section the underlying biological and neurological background of regular sleep, sleep paralysis and possible hallucinations during a sleep paralysis will be discussed. The model proposed in the current study will be based upon the following neurological background.

2.1 Regular Sleep

Different internal and external factors have influence on regulating the sleep-wake cycle. Firstly, the human body itself has multiple complex systems that contribute to regulating the sleep-wake cycle. These systems can be narrowed down to sleep promoting circuits and wakefulness promoting circuits; between these circuits is a mutual inhibition to ensure stability. The switching between the circuits function analogously to an electronic 'flip-flop' switch (Fuller et al. 2006). Additionally, various external factors influence these the sleep-wakefulness regulation e.g., blue light (Lawrenson et al. 2017) and caffeine (Snel and Lorist 2011). The wakefulness promoting systems use a number of neurotransmitters to activate the brain; one of these neurotransmitters is serotonin. Serotonin promotes wakefulness (Brown et al. 2012), but also inhibits the REM sleep (Ursin 2002).

The Global Workspace Theory from Baars (1997, 2002) describes that an individual experiences a single flow of consciousness, but in underlying processes there are multiple potential sources of consciousness competing to be experienced. The comparison can be made with a theater stage on which the potential consciousnesses are competing and where the 'spotlight' of attention can only shine on one flow of consciousness. The idea is that a winner-takes-it-all competition makes up which consciousness will be experienced by the individual. It is put forward by Hesslow (2012), Damasio (1999) and Goldman (2006) that flows of consciousness can be related to the concept of internal (mental) simulation. In this internal simulation, sensory representation states (for example mental images) are activated and in response preparation states are activated for actions or bodily changes. The preparations state itself can trigger (as a way of generating possible predicted effects) other sensory representation states and the bodily changes can eventually trigger the amygdala through the as-if body loop (Damasio 1999) and make the individual experience (feel) certain emotions. In the internal simulation, the prefrontal cortex has a regulating role towards the amygdala and the sensory representation states (Morgan et al. 1993).

Mental images during wakefulness are built up through input from the visual system. Levin and Nielsen (2007) discuss how mental images during dreams get created by

combining elements of different memories and how memory elements could be selected driven by emotions. Sleep paralysis has overlap of subjective descriptions and underlying brain mechanisms with dreams (Waters et al. 2016), therefore the theory on the emergence of dreams by Levin and Nielsen (2007) could be considered similarly for hallucinations.

2.2 Sleep Paralysis

Sleep paralysis is a state where a person is awake but experiences characteristics from REM sleep, namely not being able to move (Sharpless and Barber 2011). Jalal (2018) proposes a crucial role for an increased level of the neurotransmitter serotonin when experiencing a sleep paralysis. Prior research has suggested several predisposing factors for sleep paralysis, among which sleep deprivation (Stefani and Högl 2020) and alcohol use (Munezawa et al. 2011). Both sleep deprivation (Hery et al. 1970; Peñalva et al. 2003) and alcohol use (Chastain 2006) can increase the serotonin levels in the brain. Serotonin has numerous functions in the brain and body; one of the functions is regulating sleep. Serotonin promotes wakefulness and inhibits REM sleep (Ursin 2002). Taking the above description into account makes the role of serotonin plausible.

For sleep, four different stages are distinguished. The last stage, rapid eye movement (REM) sleep, is characterized by random rapid eye movements, motor atonia and the propensity of the sleeper to dream vividly. To prevent dangerous movements from being made when dreaming, REM sleep shuts down the motor neurons. The individual cannot use his or her muscles to talk or move; this is called motor atonia (Sharpless and Barber 2011). Furthermore, during REM sleep the prefrontal cortex gets stimulated (Vandekerckhove and Cluydts 2010). There are various theories about the mechanism and the involved neurotransmitters that cause the motor atonia during REM sleep. Brooks and Peever (2012) suggest a model where reduced motor neuron excitation strengthens the paralysis, but the inhibition of the motor neurons is the driving force behind the paralysis. They suggest that among others GABA neurons trigger the paralysis by directly inhibiting motor neurons during REM sleep. Alcohol intake (Kumar et al. 2009) and sleep deprivation (Modirrousta et al. 2007) could also stimulate GABA neurons.

2.3 The Hallucinations

In a survey under students in Poland, Wróbel-Knybel et al. (2020) found that 66% of their sample experienced hallucinations during a sleep paralysis: 37% visual, 31% auditory and 25% tactile hallucinations. Jalal (2018) proposes two processes that cause the hallucinations during a sleep paralysis. The first process starts with the motor-execution being blocked while at same time the sensory input from the body still is intact. This causes a disturbance at the temporoparietal junction (TPJ). The TPJ gets input from many brain areas among which are visual, auditory and somatosensory systems. The area is a crucial structure for the conscious experience of the normal self (Blanke et al. 2005). Research from Arzy et al. (2006) shows that stimulation of the TPJ can trigger the uncanny feeling that someone is nearby, out of body experiences and visual hallucinations of human-like figures.

The second process proposed by Jalal (2018) has a central role for the serotonin 2A receptor (5-HT2AR). Serotonin can bind to many different receptors and 5-HT2AR is one of them (Nichols and Nichols 2008). This receptor is present in different regions of the brain and there are several reasons to assume that this receptor is involved with the hallucinations. Firstly, Parkinson (Huot et al. 2010) and schizophrenic (González-Maeso et al. 2008) patients with visual hallucinations show an increased number of 5-HT2AR receptors in the visual cortex compared to patients without visual hallucinations. Secondly, when a highly selective 5-HT2A inverse agonist medicine Pimavanserin is given to a Parkinson patient the severity of hallucinations decreases (Meltzer et al. 2010). Furthermore, the activation of the serotonin 2A receptors also mediates visual hallucination induced by Psilocybin (Kometer et al. 2013). The serotonin 2A receptor is also present in frontoparietal cortical regions, among which is the TPJ (Tagliazucchi et al. 2016). Taking the above into account, it is plausible that the 5-HT2A receptor influences visual hallucinations.

Moreover, 5-HT2A receptors are densely concentrated in the amygdala; therefore, it could activate and increase activation the intensity of emotions felt (Frokjaer et al. 2008). The amygdala modulates among other things fear responses in humans (Ressler 2010). Increased intense negative emotions could contribute to retrieval of negative memory elements and while an individual already is in fear due not being able to move, this could explain why the hallucinations during a sleep paralysis are mostly terrifying.

Genes and medicine can also have an influence on the hallucinations experienced during sleep paralysis. The gene coding for HTR2A is found to be located on the long arm of chromosome 13 (Badner and Gershon 2002). Jalal (2018) proposes the medicine Pimavanserin to reduces visual hallucinations and fear during a sleep paralysis. This medicine is a highly selective 5-HT2A inverse agonist. As noted above, the medicine is used successfully to reduce hallucinations among Parkinson patients (Meltzer et al. 2010).

3 The Modeling Approach Used

In this section a brief summary will be given of the adaptive network modelling approach developed by Treur (2020). This approach was used to design the model that is introduced in this paper. Network-Oriented Modelling uses nodes and vertices; vertices are the connections between the nodes. Nodes are states with values (usually between 0 and 1) that vary over time, while the connections indicate the impact that states have on each other. The latter can be interpreted as the causal relationships between the nodes. In an adaptive network model, besides the states also the causal relationships can change over time. Table 1 summarizes the main concepts of network-oriented modelling. Every connection has a connection weight, which is a numerical value indicating the connection strength. The connections and their weights define the network's *connectivity characteristics.*

In addition to the latter, every state has a combination function that describes the manner in which the incoming impacts per connection are combined to form an aggregated impact. This defines the network's *aggregation characteristics*. The combination function can be chosen from the available Combination Function Library. The choice on which combination function is used, depends on the application and can also be node-specific. To define the network's *timing characteristics*, every state has a speed factor that determines how fast a state changes because of its received causal impact.

Table 1. An overview of the concepts in the conceptual component of temporal-causal networks.

Concepts	Notation	Explanation
States and connections	X, Y $X \rightarrow Y$	Denotes the nodes and edges in the conceptual representation of a network model
Connection weights	$\boldsymbol{\omega}_{X,Y}$	A connection between states X and Y has a corresponding *connection weight.* In most cases $\boldsymbol{\omega}_{X,Y} \in [-1,1]$
Aggregating multiple impacts on a state	$\mathbf{c}_Y(..)$	Each state has a *combination function* and is responsible for combining causal impacts of all states connected to Y on that same state
Timing of the effect of causal impact	$\boldsymbol{\eta}_Y$	The *speed factor* determines how fast a state is changed by any causal impact. In most cases: $\boldsymbol{\eta}_Y \in [0,2]$

The numerical representation derived from the network characteristics is summarized in Table 2. The last row of this Table 2, shows the difference equation. Adaptive networks are networks for which some of the characteristics $\boldsymbol{\omega}_{X,Y}$, $\mathbf{c}_Y(..)$, $\boldsymbol{\eta}_Y$ change over time. To model this, extra states are added that represent the adaptive characteristics. For example, for an adaptive connection weight $\boldsymbol{\omega}_{X,Y}$ a new state $\mathbf{W}_{X,Y}$ is added representing the dynamic value of $\boldsymbol{\omega}_{X,Y}$; this new state is called a self-model or adaptation state for $\boldsymbol{\omega}_{X,Y}$.

Table 3 shows an overview of the combination functions used in the designed network model. The first is the identity function, **id(.)**, which is commonly used when a state only has one incoming connection. The advanced logistic sum function, $\mathbf{alogistic}_{\sigma,\tau}(..)$, is used to aggregate impact for each state that has multiple incoming connections; it has as parameters steepness $\boldsymbol{\sigma}$ and threshold $\boldsymbol{\tau}$.

Table 2. Numerical representations of temporal-causal networks.

Concepts	Notation	Explanation
State value at time t	$Y(t)$	For every time t a state Y has a value in [0,1]
Single causal impact	$\mathbf{impact}_{X,Y}(t) = \boldsymbol{\omega}_{X,Y}X(t)$	At any time t a state X, when connected to Y, impacts Y via connection weight $\boldsymbol{\omega}_{X,Y}$
Aggregating multiple impacts on a state	$\mathbf{aggimpact}_Y(t) = \mathbf{c}_Y(\mathbf{impact}_{X_1,Y}(t), \ldots, \mathbf{impact}_{X_K,Y}(t)) = \mathbf{c}_Y(\boldsymbol{\omega}_{X_1,Y}X_1(t), \ldots, \boldsymbol{\omega}_{X_K,Y}X_k(t))$	The combination function $\mathbf{c}_Y$ determines the aggregated causal impact of states X_i on Y
Timing of the effect of causal impact	$Y(t+\Delta t) = Y(t) + \boldsymbol{\eta}_Y [\mathbf{aggimpact}_Y(t) - Y(t)]\ \Delta t = Y(t) + \boldsymbol{\eta}_Y [\mathbf{c}_Y(\boldsymbol{\omega}_{X_1,Y}X_1(t), \ldots, \boldsymbol{\omega}_{X_K,Y}X_k(t)) - Y(t)]\ \Delta t$	The speed factor Y determines how fast a state Y is changed by the aggregated causal impact of states X_i

Table 3. Overview of the combination functions used.

Combination function	Description	Formula $\mathbf{c}_Y(V_1,\ldots,V_k) =$
id(.)	Identity	V
$\mathbf{alogistic}_{\sigma,\tau}(..)$	Advanced logistic sum	$\left[\frac{1}{1+e^{-\sigma(V_1+\cdots+V_k-\tau)}} - \frac{1}{1+e^{\sigma\tau}}\right](1+e^{-\sigma\tau})$

4 The Designed Adaptive Network Model

In the current section, an adaptive network model covering risk factors and working mechanisms of sleep paralysis will be introduced. In Table 4 all states are presented with a corresponding explanation. The fixed states remain at the initial value and do not change during a simulation; they make use of the **id(.)** function, while the other states make use of the $\mathbf{alogistic}_{\sigma,\tau}(..)$ function. The last four states in the table are self-model states (adaptation states) and the other states are base level states. In the Appendix at URL https://www.researchgate.net/publication/353206807 the initial values (**iv**) of the states and the full specification by role matrices (Treur 2020) are presented: base connections (**mb**), connection weights (**mcw**), speed factors (**ms**), combination function weights (**mcfw**) and combination function parameters (**mcfp**).

In Fig. 1, a graphic conceptual representation is given of the connectivity of the introduced adaptive network model. It is shown that the wakefulness promoting circuits and sleep promoting circuits work analogously to a flip-flop switch. They inhibit each other and alternate by stimulating each other's activators; in this manner both promoting circuits will take turns in being active. One of the tools of the wakefulness promoting systems to activate the brain is serotonin. The serotonin level gets stimulated by the wakefulness promoting circuits and helps to stimulate wakefulness. Both external factors sleep deprivation and alcohol use can also stimulate the serotonin level. REM sleep gets stimulated by sleep promoting circuits and is inhibited by serotonin. During REM sleep the motor atonia increases; this connection is adaptive. The state context REM-Atonia stimulates the adaptive weight state W-REM-Atonia for a base level; alcohol use and sleep deprivation could strengthen the connection. When an individual is under motor atonia, the disturbance of TPJ and the activity of the amygdala could increase. Increased amygdala activity could lead to the feeling of fear, which in its turn could lead to retrieval of negative memory elements. Motor atonia can only have this effect when a person is awake; the outgoing weights states of the motor atonia to the amygdala and the disturbance of TJP are fully dependent of input from the wakefulness state.

In the model, the competition for the 'spotlight' of consciousnesses is represented by the states of dream, hallucinations and reality inhibiting each other. Each of those states gets stimulated by a corresponding sensory representation state and each sensory representation state has a feedback loop to the preparation state.

Furthermore, each sensory representation state gets negative feedback via a corresponding control state. The sensory representation state of dream further gets input from REM sleep, negative memory ER, and 5-HT2AR receptor. The sensory representation state of hallucinations further gets input from wakefulness, 2-HT2AR receptor, negative memory ER and disturbance TPJ. The sensory representation state of reality further gets input from wakefulness. The amygdala has a (negative) feedback loop with each of the control states and the preparation state. The 5-HT2AR receptor gets stimulated by the serotonin level and the threshold is represented by the T - 5-HT2AR receptor state and gets a base line input from the context receptor and further negative input from the HT2AR gene and positive input from the medicine Pimavanserin.

Table 4. All states of the introduced adaptive network model with explanations.

	State	Explanation
X_1	Sleep deprivation	**Fixed** state, indication of whether an individual is sleep deprived (1) or not (0)
X_2	Alcohol use	**Fixed** state, indication of whether an individual has used alcohol (1) or did not (0)
X_3	Serotonin level	The level of the neurotransmitter serotonin in the brain
X_4	Wakefulness	The likelihood of an individual being awake; a brain state where an individual is conscious and engages in mental processes with the external world
X_5	REM sleep	The likelihood that an individual is in a REM sleep phase
X_6	5-HT2AR receptor	The intensity of serotonin binding to the 5-HT2AR receptor
X_7	Motor atonia	Motor atonia is a condition where an individual can't move his or her muscles due to inhibition by the brain
X_8	Disturbance TPJ	This state represents potential disturbance in the temporoparietal junction
X_9	Amygdala	The activity of the amygdala, brain area that is responsible for emotional responses
X_{10}	Control dream	Activity of the part of prefrontal cortex inhibiting the amygdala and the sensory representation state for dreaming
X_{11}	Control hallucinations	Activity of the part of prefrontal cortex inhibiting the amygdala and the sensory representation state for hallucinating
X_{12}	Control reality	Activity of the part of prefrontal cortex inhibiting the amygdala and the sensory representation state for experiencing reality
X_{13}	Fear	Likelihood of an individual experiencing fear, an unpleasant emotion often caused by potential danger or harm
X_{14}	Negative memory ER	The probability that memory elements with a negative association will be retrieved
X_{15}	Wakefulness PCA	The activity of wakefulness promoting circuits activators
X_{16}	Sleep PCA	The activity of sleep promoting circuits activators
X_{17}	Wakefulness PC	The activity of wakefulness promoting circuits
X_{18}	Sleep PC	The activity of sleep promoting circuits
X_{19}	Preparation state	The activity of the preparation state
X_{20}	SRS dream	The activity of the sensory representation state of a dream
X_{21}	SRS hallucinations	The activity of the sensory representation state of hallucinations
X_{22}	SRS reality	The activity of the sensory representation state of a reality
X_{23}	Dream	The likelihood of an individual experiencing a dream

(*continued*)

Table 4. (*continued*)

	State	Explanation
X_{24}	Hallucinations	The likelihood of an individual experiencing hallucinations
X_{25}	Reality	The likelihood of an individual experiencing the reality
X_{26}	HT2AR gene	**Fixed state**, indication of whether an individual has a genetic predisposition for the creating 5-HT2AR receptors (1) or not (0)
X_{27}	Pimavanserin	**Fixed state**, indication of whether an individual has taken the medicine Pimavanserin (1) or not (0)
X_{28}	Context receptor	**Fixed state**, this state represents the influence of context factors on the threshold of the 5-HT2AR receptor
X_{29}	Context REM-atonia	**Fixed state**, this state represents the influence of context factors on the connection weight between REM sleep and motor atonia
X_{30}	$\mathbf{W}_{\text{Atonia-TJP}}$	This self-model state represents the value of the weight between the states motor atonia and temporoparietal junction
X_{31}	$\mathbf{W}_{\text{Atonia-Amygdala}}$	This self-model state represents the value of the weight between the states motor atonia and amygdala
X_{32}	$\mathbf{W}_{\text{REM-Atonia}}$	This self-model state represents the value of the weight between the states REM sleep and motor atonia
X_{33}	$\mathbf{T}_{\text{Receptor 5-HT2AR}}$	This self-model state represents the value of the threshold of the 5-HT2AR receptor state

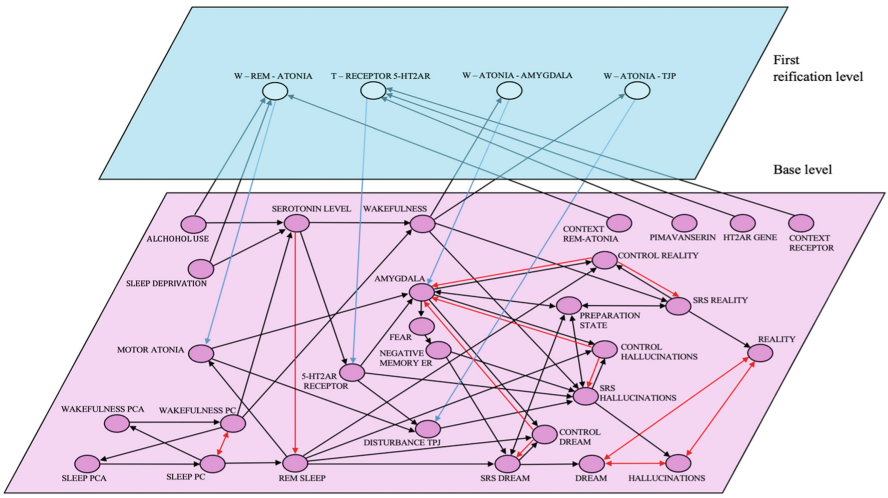

Fig. 1. Graphical representation of the connectivity of the introduced model.

5 Simulation Experiments

In this section, the simulation experiments performed are presented. The different scenarios were created merely by changing the initial values of sleep deprivation, alcohol use, HT2AR gene and Pimavanserin. The sleep and wakefulness promoting circuits are not influenced by other states and therefore remain identical for all the scenarios. Figure 2 shows how the sleep and wakefulness promoting circuits alternate with each other. When one system is active, the activator of the other system is stimulated and so they rotate in a loop. The x-as is an indication of time in hours.

5.1 Simulation of a Regular Sleep Cycle

In the first scenario a regular sleep cycle is simulated. For this scenario, the initial values of the following states are all set to 0: sleep deprivation, alcohol use, HT2AR gene and Pimavanserin.

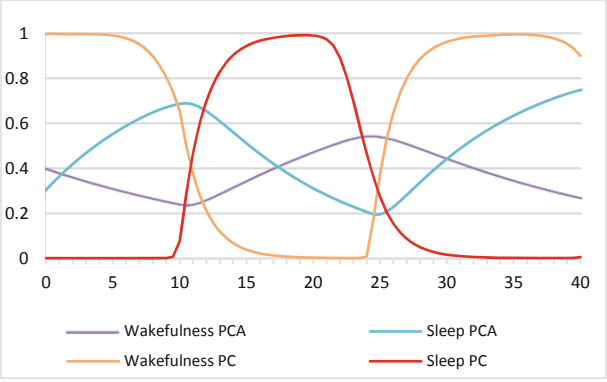

Fig. 2. Simulation of sleep-wake cycle.

Figure 3 left shows how the sleep-wake cycle is not interrupted and how wakefulness doesn't overlap with REM sleep nor motor atonia; there is no sleep paralysis. Figure 3 right shows how the activity of the dream, hallucinations and reality states with corresponding control and sensory representation states fluctuate over time.

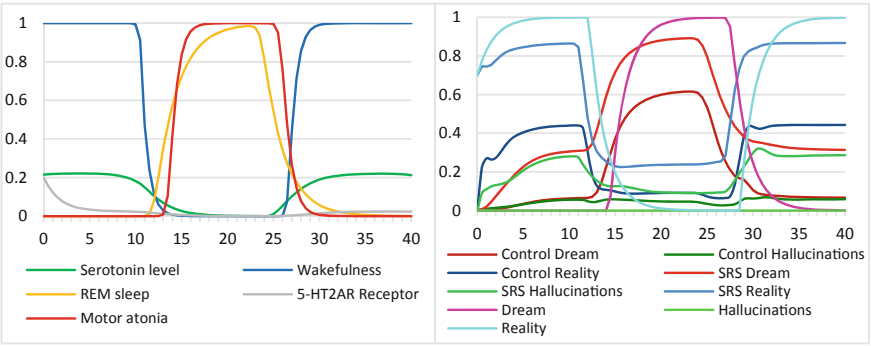

Fig. 3. Simulation of serotonin, wakefulness and REM sleep related states (left) and of dream, hallucinations and reality states with corresponding sensory representation and control states (right) during the first scenario.

It can be seen that the possibility of experiencing reality alternates with the possibility of experiencing a dream; the possibility of hallucinations does not surface in this scenario. As wakefulness does not overlap with motor atonia, the connection between REM sleep and motor atonia isn't strong when the motor atonia is active. As a result, the disturbance of TPJ and the amygdala only increases slightly during the transition between sleep and wakefulness (Fig. 4). Furthermore, the serotonin level remains low and the threshold of the 5-HT2AR remains high. The consequence is that the 5-HT2AR receptor has low activity.

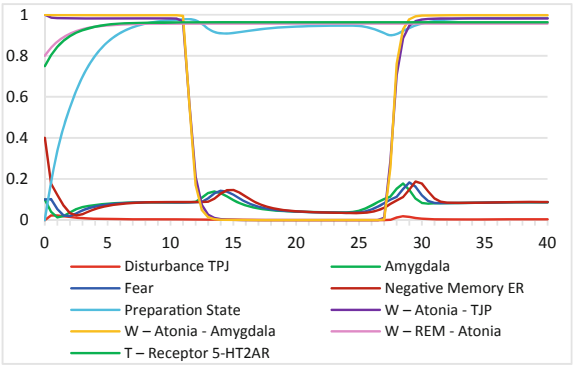

Fig. 4. Simulation of the disturbance of TPJ, emotion related states and adaptive states during the first scenario.

5.2 Simulation of Sleep Paralysis

In this scenario, the states alcohol use and sleep deprivation were set to 1. The other fixed states HT2AR gene and Pimavanserin remain 0. The effect of alcohol use and sleep

deprivation is that the serotonin level rises (Fig. 5 left) and that connection between REM sleep and motor atonia get strengthened (Fig. 6). This causes the sleep-wake cycle to be disturbed; wakefulness, REM sleep and motor atonia now overlap during the transitions between sleep and wakefulness. This means that both at the beginning and at the end of the sleep cycle the probability of sleep paralysis is high. Similar to the previous scenario the likelihood of experiencing a dream and reality alternate each other. Only in this scenario experiencing a dream takes up less time, which is substituted by experiencing the reality for a longer period of time. The likelihood of hallucinations increases, but doesn't overrule the likelihood of experiencing a dream nor experiencing reality (Fig. 6 right). The amygdala, fear, negative memory retrieval and disturbance of TPJ all increase considerably around the sleep-wake transition areas. But this apparently doesn't stimulate the sensory representation state of hallucinations enough to rise above the other sensory representation states. An explanation for this could be found in a rather low activity of the 5-HT2AR receptor.

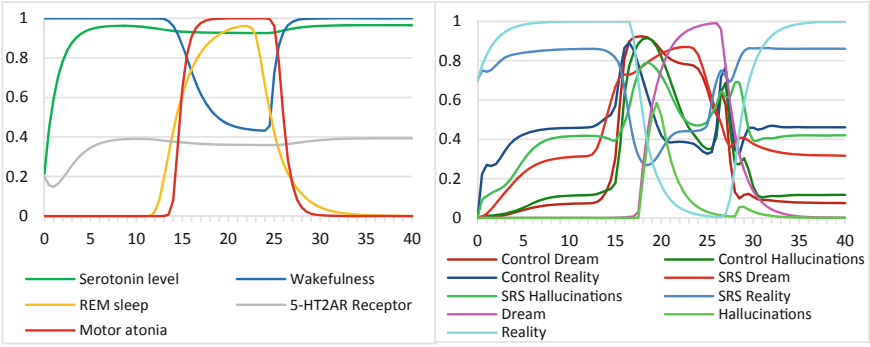

Fig. 5. Simulation of serotonin, wakefulness and REM sleep related states (left) and of dream, hallucinations and reality states with corresponding sensory representation and control states (right) during the second scenario.

5.3 Simulation of Sleep Paralysis with Genetic Vulnerability for Hallucinations

For this scenario, the states alcohol use, sleep deprivation and HT2AR gene were all set to 1. The fixed state Pimavanserin remains 0. Identical to the previous scenario, the probability of sleep paralysis is high during the transitions between sleep and wakefulness. The difference with the previous scenario is that during the current scenario hallucinations have a high likelihood of being experienced with potential sleep paralyses during the transitions between sleep and wakefulness (Fig. 7 left).

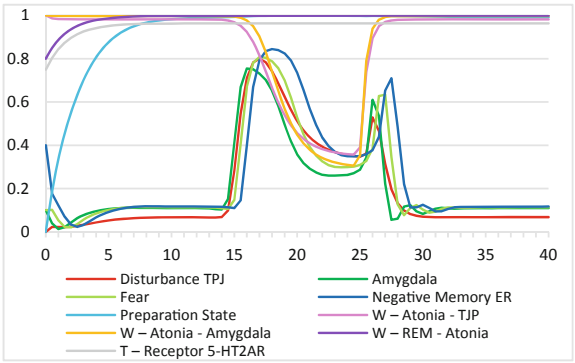

Fig. 6. Simulation of the disturbance of TPJ, emotion related states and adaptive states during the second scenario.

This is caused by the onset of the HT2AR gene; the gene is responsible for a greater number of 5-HT2AR receptors in the brain. In Fig. 7 right is shown how the threshold of the 5-HT2AR receptor decreases. As a result, the activity of 5-HT2AR receptor itself increases. The increased 5-HT2AR receptor activity has a direct influence on the sensory representation state of hallucinations. Additionally, it has an indirect influence on hallucinations through stimulating the disturbance of the TPJ and amygdala.

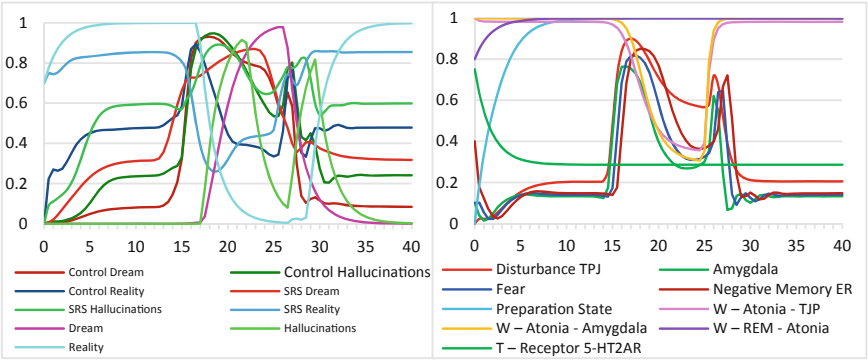

Fig. 7. Simulation of and of dream, hallucinations and reality states with corresponding sensory representation and control states (left) and of the disturbance of TPJ, emotion related and adaptive states (right) during the second scenario.

5.4 Simulation Sleep Paralysis Without Hallucination Due to Pimavanserin

For this scenario the initial values of the following states were all set to 1: sleep deprivation, alcohol use, HT2AR gene and Pimavanserin. Again, the probability of sleep paralysis is high during the transitions between sleep and wakefulness. During the current scenario the medicine Pimavanserin binds to the 5-HT2AR receptor, counteracting

the regular effect. The threshold of the 5-HT2AR receptor eventually increases slightly, instead of decreasing as in the previous scenario. The outcomes of the current scenario are almost identical to the sleep paralysis scenario with no genetic vulnerability.

6 Discussion

In the study reported here, an adaptive network model was introduced that addresses the risk factors and working mechanisms of sleep paralysis. The presented network model is based on prior literature and validated by the simulations. The model has shown how risk factors such as alcohol use and sleep deprivation may increase the serotonin level and thereby disrupting the sleep-wake cycle. This may cause a sleep paralysis as wakefulness, REM sleep and most importantly motor atonia could overlap during the transition between wakefulness and sleep. This overlap causes fear and disturbance of the TPJ but doesn't necessarily cause hallucinations. The likelihood of experiencing hallucinations strongly increases if an individual has a genetic predisposition for creating a greater number of 5-HT2AR receptors. The receptor can directly stimulate hallucinations, but also indirectly by stimulating disturbance at the TPJ. Levin and Nielsen (2007) discuss how the selection of memory elements for creating mental images can be driven by emotions. And while the individual is likely to experience a lot of fear during a sleep paralysis, this could be an explanation for the fact that hallucinations often occur in a terrifying manner. Additionally, stimulation of the 5-HT2AR receptor can cause increased amygdala activity. The model further shows how the medicine Pimavanserin proposed by Jalal (2018) can counteract the genetic vulnerability and strongly decrease the likelihood of experiencing hallucinations.

The model is based on the latest literature available anno 2021; many components and working mechanisms of sleep are currently still under discussion and are subject of further research. For the model introduced, it has been attempted to find as much empirical literature as possible. However, for many components of the model there was no numerical empirical data available and qualitative empirical information had to be used. Based on the latter the current model has to be interpreted with a certain degree of prudence.

The model introduced leaves much room for further exploration in future studies. Both the risk factors and the working mechanisms could be refined or extended. Several predisposing factors for sleep paralysis have not yet been included in the current model, for example, jetlag (Stefani and Högl 2020) and the use of antidepressants (Sandman et al. 2015). Furthermore, Jalal and Ramachandran (2017) propose a role for mirror neurons as working mechanism for hallucinating during sleep paralysis.

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