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A Computational Model for the Second-Order Adaptive Causal Relationships between Anxiety, Stress and Physical Exercise

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Abstract. Mental disorders are more and more seen as based on complex networks of symptoms and predispositions that create the disorder as an emergent behaviour of the network's dynamics. This paper aims to provide a computational model reflecting the adaptive causal relations between anxiety, stress and physical exercise based on a network-oriented modelling approach. The model was evaluated by executing several simulations and validated through an examination of its emergent properties and their cross-reference to the available literature. The created model offers the possibility of simulating different treatments, and offers a basis to develop a virtual patient model.

Keywords: Anxiety, Stress, Physical Exercise, Adaptive Network, temporal-causal

1 Introduction

Everyone likely experiences stress and anxiety from time to time in their life. While the majority will only experience mild non-clinical symptoms, one should still be cautious. Although mild symptoms might not impact one's life substantially, mild symptoms can still have adverse effects on the affected individuals [14]. Furthermore, leaving mild symptoms untreated increases the individual's risk of their anxiety progressing to a clinical state [14]. It is estimated that the prevalence of anxiety symptoms in the general population is around 32% [17], and around 25% of the American population had at least one episode of anxiety [25]. Also, urgency arises from the current stressful situation created through the COVID-19 pandemic and its related effects, as the prevalence of anxiety and related psychological disorders seem to increase [17]. Therefore, to reduce the risk of onsetting severe anxiety, a range of treatment approaches should always be considered. One of such approaches could be physical activity. Not only does this bring general health benefits with it, but it is also something easy and accessible to reduce one's symptoms without extra medication [9, 14, 25].

In the classical psychopathological model, it is assumed that the disorder causes the combination of symptoms to appear, as it requires both a categorical and dimensional

latent classification for symptoms of psychological disorders and does not take causal relations between the symptoms into account. Lately, however, a network-oriented approach towards psychological disorders was proposed. This approach offers another view, stating that a mental disorder is not the reason for its underlying symptoms to co-appear, but a mental disorder is based on a network of distinct symptoms, dynamically interacting with each other ultimately causing the emergent behaviour classified as, e.g., Anxiety [4, 6, 10, 15]. In this effort, given the complexity of the underlying neurobiological mechanisms [25] and the necessity of further research in the domain, an integrated approach showing changes in anxiety in response to physical activity is of great value enabling to simulate the effects of different exercise frequencies.

2 Literature Overview

Many meta-studies have observed a significant but modest anxiety-reducing effect of physical exercise [13-14, 25] and an overall increase in one's mood [25]. However, there still is a debate if clinical or non-clinical populations benefit more from the effects of physical exercising [14, 25]. Physical exercise however should still be promoted given its preventative and rehabilitative qualities, through direct and indirect effects [13-14, 25], concerning brain-related psychiatric disorders. Although exercise always has a positive effect on one's emotions [8], its anxiety-reducing effects depend on the intensity of the exercise. Research has shown that the anxiolytic effect of exercise is highest when it is performed under the ventilatory threshold, the threshold at which the breathing rate increases disproportionally regarding the oxygen uptake. Training above this threshold reduces the mood-increasing effects of the exercising [8]. The literature here suggests that aerobic training of around 20 to 35 minutes is most beneficial [8, 13, 25], with first positive effects on an individual's mood already showing after 15 to 20 minutes [8]. Therefore, a timescale of 30 minutes is considered to induce exercises' anxiolytic effect, considering the above-mentioned factors.

Research additionally has shown that physical exercise leads to a positive effect on an individual's stress response [9, 25]. Due to a positive correlation between anxiety and stress [11-12], this also effects one's anxiousness. This correlation already begins to show with only mild stress symptoms [11]. Further studies have also shown a reduction of the activation of the general stress response in physically active individuals [9]. A reduction in amplitude of the stress response and the time needed to recover to baseline levels, could also be observed in physically active individuals [25]. This is important given a stress response is mostly defined by the individuals' ability to quickly recover to the baseline following a stressful event [3]. Furthermore, the interconnection of stress and anxiety urges one to also address indirect effects on anxiety.

In a typical stress response, when a stressor is apparent, the threat recognising brain regions (amygdala, prefrontal cortex, and hippocampus) will initiate the release of Corticotrophin-releasing factors (CRFs). This release triggers the production of glucocorticoid in the HPA Axis leading to the release of cortisol and the production of epinephrine. Usually this is described as the "Fight or Flight response" of the body [20]. The ways in which exercise influences this stress response are multifold [9], and seem to be

of both psychological and neurophysical nature [14]. Part of the neurophysical changes can be united under their effects on the biological stress response. One of these effects can be seen in a reduction of anxiety through effects on the hypothalamic-pituitary adrenal axis (HPA axis) [25] Another can be observed in an increase of the threshold that needs to be surpassed by stressful events to induce biological stress response, due to a reduction in the likelihood of the sympathetic nervous system's activation [9]. This is mostly based on changing baseline cortisol levels [13, 25] and a reduction of the level of cortisol in an active stress response [13]. Other long-term changes of the HPA Axis through exercise also include inhibition of cortisol synthesis and a higher functioning of mineralocorticoid receptors [13]. This suggests an decreased sensitivity of the HPA axis due to an increased activation threshold following physical exercise.

Other effects of neurological nature indirectly influence the psychological responses and emotion regulation abilities [19]. Physical exercise does not only increase blood flow and the oxygenation and metabolism of the brain, it also induces the release of opioids and endocannabinoids, which were shown to have a direct anxiolytic effect [13]. Furthermore, it induces the release of neurotrophic factors [13, 25] and the synthesis of several neurotransmitters (BDNF, IGF-1, WEGF, NT3, FGF-2, GDNF, EGF and NGF) [13]. The release of these neurotransmitters and neurotrophic factors has several effects. First the neurotransmitter release contributes to a less suppressed cortical activity [13]. Second, neurogenesis-reducing effects of stress and neurodegenerative diseases are counteracted by an increased neurogenesis, attributable to a higher neurotrophin availability. Third, the ability to adapt to stress is increased due to an increased neuroplasticity induced by the neurotrophic factors [13]. The improved neuroplasticity also strengthens the adaptivity of the ventromedial prefrontal cortex, a brain region significantly involved in the emotional and behavioural control network [19]. Furthermore, the release of brain-derived neurotrophic factors (BDNF) due to exercise also leads to a better responsivity to environmental stress, given BDNF's enhancing effect on the synaptic connectivity and signal transduction [21]. Therefore, this research indicates that one's emotion regulation ability is indirectly influenced by exercise induced release of neurotransmitters and neurotrophic factors.

Drawing on the above-mentioned correlations and underlying mechanisms, this paper aims to contribute to the research effort by providing a computational model. Surprisingly, until today the above-mentioned factors have not yet been incorporated into an computational model, despite the growing interest in an network-oriented approach of modelling neurophysical diseases [4, 6, 10, 15]. While the created model should offer the possibility of simulating different treatment procedures, it could later also be used as a possible basis or extension of a virtual agent model representing and simulating a patient.

3 The Adaptive Computational Network Model

3.1 The modelling approach used

The adaptive computational network model introduced here was developed using the adaptive network-oriented modeling approach described in Treur [23-24], which is based on self-modeling network models and a dedicated software environment. The adaptive computational network model's dynamics can be defined by a number of network characteristics: its connections and their weights, its timing via speed factors, and the aggregation characteristics as follows.

Connections
The connections between nodes in this type of network represent a translation of real-world causal relations into a network structure. They are defined by the nodes X and Y they connect and by their weight $\boldsymbol{\omega}_{X,Y}$ demonstrating the strength of that connection. In adaptive networks, this weight is represented by another (self-model) node (see below).

Timing
The model's timing is determined by the speed factors $\boldsymbol{\eta}_Y$ of each node Y, which can be understood as indication of the node's rate of change.

Aggregation
The type of aggregation chosen determines how multiple incoming inputs are combined into one effect on the destination node. For this, various combination functions $\mathbf{c}_Y\mathbf{(..)}$ are provided by a dedicated library.

Standard difference equation
All network nodes Y (also called states) have time-dependent values $Y(t)$ where t indicates time. Based on the network characteristics for connectivity, aggregation and timing defined above, the following difference equation defines the network's dynamics for any state Y:

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

Here, $X_1, \ldots, X_k$ are the network states from which Y has incoming connections.

Higher-order self-models
Self-models can represent adaptive characteristics of the network. These usually consist of self-model states, also called reification states [24]. Typically, the weight $\boldsymbol{\omega}_{X,Y}$ of a connection or a node's speed factor $\boldsymbol{\eta}_Y$ can be made adaptive through including a self-model state named $\mathbf{W}_{X,Y}$ (for connection weight) or $\mathbf{H}_Y$ (for speed factor) for it. However, self-model states can also represent other network characteristics like the parameters of combination functions.

Combination functions

The dedicated software environment comes with a pre-selection of many (more than 45) commonly used combination functions with differing parameters and use cases. A selection of combination functions used in the introduced model is given here.

The *identity combination function* **id(..)** is mostly used to directly transfer the activation of the source node to the receiving node or in a circular manner of a node to itself to model persistence of activation by keeping the already reached numerical value. It has no parameters, and its formal definition is as follows:

$$\mathbf{id}(V) = V \tag{2}$$

The *Hebbian learning combination function* $\mathbf{hebb}_{\boldsymbol{\mu}}(..)$ is used to model the Hebbian learning principle. It has one parameter $\boldsymbol{\mu}$, which describes the persistence factor. Its formal definition is as follows:

$$\mathbf{hebb}_{\boldsymbol{\mu}}(V_1, V_2, W) = V_1 V_2 (1-W) + \boldsymbol{\mu}\, W \tag{3}$$

The *advanced logistic sum combination function* $\mathbf{alogistic}_{\boldsymbol{\sigma},\boldsymbol{\tau}}(V_1, \ldots, V_k)$ is an often-used function to combine inputs when a node has multiple incoming connections. It has two parameters $\boldsymbol{\sigma}$, which describes the steepness of the logistic function, and $\boldsymbol{\tau}$, which describes the threshold. Its formal definition is as follows:

$$\mathbf{alogistic}_{\boldsymbol{\sigma},\boldsymbol{\tau}}(V_1, \ldots, V_k) = \left[\frac{1}{1+e^{-\boldsymbol{\sigma}(V_1+\cdots+V_k-\boldsymbol{\tau})}} - \frac{1}{1+e^{\boldsymbol{\sigma}\boldsymbol{\tau}}}\right](1+e^{-\boldsymbol{\sigma}\boldsymbol{\tau}}) \tag{4}$$

Additionally, two custom *step mod functions* will be used to create cyclically recurring inputs; see the Appendix [2].

Software environment used

Designed network models are usually specified in a standard table format, called the role matrices format, for which examples and explanations can be found in [24], Ch. 9. Given these specifications and each state's initial values, the simulation can be performed using the provided dedicated software environment, implemented in the form of MatLab scripts and functions; for more details, see [24], Ch. 9. The script for simulation of adaptive (self-modeling) network models used here will then calculate the state values over time based on the dynamics for the states described by equation (1), thereby picking the values for the adaptive characteristics from the self-model states representing them, and gives the possibility to export the generated data for further investigation and visualization.

3.2 The designed adaptive self-modeling network model

Following the approach to Network-Oriented Modelling described in [24], a network model was created based on the literature review. The graphical representation of the connectivity of this model is depicted in Fig. 1, while Table 1 describes the states shown

in Fig. 1 in more detail. The model consists of 19 states, of whom twelve represent base level states, three portray first-order self-model states and four display second-order self-model states. The states in the first reification level (or first-order self-model level) represent the principle of Hebbian learning for two connections at the base level, while the states in the second reification level (or second-order self-model level) express metaplasticity, namely persistence of what is learned and the speed of learning.

The base-level causal relations can be described according to two groups, one concerning the general stress and anxiety response, the other regarding the effects of physical exercise on the general stress and anxiety response. The general stress and anxiety response is mostly defined by the biological stress response and psychological emotion regulation. Like Fig. 1 shows, a stressor (**ws**) will lead to the preparation of the stress response (**pssr**), which will then have two effects. On one side, it will trigger the biological stress response (**bsr**) through activation of the HPA axis and the release of cortisol. On the other side is the psychological stress response, depicted in the stress regulation control state (**sr**). The biological and psychological stress response together will then cause the stress feeling (**sf**) in the individual, which through further causal relations will cause the feeling of anxiety (**anx**)**,** which is controlled by the anxiety regulation control state (**ar**).

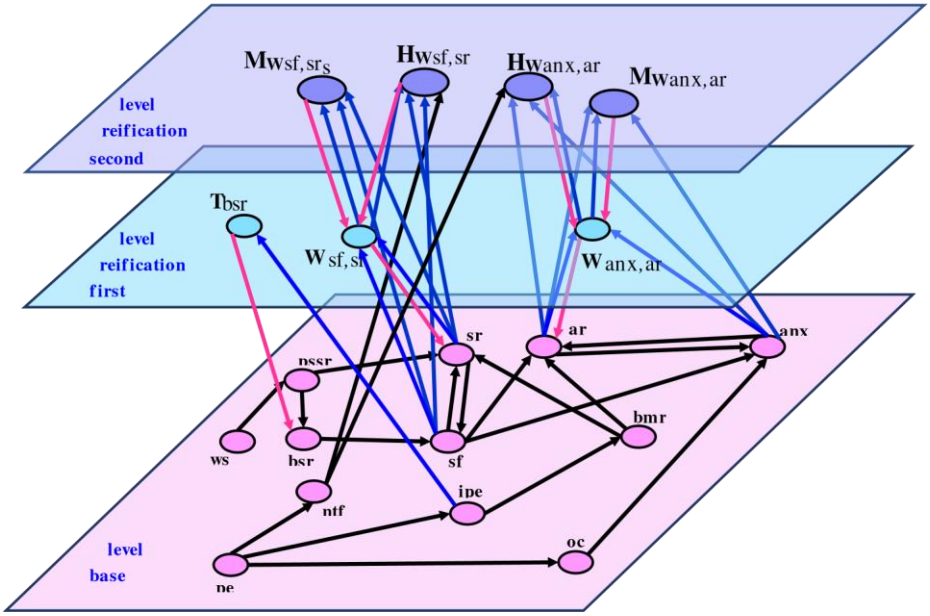

Fig. 1. Connectivity of the second-order adaptive network model concerning anxiety and stress emotion regulation covering plasticity and metaplasticity. Interlevel relations are depicted by upward connections (blue) and downward connections (pink-red). With a base level (pink plane) depicting the basic causal relations (black arrows), a first reification level (blue plane) depicting neuroplasticity and a second reification level (purple plane) depicting metaplasticity.

Both the causal relation between the stress feeling and the stress regulation, as well as the connection between the anxiety feeling and the anxiety regulation, are, however, influenced by the first-order self-model states. In this model, they are used to represent the Hebbian learning principle '*what fires together, wires together*' ([18], p. 64). Therefore, the weight of the connection from the feeling towards its respective control state was made adaptive by introducing the self-model states **W**. The **W**-states, representing the plasticity that is described by Hebbian learning, furthermore are influenced by a second-order self-model state representing the concept of metaplasticity. The respective **H**-states represent the speed factor η of **W,** while the **M**-states represent the persistence factors μ of **W**. The **H**-states intend to represent the principle of an increased adaptation speed and therefore learning speed. Research has shown that the adaptation speed itself shows adaptive characteristics, most notably an acceleration of the adaptation process if stimulus exposure increases [16], also known as meta-adaptation [1]. In this model, the **H**-states' activation should increase in value relative to its exposed stimuli. Therefore, such an increased level of stimulus exposure should relate to an increase in the learning speed over time. In this model, the **H**-state values are further influenced by neurotrophic factors (**ntf**). These lead to an increased adaptation speed, which will translate into improved neuroplasticity.

Table 1. Description of the states of the second-order adaptive network model

State nr	State name	Explanation	Level
X_1	ws	World state for stressor	Base level
X_2	pssr	Preparation state of stress response	
X_3	bsr	Biological Stress Response (HPA Axis)	
X_4	sf	Feeling of Stress	
X_5	sr	Stress Regulation control state	
X_6	anx	Feeling of Anxiety	
X_7	ar	Anxiety Regulation control state	
X_8	bmr	Behavioural and Mood Responses	
X_9	ipe	Indirect Effects of Physical Exercise	
X_{10}	pe	Physical Exercise	
X_{11}	ntf	Neurotrophic Factors	
X_{12}	oc	Opioids and Cannabinoids	
X_{13}	$\mathbf{T}_{bsr}$	First-order self-model state for the Biological Stress Responses activation treshold	First-order self-model level
X_{14}	$\mathbf{W}_{anx,ar}$	First-order self-model state for connection weight $\omega_{anx,ar}$	
X_{15}	$\mathbf{W}_{sf,sr}$	First-order self-model state for connection weight $\omega_{sf,sr}$	
X_{16}	$\mathbf{HW}_{anx,ar}$	Second-order self-model state for speed factor $\eta\mathbf{W}_{anx,ar}$ first-order self-model self-model state $\mathbf{W}_{anx,ar}$	Second-order self-model level
X_{17}	$\mathbf{MW}_{anx,ar}$	Second-order self-model state for persistence factor parameter $\mu\mathbf{W}_{anx,ar}$ first-order self-model self-model state $\mathbf{W}_{anx,ar}$	
X_{18}	$\mathbf{HW}_{sf,sr}$	Second-order self-model state for speed factor $\eta\mathbf{W}_{sf,sr}$ first-order self-model self-model state $\mathbf{W}_{sf,sr}$	
X_{19}	$\mathbf{MW}_{sf,sr}$	Second-order self-model state for persistence factor parameter $\mu\mathbf{W}_{sf,sr}$ first-order self-model self-model state $\mathbf{W}_{sf,sr}$	

The above-described network, however, can be influenced by the effects of physical exercise. Physical exercise affects the stress and anxiety response of an individual in both a direct and an indirect manner.

The indirect effect of physical exercise is represented by the ipe state, which influences both the HPA axis sensitivity through its threshold (represented by self-model state $\mathbf{T}_{\mathbf{bsr}}$) and the behavioural and mood responses (**bmr**). The indirect effect of physical exercise on the HPA axis is based on an increase of the biological stress responses threshold, while the behavioural and mood responses elevate the emotion regulation ability. The **T**-state in this network models influences the excitability of the Biological Stress Response related neurons. These influences result in a modification of the neuron's response to the triggering synaptic activity [5, 7]. Therefore, an increase in the value of the self-model state $\mathbf{T}_{\mathbf{bsr}}$ would result in lessened excitability of the HPA axis activating neurons. This should result in a changed reaction of the Biological Stress Response state (**bsr**) to input it receives from the stress response preparation state (**pssr**).

The direct effects of physical exercise on stress and anxiety, comprise the release of Opioids and Cannabinoids (**oc**), which directly reduce the feeling of anxiety and the release of neurotrophic factors (**ntf**), which increase the brains' neuroplasticity. In the model, this causal relation is realized through a direct increasing influence of the neurotrophic factors on the **H**-states, representing the learning speed.

4 Simulations

To obtain a full detailed design description of all characteristics of the model, it was specified in a standardized form in role-matrices format. This format describes all characteristics that define the model: the connections and their weights, the speed factors and the combination functions used with their parameters. Moreover, the available dedicated software environment can use these matrices as input and run simulations for the model from it. Table 2, 3, 4, 5 in the Appendix [2] show the role-matrices for the first run scenario.

To explore the claim of a reductive effect of physical exercise on anxiety, three different scenarios depicting different levels of physical activity integration into the individual's life are performed. The simulation will represent the time of 8:00 until 22:00 over three days. Each time point in the simulation corresponds to a timeframe of five minutes, resulting in a total of 504 Steps in our simulation. In each scenario, the individual will be presented with a stressor from 12:30 until 13:30 to observe its effect on the model's different states. In Scenario 2 (below) and 3 [2], one or more physical exercises of 30 minutes will be added to each day.

Scenario 1: No Exercising

In the first scenario, the individual will be confronted with a stressor, lasting one hour, each of the three days, with the initial values chosen to depict an individual that already has a higher base level of stress. While the simulation's full result can be seen in Fig. 9

in Appendix [2], an examination of the results based on different parts of the model will take place. For this, the general model will be divided into three sections, one for the stress response, one for the anxiety response and one for the effects of physical exercise. For the first scenario however, given no physical exercise is present, only the stress and anxiety response will be inspected. In Fig. 2 (upper graph), the stress response and its related states are shown. As expected, the preparation state for the stress response gets triggered when a stressor is present, which subsequently also relates to an activated biological stress response and feeling of stress. With a slight delay, this results in activation of the Stress Regulation Control State, increases the adaptive connection weight (**W**-stress) and speed factor (**H**-stress).

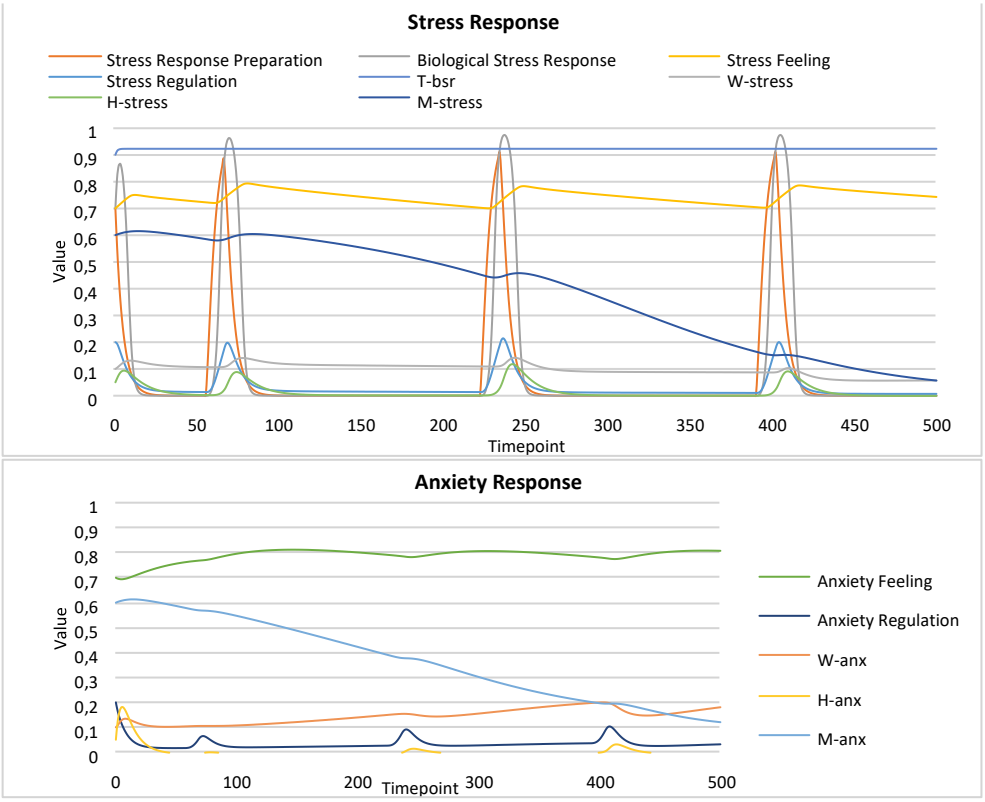

Fig. 2. Graphical Representation of the Stress Response (upper graph) and Anxiety Response (lower graph) in Scenario 1

These increases are triggered as part of the Hebbian learning process, given the simultaneous firing of the feeling and control state. Moreover, the Activation Threshold of the Biological Stress Response (**T**-bsr) stays unaffected and constant, given no physical exercise is performed. As expected, the stress response is of similar nature each day and the level of stress persists over time. Furthermore, the constant high-stress level results in a constant decrease in the persistence factor (**M**-stress) virtually blocking

learning. The process only shows plateauing while the Hebbian learning process is triggered.

Contrary to the stress response, the anxiety response (lower graph in Fig. 2) is only slightly affected by the periodic occurrence of the stressor. Due to the direct correlation between stress and anxiety, this is expected behaviour. The stress feeling state does not show much volatility over time which is reflected in the correlated anxiety state. Therefore, effects shown by the Hebbian learning states of the anxiety response occur in a significantly lower amplitude when compared with the stress responses Hebbian Learning states.

Scenario 2: Occasional Workout

In the second scenario (see Fig. 3), the individual will again be confronted with a one-hour lasting stressor each of the three days.

The initial values will again be chosen to reflect an individual with a higher baselevel of stress. Contrary to the first scenario, however, a physical exercise of 30 minutes at 10:00 in the morning will be added. This corresponds to the physical exercise state being activated during the timepoints 24-30, 192-198, 360-366. For this we will need to change the function parameter specifications according to Table 6 in (Appendix, 2021). The other specifications stay the same. To better understand the model's dynamics, the behaviour of the different subsystems will again be examined separately. However, the simulation's full result can also be seen in Fig. 10 of (Appendix, 2021). As can be seen in Fig. 3, similar responses to the stressor as in Scenario 1 take place. However, these processes are influenced and disrupted to an extend by the effects of physical exercise. As expected better behavioural and mood responses and the decreased sensitivity of the HPA axis result in a decreasing base level of the feeling of stress after physical exercise. This process is likely aided by strengthened Hebbian learning principles.

Another drastic change compared to Scenario 1 can be seen in the processes connected to Hebbian learning. As the release of neurotrophic factors increases the neurogenesis, the learning speed (**H**-stress) increases significantly, which as a result also keeps the persistence factor (**M**-stress) on an stable but slightly increasing level. Interestingly, however, the connection weight (**W**-stress) between the stress feeling and its emotion regulation control state, returns to a stable base level once outside influences subside. This suggests that there are not yet long-term persisting learning effects, at this level of exercise integration in one's life. Further, an increase in the HPA axis activation threshold (**T**-bsr) can be seen following physical activity. However, given the threshold returns to its baselevel at the time the stressor activates the biological stress response, no reduction in the response can be observed.

Contrary to the first scenario, the feeling of stress in this scenario was more volatile, resulting in higher volatility of the states connected to the anxiety response. As expected, the periodically decreased feeling of stress and the increased behavioural and mood responses after physical activity led to a decreased feeling of anxiety after exercises. This is aided by the anxiolytic effects of opioids and cannabinoids that are

released as a result of physical exercise. Moreover, the decrease of the stress feeling over time translates to a decrease of anxiety over time.

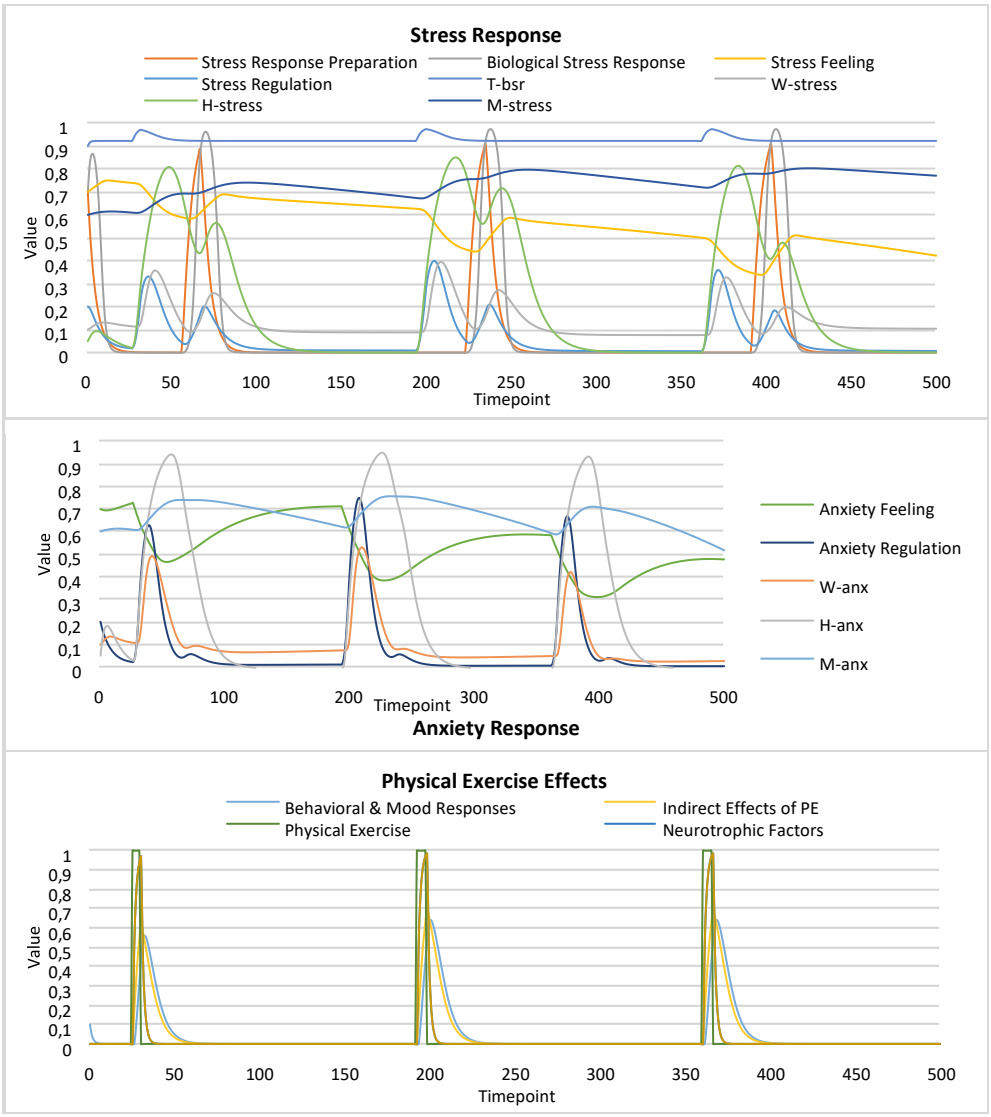

Fig. 3. Graphical Representation of the Stress Response (upper graph) and Anxiety Response (middle graph) and physical exercising (lower graph) in Scenario 2

Furthermore, the increased metaplasticity induced by the neurotrophic factors leads to a periodically increased learning speed (**H**-anx) and connection weight (**W**-anx) of

the anxiety feeling and regulation connection. This causes the persistence factor to rebound to higher levels in these times of increased activation of the Hebbian learning processes, leading to an overall fluctuating but only minimally decreasing persistence factor over time. As expected, these influences lead to a reduction of the anxiety base level.

As Fig. 3, lower graph shows, physical exercise induces the acute release of neurotrophic factors as well as opioids and cannabinoids. Through its indirect effects, physical exercise increases behavioural and mood responses for a short timeframe after the physical exercise ended.

5 Discussion

This paper aimed to describe the creation of a adaptive computational temporal-causal network model for the effects of physical exercise on stress and anxiety based on real-world causal relations. According to the proposed paradigm shift in the view of psychological disorders as dynamic networks [4, 6, 10, 15]. The model was built to recreate the known patterns as indirectly emerging from adaptive causal relations. The network model incorporates three types of adaptation principles to recreate Hebbian learning, excitability adaptation and adaptive adaptation speed.

By the use of the network model and a dedicated software environment implemented as a MatLab script, three different scenarios were simulated, depicting different levels of integration of physical exercise into the individual's life. The first scenario portrayed three days of an individual being confronted with a stressor each day with no physical exercise and showed that the stress and anxiety level will always return to the same base level. In the second scenario, a workout of 30 minutes in the morning was added to the individual's routine, which, through direct and indirect effects, lead to a visible reduction of the stress and anxiety base levels over time. In the third scenario, in addition to the morning exercise, another workout in the evening was added. This resulted in a further extended reduction of the anxiety and stress base levels throughout the simulation.

Overall, the model successfully showed emergent behaviour reflecting the empirical findings in the discussed literature, confirming its validity. Further this research contribution enables a visualisation of the underlying effects of physical exercise on stress and anxiety. While this model, by the generated simulation graphs, confirms that physical exercise could positively influence the treatment of anxiety and should be promoted, it could also act as a basis or extension of future virtual agent models to display these outcomes in a more relatable manner. Nevertheless, the presented model is not without limitations. While it reflects the causal relations on a macroscopic level, a more advanced model could incorporate the underlying mechanisms of the biological stress response and the emotion regulation in greater detail. Additionally, to create a holistic representation of the complex biological and neurological interdependencies of psychological disorders, a combination with additional computational models in this domain could be added as well.

Taking into consideration the numerous imbricating symptoms of anxiety and other psychological disorders like depression, further research may focus on the creation of a generalized model representing typical neurological and biological symptoms of serotonin and dopamine availability impacting disorders. This would enable broader possibilities in simulations and increase the comparability amid different models.

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