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An Adaptive Computational Fear-Avoidance Model Applied to Genito-Pelvic Pain/Penetration Disorder

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Abstract. This paper presents a first study to apply a computational approach to Genito-Pelvic Pain/Penetration Disorder (GPPPD) using a Fear Avoidance Model. An adaptive temporal-causal network model for fear avoidance was designed and therapeutic interventions were incorporated targeting one or two emotional states. Validation with empirical data shows that for one type of individual therapeutic intervention targeting two states can reduce pain and other complaints. For three other types of individuals, targeting two emotional states was not sufficient to reduce pain and other complaints. The computational model can address large individual differences and supports the claim that interventions for GPPPD should be multidisciplinary.

Keywords: Genito-pelvic pain/penetration disorder · Pain disorder · Fear avoidance model · Computational modelling · Adaptive temporal-causal network

1 Introduction

Genito-Pelvic Pain/Penetration Disorder (GPPPD) is a prevalent sexual dysfunction affecting approximately 20% of heterosexual women [18], but underlying mechanisms are still poorly understood. Studies suggest that treatment should be based on multidisciplinary interventions that take into account individual differences [8, 10, 21]. Thomtén and Linton [27] approached GPPPD as a pain disorder by applying the Fear Avoidance Model of Vlaeyen and Linton [30] to the disorder. This approach might be helpful to better understand how sexual pain starts and what interventions could be useful. This is the first study to apply a computational approach to GPPPD using the Fear Avoidance Model. An adaptive temporal-causal network model was designed and therapeutic interventions were incorporated targeting one or two emotional states.

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Validation with empirical data of Pazmany et al. [20] shows that only for one type of individual a therapeutic intervention targeting two emotional states reduces pain and other complaints, although recovery does not go back to baseline. For three other types of individuals, targeting two states was not sufficient to reduce pain and other complaints. This computational model can address large individual differences and supports the claim that interventions for GPPPD should be multidisciplinary. The model has the potential to be expanded to see how many states should be targeted for a specific individual. First, some background information is presented, after which the computational model is described. Next, simulation outcomes are reported followed by a description of how the model was verified and validated.

2 Background

In this section, some background information on GPPPD and the Fear-Avoidance model is given in order to facilitate a better understanding and interpretation of the computational model.

Genito-Pelvic Pain/Penetration Disorder and Vulvodynia. GPPPD is a relatively new diagnostic category of female sexual dysfunction, introduced in the DSM-5 [2]. It reflects the combination of two previous categories, dyspareunia and vaginismus, in one entity [10]. One of the following criteria have to be met for diagnosis, with at least six months duration and presence of clinically significant distress: difficulties during vaginal penetration during intercourse, marked culcovaginal or pelvic pain during vaginal intercourse or penetration attempts, marked fear or anxiety about vulvovaginal or pelvic pain in anticipation of, during, or as a result of vaginal penetration, and marked tensing or tightening of pelvic floor muscles during attempted vaginal penetration [2].

Implications. Sexual pain disorders have co-morbidity with other disorders and diseases, both physical [14, 21, 24], and mental [1, 5, 17, 20]. In addition, repeated pain during coitus has a substantial negative impact on quality of life [3, 12, 15, 28], and altered sexual functioning [6, 19, 20, 22, 23].

Interventions. The etiology of GPPPD is multi-factorial and complex, which means that biological, psychological and relational factors interact to perpetuate and maintain a women's pain response [10]. GPPPD should thus never be viewed as a purely medical or psychogenic problem but always be evaluated and treated from a biopsychosocial perspective [14, 21]. GPPPD often also impacts the partner relationship and therapy may benefit from also including the partner [9]. Cognitive and behavioral interventions – either with the women, with the partners or in group – can be useful in treating sexual pain disorders, although with varying results [4, 9, 11, 13, 25, 26]. In sum, there are different therapies but there currently is not one therapy effective for all individuals. People with sexual pain disorders generally try many different treatment modalities, often over the course of many years, before experiencing any significant relief [7].

Multidisciplinary treatment in chronic pain has held strong support but is relatively new for sexual pain specifically. In order to create specific multidisciplinary treatment programs that fit for individual cases, the underlying mechanisms of vaginal pain should be better understood. There still is a lack of theoretical models that describe the psycho-social mechanisms involved in the development of GPPPD.

The Fear Avoidance Model. GPPPD is classified as a sexual dysfunction and is thereby the only pain disorder outside the category of 'pain disorders'. Vlaeyen and Linton [30] introduced the fear-avoidance (FA) model to understand musculo-skeletal pain disorders in the transition from acute to chronic pain. Thomtén and Linton [27] have reviewed, adapted and extended the Fear Avoidance Model in the light of pain during vaginal penetration.

This adapted FA model could thus be helpful in understanding GPPPD as a multifaceted sexual disorder but also as a pain disorder. Thomtén and Linton [27] state that the model needs to be further examined by evaluating interventions targeting the specific concepts (e.g., fear, catastrophizing). Figure 1 shows the adapted version of the Fear Avoidance Model presented by Thomtén and Linton [27]. This informal model will be used as a basis for our computational model in order to get more insight in the mechanisms underlying GPPPD and the possibilities for intervention.

The computational model is similar, though adjusted in a few ways. The nodes representing a verb have been changed to an emotional state of being. The arrows represent the actions, and the nodes represent a state of (emotional) being. Furthermore, the branch 'exiting' the fear-avoidance loop (recovering from GPPPD) has been removed, because in the computational model this occurrence will be represented by low values of pain, fear et cetera.

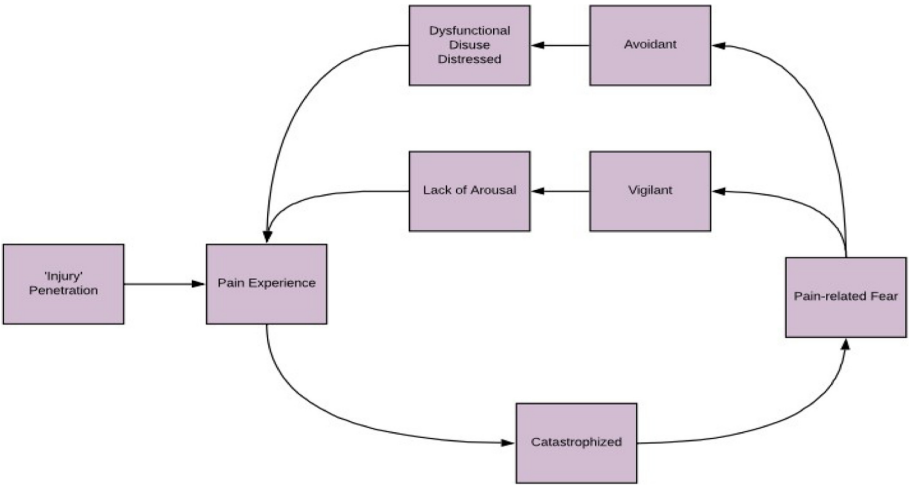

Fig. 1. Adjustment of the fear avoidance model to GPPPD. Adapted from [27].

3 The Designed Computational Network Model

In this section the adaptive temporal-causal network model is presented; see Fig. 2.

The Modeling Approach Used. The adaptive computational model is based on the Network-Oriented Modelling approach based on reified temporal-causal networks [29]. The *network structure characteristics* used are as follows. A full specification of a network model provides a complete overview of their values in so-called role matrix format.

- **Connectivity:** The strength of a connection from state X to Y is represented by weight $\boldsymbol{\omega}_{X,Y}$
- **Aggregation:** The aggregation of multiple impacts on state Y by combination function $\mathbf{c}_Y(..)$.
- **Timing:** The timing of the effect of the impact on state Y by speed factor $\boldsymbol{\eta}_Y$

Given initial values for the states, these network characteristics fully define the dynamics of the network. For each state Y, its (real number) value at time point t is denoted by $Y(t)$. Each of the network structure characteristics can be made adaptive by adding extra states for them to the network, called *reification states* [29]: states $\mathbf{W}_{X,Y}$ for $\boldsymbol{\omega}_{X,Y}$, states $\mathbf{C}_Y$ for $\mathbf{c}_Y(..)$, and states $\mathbf{H}_Y$ for $\boldsymbol{\eta}_Y$. Such reification states get their own network structure characteristics to define their (adaptive) dynamics and are depicted in a higher level plane, as shown in Fig. 2. For example, using this, the adaptation principle called Hebbian learning, considered as a form of plasticity of the brain in cognitive neuroscience ("neurons that fire together, wire together") can be modeled; e.g., see [29], Ch 3, Sect. 3.6.1.

A dedicated software environment is available by which the conceptual design of an adaptive network model is automatically transformed into a numerical representation of the model that can be used for simulation; this is based on the following type of (hidden) difference of differential equation defined in terms of the above network characteristics:

$$Y(t+\Delta t) \;=\; Y(t) + \boldsymbol{\eta}_Y[\mathbf{aggimpact}_Y(t) \text{ - } Y(t)]\Delta t \text{ or } \mathrm{d}Y(t)/\mathrm{d}t \;=\; [\mathbf{aggimpact}_Y(t) \text{ - } Y(t)] \quad \text{with } \mathbf{aggimpact}_Y(t) = \mathbf{c}_Y(\omega_{X_1,Y}X_1(t),\ldots,\omega_{X_k,Y}X_k(t)) \tag{1}$$

where the X_i are all states from which state Y has incoming connections. Different combination functions are available in a library that can be used to specify the effect of the impact on a state (see Treur, 2016, 2020). The following two are used here:

- the *advanced logistic sum* combination function with steepness $\boldsymbol{\sigma}$ and threshold $\boldsymbol{\tau}$

$$\mathbf{alogistic}_{\sigma,\tau}(V_1,\ldots,V_k) \;=\; \left(\frac{1}{1+\mathrm{e}^{-\sigma(V_1+\cdots+V_k-\tau)}} \text{ - } \frac{1}{1+\mathrm{e}^{\sigma\tau}}\right)(1+\mathrm{e}^{-\sigma\tau}) \tag{2}$$

- the *Hebbian learning combination function* $\mathbf{hebb_{\mu}(..)}$

$$\mathbf{hebb_{\mu}}(V_1, V_2, W) = V_1 V_2 (1 - W) + \boldsymbol{\mu} W \quad (3)$$

with $\boldsymbol{\mu}$ the persistence parameter, where V_1 stands for $X(t)$, V_2 for $Y(t)$ and W for $\mathbf{W}_{X,Y}(t)$, where X and Y are the two connected states

The Introduced Adaptive Network Model. The specific adaptive network model introduced here consists of 13 nodes or states and 22 connections; see Fig. 2. The 13 states of the adaptive network model are explained in Table 1. Each node stands for a physical or emotional (re)action or experience and the connections represent causal relations. For example, an occurring injury will cause a pain experience, so an arrow points from the node injury to the node pain experience. In simulations by this model, a spiral can be found of how an experience of pain can cause a closed loop resulting in a continuing non-descending pain experience. Note that the dysfunction, disuse and distress state were simplified. In addition, the link between disuse and increased levels of pain has been theorized, but not supported with empirical data [27]. The full specification of the network characteristics of the introduced network model (connection weights $\boldsymbol{\omega}_{X,Y}$, speed factors $\boldsymbol{\eta}_Y$, and combination functions $\mathbf{c}_Y\mathbf{(..)}$ and their parameters $\boldsymbol{\sigma}$, $\boldsymbol{\tau}$, and $\boldsymbol{\mu}$) and the initial values can be found in the role matrices in the Appendix at https://www.researchgate.net/publication/338410102. The states X_1 to X_8 are also displayed in Fig. 1. The other states X_9 to X_{12} shown in Fig. 2 in addition address emotion regulation by control states, strengthening of emotion regulation by learning, and therapy to support that. In this model, there are control states for both the catastrophized state and the dysfunction/distress state. The graphical representation shown in Fig. 2 displays the overall *connectivity* of this network model, also shown in role matrix **mb** in the abovementioned Appendix.

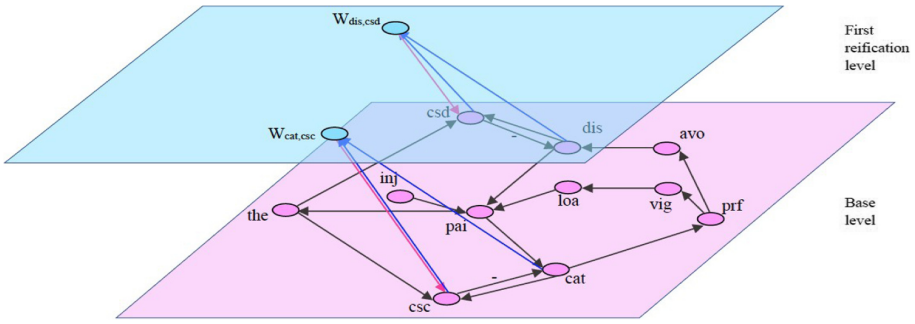

Fig. 2. Overview *of the reified network architecture for plasticity and meta*-plasticity with base level (lower plane, pink) and first reification level (upper plane, blue) and upward causal connections (blue) and downward causal connections (red) defining inter-level relations. (Color figure online)

As can be seen in role matrix **mcw** in the Appendix, most connection weights are positive, the only exceptions being the weights of the connections from control states X_{10} and X_{12} to emotion states X_3 (catastrophized) and X_8 used for emotion regulation.

Table 1. Representation of all states used in the adaptive temporal-causal network

State number	State name	Description	Level
X_1	inj	injury	Base level
X_2	pai	experienced pain	
X_3	cat	catastrophized	
X_4	prf	pain-related fear	
X_5	vig	vigilant	
X_6	loa	lack of arousal	
X_7	avo	avoidance	
X_8	dis	distressed	
X_9	the	therapy	
X_{10}	csc	control state for catastrophizing	
X_{12}	csd	control state for distress	
X_{11}	$\mathbf{W}_{\text{cat,csc}}$	reified representation state for connection weight $\boldsymbol{\omega}_{\text{cat,csc}}$	First reification level
X_{13}	$\mathbf{W}_{\text{dis,csd}}$	reified representation state for connection weight $\boldsymbol{\omega}_{\text{dis,csd}}$	

If one node negatively affects another node, in the picture also an arrow points to the affected node, but labeled with a negative sign (-). For *aggregation*, the combination function $\mathbf{hebb}_{\boldsymbol{\mu}}(..)$ is used for the two **W**-states X_{11} and X_{13} in the upper plane and $\mathbf{alogistic}_{\boldsymbol{\sigma},\boldsymbol{\tau}}(..)$ for all other states in the base plane (role matrix **mcfw** in the Appendix). The values for parameters $\boldsymbol{\sigma}$, $\boldsymbol{\tau}$ and $\boldsymbol{\mu}$ for these combination functions can be found in role matrix **mcfp**; for example, $\boldsymbol{\mu} = 0.99$ and steepness $\boldsymbol{\sigma}$ mostly varies from 5 to 7. The *timing* of the states is shown in role matrix **ms** in the Appendix: the experienced pain has a high speed factor 0.9 and all other states have speed factor 0.1.

The incorporated adaptive emotion regulation can be explained in the following way. An individual may experience a state of feeling catastrophized (experiencing thoughts like 'I am not a real woman', 'my partner will leave me', etc.), but may be able to consciously think about this by rational reasoning (e.g., 'I am not the only woman who experiences problems', 'my partner loves me', etc.). The higher the activation of feeling catastrophized, normally the higher the activation of the control state will be. Conversely, the higher the activity of the control state, the lower the activity of the feeling catastrophized state will become. So, by controlling the emotions one is experiencing, the emotions may become less intense. The idea is that this process is adaptive in the sense that the strength of the connection from the catastrophized state to the control state can be 'trained' by interventions such as therapies.

Different types of therapies may target one or more states. However, it is unclear which specific therapy targets which specific state(s), and we shall thus continue using the general term 'therapy' that targets some specific state(s), instead of e.g., cognitive behavior therapy that is said to target the catastrophized state. The model is adaptive in the sense that the weights of the incoming connections for the control states supporting

emotion regulation can be adapted by learning. For example, if a healthy individual starts to catastrophize, normally spoken she will learn to control this state. The adaptive **W**-states for these incoming connections are the states portrayed on a higher level in the model (the upper plane) in Fig. 2. Therapy will positively affect one or more of the control states. In turn, the activation values of the catastrophized state and control state together can strengthen (or weaken) the connection from the catastrophizing state to the control state: i.e., Hebbian learning to control the catastrophizing level. This happens by the Hebbian combination function (2) applied to the **W**-states (X_{11} and X_{13}) in the upper plane. The Hebbian learning function takes in the values of the two connected states from the base level and of the connection weight itself, and uses a certain persistence factor as parameter $\boldsymbol{\mu}$: if $\boldsymbol{\mu} = 1$, the connection weight keeps its strength for 100%, and if it is, for example, $\boldsymbol{\mu} = 0.99$, every time unit the connection loses one percent of its strength.

4 Simulation Results

Using the computational model, simulations have been performed for different scenarios. The first scenario includes no therapy and therefore should show that the pain experience increases and finally becomes high. In the second scenario, therapy targeting the control state of the catastrophized state, was included. The third scenario also makes use of a therapy, targeting both the control states catastrophized and distress; thus two states in the cycle. Both therapies strengthen the control state of the state, with the idea that therapy helps people control these states, to break to cycle. The simulations were run until an equilibrium is reached to see what the end state will be.

Scenario 1: No Therapy. In Fig. 3 left it is shown that the injury triggers the experienced pain. This in turns creates a wave of catastrophisation, followed by a pain-related fear. The pain-related fear triggers both vigilance and avoidance behavior. These states in turn increase the lack of arousal and the state of distress. The lack of arousal, combined with dysfunction/distress, feed back into the pain experience, finishing off the loop. There is no way in this loop to break the cycle.

Scenario 2: Therapy Targeting One State. The model has a control state embedded for the state of being catastrophized. The idea is that the individual can consciously think about her emotions and feelings and control these to some extent. The activation of the control state thus negatively affects the state of being catastrophized.

In the model, the strength by which this happens, is typically variable per person and situation, and it can be positively affected by therapy. A therapy was incorporated that targets the strength of the control state, and thus weakens the catastrophized state, hopefully breaking the cycle. Simulation results of therapy targeting one state is presented in Fig. 3 middle. What is seen, however, is indeed a dip in the cycle, but the therapy is not strong enough to actually break the cycle, and eventually the pain and all other states again get higher values. This suggests that it concerns a system problem, where the problem cannot be solved by solving only one particle in the system, but the system as a whole needs to be revised. Therefore, in a following scenario two parts in the cycle are targeted.

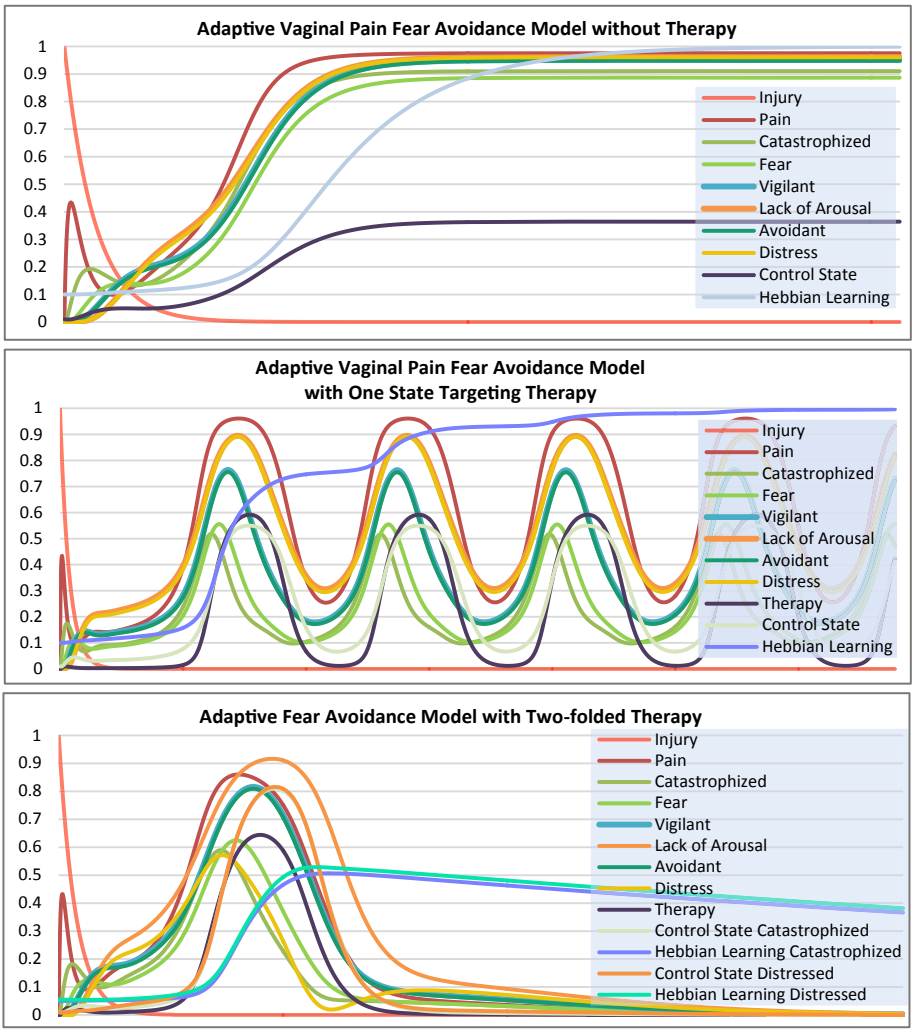

Fig. 3. Results from running the Fear Avoidance model for an individual with GPPPD (a) Left: without a therapeutic intervention. After the injury, we see how a cycle is increasingly elicited by an initial increase in pain experience, resulting in high values for all states in the GPPPD cycle. (b) Middle: with a therapy targeting the control state for the catastrophized state. After the injury, we now also see therapy becoming active, which lowers the cycle states. However, the therapy alone is not enough to break the cycle, and when the experienced pain goes down, and therefore also the therapy, the cycle repeats itself. Even though there is an increase in the connection between the control state and the emotion, the therapy in this form is not strong enough to keep the GPPPD under control. (c) with a therapy targeting both the control state for the catastrophizing state and for the distress state. The therapy is effective: the cycle is stopped and the pain and other parts of the cycle are controlled after the therapy is discontinued.

Scenario 3: Therapy Targeting Two States. Another control state was incorporated for being distressed, and the therapy was targeting both control states in the cycle. The result of the simulation can be seen in Fig. 3 right. The therapy successfully alters the nature of the system, suppressing the activity of the reoccurring pain experience, and thus breaking the cycle of GPPPD.

5 Verification by Mathematical Analysis

The model can be verified per state, by taking a state value at a time point that the system is in equilibrium, considering the incoming connections, and calculating the aggregated impact on the state. The difference between the simulation result and the aggregated impact shows a certain measure of accuracy of the model, as theoretically they are equal in an equilibrium. The states that have been chosen to verify, are X_2, X_{11}, and X_{13}. Time point $t = 498.2$ was used as a reference time point. State X_2 has three incoming connections: X_1, X_6 and X_8.The aggregated impact for this state X_2 is the logistic function, as defined above in (1). The values found for the incoming connections of X_2 are: $X_1 = 1.42 \cdot 10^{-18}$, $X_6 = 0.0049$, $X_8 = 0.0037$. The steepness and threshold of the logistic function are $\boldsymbol{\sigma} = 5$, $\boldsymbol{\tau} = 0.6$. This results in the outcomes shown in Table 2. States X_{11} and X_{13} use the Hebbian learning function (2) for aggregation. Their incoming connections and their values are, respectively: $X_3 = 0.0013$, $X_{10} = 0.29481$, $X_{11} = 0.3673$ and $X_8 = 0.0037$, $X_{12} = 0.71057$, $X_{13} = 0.3822$. Both persistence parameter values are $\boldsymbol{\mu} = 0.99$. The aggregated impacts for these two states were calculated by:

$$\mathbf{aggimpact}_{X_{11}}(t) = X_3X_{10}(1 - X_{11}) + \boldsymbol{\mu}X_{11}$$
$$\mathbf{aggimpact}_{X_{13}}(t) = X_8X_{12}(1 - X_{13}) + \boldsymbol{\mu}X_{13}$$

Table 2. The values for three states in an equilibrium time point have been extracted from simulation data and compared with the aggregated impact of the incoming states. These states have been chosen because they have the highest number of incoming connections (three), and thus the highest probability of deviating strongly from the theoretical equilibrium point. The highest deviation is the 0.04 found in X_2, but still small enough not to suggest an error in the model.

State X_i	X_2	X_{11}	X_{13}
Time point t	498.2	498.2	498.2
$X_i(t)$	0.0015	0.3673	0.3822
$\mathbf{aggimpact}_{X_i}(t)$	0.0417	0.364	0.380
deviation	0.0403	−0.00343	−0.00220

The highest deviation that we found was for stat X_2, being 0.0417671, which is not considered to indicate a problem for our model as it is close enough to 0.

6 Validation of the Model

The simulations in Sect. 3 were run without using numerical empirical data. In order to validate the model, data from [20] were used. In this study, data for levels of pain, sexual arousal and distress were acquired with validated questionnaires in women with GPPPD. From this data set, data of four different individuals with GPPPD with different levels of pain, arousal, and distress were used. The model will therefore be validated for four different types of individuals. The numbers are based on the different questionnaires that the women filled out, giving a score regarding several aspects in their sexual life. The numbers were scaled to a [0, 1] range for the optimization program by dividing the score on the questionnaire by the maximum score. The Female Sexual Function Index questionnaire is used to determine sexual pain and sexual arousal, with higher scores being more positive. Both scores were subtracted from 1, since higher scores on pain and lack of arousal indicate higher levels of pain and lower levels of arousal. Simulated Annealing was used as optimization method, which makes use of a cooling schedule to find the best fitting parameter values [16]. The empirical values of the three states for the four individuals can be found in Table 3, including the indication whether the value is low, medium or high.

The four individuals all experience medium to high levels of pain, but varying levels of (lack of) arousal and distress. The values have been added to the optimization program at a time point where the therapy (targeting both catastrophizing and distress) has not been activated yet, but the symptoms of GPPPD are significantly prevalent (t = 50). The model was tuned for all connection weights, except for the connection weight going from pain to therapy. The model was also tuned for all the function parameter values (threshold and steepness), except for parameter values that belonged to the therapy. Values for the remaining RMSE (Root Mean Square Error) found were around 0.04, 0.04, 0.05, 0.25, respectively, for persons 1 to 4.

Table 3. Empirical data of the four individuals are shown in this table. For each individual, three levels have been used in the parameter tuning optimization procedure: experienced pain, lack of arousal, and distress.

State	Explanation	Individual			
Number		1	2	3	4
X_2	**Experienced pain** level	0.667 *high*	0.400 *medium*	0.467 *medium*	1.000 *high*
X_6	**Lack of arousal** level	0.750 *high*	0.050 *low*	0 *low*	1.000 *high*
X_8	**Distress** level	0.125 *low*	0.750 *high*	0.0417 *low*	1 high

7 Conclusion and Discussion

In this paper, a computational model for GPPPD has been introduced. This was done by building a temporal-causal network based on the Network-Oriented Modelling Approach from [29]. In this model, GPPPD is classified as a sexual disorder and characteristics of the Fear Avoidance Model are incorporated by adding the cyclic component. The Fear Avoidance model suggests that GPPPD can also be modelled as a pain disorder. Perceiving GPPPD not only as a sexual disorder, but also as a pain disorder opens the doors for new types of interventions. For example, acceptance and commitment therapy has recently been developed for chronic pain disorders [11]. It could be useful to apply this type of therapy for GPPPD as well.

Characteristics of GPPPD and interventions targeting one or two states have been captured, by creating an adaptive temporal-causal network. Different therapeutic interventions can target different states of the model. For example, catastrophizing is the primary target of CBT [11]. However, when modeling a therapy that only targets catastrophizing, the therapy does not seem to be effective. CBT for GPPPD is described as the reframing and restructuring of basic (irrational) beliefs that interfere with sexual function [9]. It could thus be hypothesized that CBT does not only target catastrophizing, but also distress (e.g., feelings shame and guilt) and maybe even more states. Which states are targeted, could even differ per psychiatrist, individual and/or couple.

It would be interesting to research which states exactly are being targeted with different therapies and whether this indeed does differ between psychiatrist, individual and/or couple. In addition, it would be interesting to collect data of different states during a therapy over time. This model could be easily extended to incorporate control states for other states in the model. In this way, a model can be created to see which states could and should be targeted in different individuals. The obtained results show that for an individual with low distress and high sexual arousal levels, targeting two states lowers the values of the states, but do not get the individual back to baseline. For women with either high distress, low arousal or both therapy that targets two states does not lower the state levels at all. These results thus show that for most individuals, targeting two states is not enough to break the cycle. This supports claims that there are large individual differences between GPPPD patients and that interventions for GPPPD should be multidisciplinary and tailored to individuals specifically.

This study is the first to apply a computational approach to GPPPD. The parameter tuning suggests that therapy targeting two states is not sufficient for most individuals with GPPPD to lower the pain and other complaints. Future research could collect more empirical data of different states before and after therapy. Adding more control states, to more states of the model would also create a more elaborate model that would be able to characterize more kinds of therapies, and thus increasing the effectiveness of the therapies, specialized for different kinds of individuals. Another future extension of the model may incorporate metaplasticity by making the learning speeds and the persistence factors adaptive, for example, following [29], Ch. 4, so that a second-order adaptive network is obtained taking into account the effect that circumstances may have on a person's learning capabilities and that even may block learning.

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