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An Adaptive Cognitive Temporal-Causal Model for Extreme Emotion Extinction Using Psilocybin

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Abstract. In this paper, an adaptive cognitive temporal-causal model using psilocybin for a reduction in extreme emotion is presented. Extreme emotion has an effect on some brain components such as visual cortex, auditory cortex, gustatory cortex, and somatosensory cortex as well as motor cortex such as primary motor cortex, and premotor cortex. Neuroscientific literature reviews show that using psilocybin has a significant effect mostly on two brain components, cerebral cortex, and thalamus. Network-oriented modeling via temporal-causal network-oriented modeling is presented to show the influences of using psilocybin on the cognitive part of the body, same as the brain components. Hebbian learning used to show the adaptivity and learning section of the presented model.

Keywords: Network-oriented modeling · Extreme emotion · Psilocybin · Temporal-causal network

1 Introduction

An intense stimulus, life-threatening phenomena such as an accident, war, leads to a well-known impact on the brain called stress. Based on the psychological peace of researches, 5 to 8% of men and 10 to 14% of women and up to 25% of combat veterans are involved in post-traumatic stress disorder (PTSD) in their lives. Extreme emotions such as stress have a significant impact on both cognitive and psychological functionality on the patient's brains [27].

In this paper, the influence of using psilocybin on patients with anxiety and depression disorders has been investigated. Neuroscience literature review shows a significant role of using psilocybin on two main brain components namely thalamus and cerebral cortex. The aim of this paper is to show the effect of using this drug on the brain components and having prediction of activity of those on patient. In [9] the definition and performance of using aforementioned drug has been mentioned as follows.

‘Psilocybin is the prodrug of psilocybin (4-hydroxy-dimethyltryptamine), the primary hallucinogenic component of magic mushrooms, and a classic psychedelic (‘mind-manifesting’) drug. Psilocybin has been used for centuries in healing

ceremonies (1) and more recently in psychotherapy (2); it is capable of stimulating profound existential experiences (3), which can leave a lasting psychological impression (4).’

The use of psilocybin goes back to many years ago in religious rituals and medical usages [11]. There are several network-oriented modelings via the temporal-causal network for therapies or drugs taking to decrease the extreme emotion [13–26]. In this paper, the effect of using psilocybin has been used to demonstrate this event.

The paper is organized as follows. In Sect. 2 the underlying neurological principles concerning the parts of the brain involved in stress and in the suppression of stress are addressed. In Sect. 3 the integrative temporal-causal network model is introduced. In Sect. 4 the results of the simulation model are discussed, in Sect. 5 the mathematical analysis of the model is presented and eventually in the last section a conclusion is presented.

2 Underlying Neurological Principles

Psilocybin remarkably decreases brain blood flow and venous oxygenation in a way that related to the subjective impacts on the brain. Psilocybin based on the dose of use has different effects in other words it is dose-dependently and has a huge influence on changing the mood, perception, self-experience and thought and based on the result appeared in [10] experienced pleasurable moments, and non-threatening situations.

Neuroscience research shows that psilocybin has a significant agonist 5-HT (serotonergic) pathways and receptors located in the Thalamus and the cerebral cortex [1]. Triggering receptors such as 5-HT_{2A} in the thalamus by using, which is responsible for sensory input, decreases the activity of the thalamic performance. Having decreased the thalamic activity, leading to hallucinations [2, 3]. As far as anxiety disorders and depression cause abnormalities in sensory perception of individuals, psilocybin has an impact on these areas in line with serotonin hormone. In [4] the moderate advantages of the effect of using psilocybin on patients in the Netherlands have been discussed. In [5] also the influence of periodical use of psilocybin and the psychological disorders investigated and also the playing role of psilocybin on decreasing the sense of committing suicide and improvement of moods have been highlighted. In comparison to methylphenidate the effective role of psilocybin on increasing social activity, behavior and positive behavior have been noted [6]. Some medical experiments [7, 8] have been done to show the positive effect of using psilocybin on patients with depression and anxiety due to tough diseases such as cancer. The reduction in an anxiety in patients with cancer was remarkable during the period between 1 to 3 months of posttreatment with psilocybin and had lower negative effect comparing to patients who used niacin [6].

Decreased activity in thalamus, cerebral cortex has been mentioned in [1, 9]. Cerebral cortex contains some brain components such as gustatory cortex, visual cortex, somatosensory cortex, and auditory cortex. These cortices received information, and input from thalamus when a patient uses psilocybin. Other components of cerebral cortex involved in decreasing anxiety consists of primary motor and premotor cortex.

The timing process of the effect of using psilocybin mentioned in [9] precisely:

‘The subjective effects began toward the end of the infusion period and reached a sustained peak after ~ 4 min (5). The first level results were entered into higher level analysis, contrasting cerebral blood flow (CBF) after placebo for all 15 subjects.’

In [10] acute, short and long-term subjective effect of using psilocybin in healthy individuals have been examined. The remaining cognitive parts of the model namely sensory representation state, preparation state, feeling state are based on the cognitive behavior of mind during extreme emotion.

3 The Temporal-Causal Network Model

First the Network-Oriented Modelling approach used to model the integrative overall process is briefly explained. As discussed in detail in [11, Ch 2] this approach is based on temporal-causal network models which can be represented at two levels: by a conceptual representation and by a numerical representation. A conceptual representation of a temporal-causal network model in the first place involves representing in a declarative manner states and connections between them that represent (causal) impacts of states on each other, as assumed to hold for the application domain addressed. The states are assumed to have (activation) levels that vary over time. In reality, not all causal relations are equally strong, so some notion of *strength of a connection* is used. Furthermore, when more than one causal relation affects a state, some way to *aggregate multiple causal impacts* on a state is used. Moreover, a notion of *speed of change* of a state is used for timing of the processes. These three notions form the defining part of a conceptual representation of a temporal-causal network model:

- **Strength of a connection $\omega_{X,Y}$.** Each connection from a state X to a state Y has a *connection weight value* $\omega_{X,Y}$ representing the strength of the connection, often between 0 and 1, but sometimes also below 0 (negative effect) or above 1.
- **Combining multiple impacts on a state $c_Y(\cdot)$.** For each state (a reference to) a *combination function* $c_Y(\cdot)$ is chosen to combine the causal impacts of other states on state Y .
- **Speed of change of a state η_Y .** For each state Y a *speed factor* η_Y is used to represent how fast a state is changing upon causal impact.

Combination functions can have different forms, as there are many different approaches possible to address the issue of combining multiple impacts. Therefore, the Network-Oriented Modelling approach based on temporal-causal networks incorporates for each state, as a kind of label or parameter, a way to specify how multiple causal impacts on this state are aggregated by some combination function. For this aggregation a number of standard combination functions are available as options and a number of desirable properties of such combination functions have been identified.

Figure 1 represents the conceptual representation of the temporal-causal network mode. The components of the conceptual representation shown in Fig. 1 are explained here. The state ws_c shows the world state of the contextual stimulus c . The states ss_c and ss_{ee} are the sensor state for the context c and sensor state of the body state ee for the

extreme emotion. The states srs_c and srs_{ee} are the sensory representation of the contextual stimulus c and the extreme emotion, respectively. The state srs_{ee} is a stimulus influencing the activation level of the preparation state. Moreover, ps_{ee} is the preparation state of an extreme emotional response to the sensory representation srs_c of the context c , and fs_{ee} shows the feeling state associated to this extreme emotion. The state es_{ee} indicates the execution of the body state for the extreme emotion. All these relate to the affective processes. The (cognitive) goal state shows the goal for using psilocybin in the body. The (cognitive) state ps_{psil} is the preparation state of taking a psilocybin. The state es_{psil} is the execution state of taking psilocybin. The other states relate to biological brain parts, cerebral cortex which consists of two major components namely sensory areas (gustatory cortex, visual cortex, somatosensory cortex, auditory cortex) and motor areas (primary motor cortex, premotor cortex), thalamus, hippocampus, medial prefrontal cortex, and amygdala which are involved in the stress condition, and in the influence of the psilocybin applied (Table 1).

Table 1. Explanation of the states in the model

X_1	ws_{ee}	World (body) state of extreme emotion ee
X_2	ss_{ee}	Sensor state of extreme emotion ee
X_3	ws_c	World state for context c
X_4	ss_c	Sensor state for context c
X_5	srs_{ee}	Sensory representation state of extreme emotion ee
X_6	srs_c	Sensory representation state of context c
X_7	fs_{ee}	Feeling state for extreme emotion ee
X_8	ps_{ee}	Preparation state for extreme emotion ee
X_9	es_{ee}	Execution state (bodily expression) of extreme emotion ee
X_{10}	goal	Goal of using psilocybin
X_{11}	ps_{psil}	Preparation state of using psilocybin
X_{12}	es_{psil}	Execution of using psilocybin
X_{13}	Gustatory cortex	Brain part
X_{14}	Visual cortex	Brain part
X_{15}	Somatosensory cortex	Brain part
X_{16}	Auditory cortex	Brain part
X_{17}	Thalamus	Brain part
X_{18}	Hippocampus	Brain part
X_{19}	Medial prefrontal cortex	Brain part
X_{20}	Amygdala	Brain part
X_{21}	Primary motor cortex	Brain part
X_{22}	Premotor cortex	Brain part

The connection weights ω_i in Fig. 1 are as follows. The sensor states ss_{ee} , ss_{cc} have two incoming connections from ws_{ee} and ws_c (weights ω_1 , ω_2). The world state of extreme emotion ws_{ee} has one arriving connection from es_{ee} , ω_{11} as a body-loop with weight. The sensory representation state of an extreme emotion srs_{ee} has an incoming

connection weights ω_8 from state preparation state of an extreme emotion ps_{ee} . The feeling state fs_{ee} has one outgoing connection weight ω_5 from srs_{ee} and also five other connections from sensory areas of cerebral cortex such as gustatory cortex, visual cortex, somatosensory cortex, and auditory cortex, ω_{22} - ω_{25} , respectively, these connection weights are considered as the hallucination connections as it has been mentioned earlier in Sect. 2 as a neurological principles. The preparation state of an extreme emotion ps_{ee} has three incoming connection weights ω_6 , ω_7 , and ω_{55} from states srs_c and fs_{ee} , Primary cortex, respectively.

The goal has one arriving connection weight from the sensory representation srs_{ee} (ω_{12}) and preparation state ps_{psil} has an entering connection from the goal with weight ω_{13} . The execution of taking the drug (psilocybin) is called es_{psil} , and has an entering connection weight ω_{14} from preparation state of taking ps_{psil} . The state Thalamus has two entering connection weights ω_{15} , ω_{17} from es_{psil} , and Hippocampus, respectively. The sensory area in the cerebral cortex has four components, gustatory cortex, visual cortex, somatosensory cortex, and auditory cortex. All aforementioned components have outgoing connection to srs_{ee} , with connection weights, ω_{26} - ω_{29} . The Hippocampus in the brain has one incoming connection weight, ω_{16} from Thalamus. The Amygdala has two incoming connection weights from Hippocampus, and medial Prefrontal cortex, with the weights ω_{31} , ω_{33} and it has two outgoing connection weights ω_{32} , ω_{36} , ω_{37} to mPFC, Premotor cortex, and Primary cortex, respectively. Note that the connection weight between states medial Prefrontal Cortex and Hippocampus is adaptive and using Hebbian learning means through time will be changed, in this case the patient learned to cope with the stress and after some period of time she does not need to intake psilocybin. The state es_{psil} has also two outgoing connection weights to Premotor Cortex state and Primary cortex with ω_{37} and ω_{38} . Eventually, the state Primary motor cortex has an outgoing connection weight to ps_{ee} .

This conceptual representation was transformed into a numerical representation as follows [11, Ch 2]:

- at each time point t each state Y in the model has a real number value in the interval $[0, 1]$, denoted by $Y(t)$
- at each time point t each state X connected to state Y has an impact on Y defined as **impact** $_{X,Y}(t) = \omega_{X,Y} X(t)$ where $\omega_{X,Y}$ is the weight of the connection from X to Y
- The *aggregated impact* of multiple states X_i on Y at t is determined using a *combination function* $c_Y(\cdot)$:

$$\begin{aligned} \mathbf{aggimpact}_Y(t) &= c_Y(\mathbf{impact}_{X_1,Y}(t), \dots, \mathbf{impact}_{X_k,Y}(t)) \\ &= c_Y(\omega_{X_1,Y}X_1(t), \dots, \omega_{X_k,Y}X_k(t)) \end{aligned}$$

where X_i are the states with connections to state Y

- The effect of **aggimpact** $_Y(t)$ on Y is exerted over time gradually, depending on speed factor η_Y :

$$\begin{aligned} Y(t + \Delta t) &= Y(t) + \eta_Y[\mathbf{aggimpact}_Y(t) - Y(t)]\Delta t \\ \text{or } dY(t)/dt &= \eta_Y[\mathbf{aggimpact}_Y(t) - Y(t)] \end{aligned}$$

- Thus, the following *difference* and *differential equation* for Y are obtained:

$$Y(t + \Delta t) = Y(t) + \eta_Y [\mathbf{c}_Y(\omega_{X_1,Y} X_1(t), \dots, \omega_{X_k,Y} X_k(t)) - Y(t)] \Delta t$$

$$\mathbf{d}Y(t)/\mathbf{d}t = \eta_Y [\mathbf{c}_Y(\omega_{X_1,Y} X_1(t), \dots, \omega_{X_k,Y} X_k(t)) - Y(t)]$$

For states the following combination functions $\mathbf{c}_Y(\dots)$ were used, the identity function $\mathbf{id}(\cdot)$ for states with impact from only one other state, and for states with multiple impacts the scaled sum function $\mathbf{ssum}_\lambda(\dots)$ with scaling factor λ , and the advanced logistic sum function $\mathbf{alogistic}_{\sigma,\tau}(\dots)$ with steepness σ and threshold τ .

$$\mathbf{id}(V) = V$$

$$\mathbf{ssum}_\lambda(V_1, \dots, V_k) = (V_1 + \dots + V_k)/\lambda$$

Fig. 1. Conceptual representation of the temporal-causal network model

$$\mathbf{alogistic}_{\sigma,\tau}(V_1, \dots, V_k) = [(1/(1 + e^{-\sigma(V_1 + \dots + V_{k-\tau})})) - 1/(1 + e^{\sigma\tau})] (1 + e^{-\sigma\tau})$$

Here first the general Hebbian Learning is explained which is applied to ω_{40} and ω_{41} for (X_5, X_{23}) . In a general example model it is assumed that the strength ω of such a connection between states X_1 and X_2 is adapted using the following Hebbian Learning rule, taking into account a maximal connection strength 1, a learning rate $\eta > 0$ and a persistence factor $\mu \geq 0$, and activation levels $X_1(t)$ and $X_2(t)$ (between 0 and 1) of the two states involved [10]. The first expression is in differential equation format, the second one in difference equation format:

$$\begin{aligned} d\omega(t)/dt &= \eta[X_1(t)X_2(t)(1 - \omega(t) - (1 - \mu)\omega(t))] \\ \omega(t + \Delta t) &= \omega(t) + \eta[X_1(t)X_2(t)(1 - \omega(t))(1 - \mu)\omega(t)]\Delta t \end{aligned}$$

4 Example Simulation

The simulation results of the cognitive temporal causal network model, which was constructed based on the neurological science which contains qualitative empirical information (such as fMRI) both for the mechanism by which the brain components work and for emerging result of the processes, has been shown in Fig. 2.

Fig. 2. Simulation results for temporal-causal network modeling of the therapy by psilocybin

Therefore, one can imply that the best option for declining the stress level has been chosen, given the usage of psilocybin. The model used the Matlab codes which have been implemented in [12]. Using appropriate connections weights make the model numerical and adapted to qualitative empirical information. Table 2 illustrates the connection weights that has been used, where the values for are initial values as these weights are adapted over time. The time step was $\Delta t = 1$. The scaling factors λ_i for the nodes with more than one arriving connection weights are mentioned in Table 2. At

first, an external world state of an extreme emotion-stimuli context c (represented by ws_c) will influence the affective internal states of the individual by influencing the emotional response es_{ee} (via ss_c , srs_c , and ps_{ee}) conducted to show the extreme emotion by body state ws_{ee} . As a consequence, the stressed individual senses the extreme emotion (and at the same time all the biological brain components increased over time), so as a cognitive process, as a next step the goal (using psilocybin) becomes active to decrease this stress level by using psilocybin at time around 10.

Table 2. Connection weights and scaling factors for the example simulation

Connection weight	ω_1	ω_2	ω_3	ω_4	ω_5	ω_6	ω_7	ω_8	ω_{10}	ω_{11}	ω_{12}	ω_{13}
Value	1	1	1	1	1	1	1	1	1	1	1	1
Connection Weight	ω_{14}	ω_{15}	ω_{16}	ω_{17}	ω_{18}	ω_{19}	ω_{20}	ω_{21}	ω_{22}	ω_{23}	ω_{24}	ω_{25}
Value	1	-0.5	1	1	1	1	1	1	0.01	0.01	0.01	0.01
Connection Weight	ω_{26}	ω_{27}	ω_{28}	ω_{29}	ω_{30}	ω_{31}	ω_{32}	ω_{33}	ω_{34}	ω_{35}	ω_{36}	ω_{37}
Value	1	1	1	1	1	0.4	0.4	1	1	0.1	0.1	-0.5
Connection Weight	ω_{38}	ω_{39}	ω_{40}	ω_{41}	ω_{42}	ω_{43}	ω_{44}	ω_{45}	ω_{46}	ω_{47}	ω_{48}	ω_{49}
Value	-0.5	1	1	1	1	1	1	1	1	1	1	1
Connection Weight	ω_{50}	ω_{51}	ω_{52}	ω_{53}	ω_{54}							
Value	-0.5	1	1	1	1							

state	X_5	X_8	X_{14}	X_{15}	X_{17}	X_{18}	X_{19}	X_{20}	X_{21}
λ_i	2.04	3	3.4	3	1.4	2	2	2	2

As a biological process, the goal and in further steps, execution of taking psilocybin activates the alterations and suppression of execution of stress at the first state and this affects other brain components to be less active around time 10. The environment is constant, external input for the model is only the constant world state ws_c . Therefore, based on the simulation results it is illustrated that the model for the drug therapy psilocybin works as expected.

Fig. 3. Simulation results for adaptivity connection weight between amygdala and medial Prefrontal Cortex

The adaptivity connection (Hebbian learning) between two brain parts; Amygdala and medial Prefrontal Cortex (mPFC) is shown in Fig. 3. As it is indicated in the figure the adaptivity, learning to cope with stress and decreasing that over time starts at time around 50 and continues until time 70 to remain constant.

5 Conclusion

In this paper, an adaptive cognitive temporal causal-network model for extreme emotion extinction using psilocybin for patients under extreme emotion is presented. The proposed model can be used to test different hypothesis and neurological principles about the influences of the brain components and hormones and the effects that different brain components have the extinction of extreme emotion, but also on other processes such as fear.

Some simulations have been implemented, one of which was presented in the paper. This model can be used to predict human behavior dynamically in different conditions such as extreme emotion to get insight in such processes and to bring up a certain cure or treatment of individuals to perform the therapies of extreme emotions for post-traumatic disorder individuals. Future of this work can be analysis of other treatments together with this therapy.

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