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Adaptive Networks at the Crossroad of Artificial Intelligence and Formal, Biological, Medical, and Social Sciences

17

Jan Treur

“What is the role of causality in the mechanisms suggested here? Because of the constant recursivity of the process, the widespread notion of linear causality (a single cause for a single effect) cannot be applied to these mechanisms. (...) there is no simple, unidirectional sense of causality ...”

Scherer [1, p. 3470]

Summary

In this paper, it has been illustrated how a network-oriented modeling approach based on temporal-causal networks can be used to model adaptive processes in different domains and thus serve as a unifying factor for multiple sciences. The approach uses mathematical relations and functions as declarative building blocks to be used in a standard temporal-causal network format. A dedicated software environment is available, including a combination function library for aggregation of multiple impacts within a temporal-causal network, with more than 35 already predefined basic combination functions. This software environment makes design and simulation for such network models relatively easy. The approach is illustrated by three examples of adaptive network models: a first-order adaptive network model for bonding by homophily; a second-order adaptive network model for plasticity and metaplasticity for emotion regulation dysfunction in disorders; and a fourth-order adaptive network model for evolutionary processes related to pathogens and pregnancy.

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335

Ascending and Descending (Adopted from Lithograph, Escher, 1960, https://en.wikipedia.org/wiki/Ascending_and_Descending#/media/File:Ascending_and_Descending.jpg)

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1 Introduction

Although different scientific disciplines address different domains from a conceptual and methodological perspective, they have a number of important common elements. These common elements form a good basis for more progress by exchange between disciplines and forms of unification than what is achieved when staying within the boundaries of a single discipline. In this introduction, first, a few of these common elements are briefly pointed out. Next, it is discussed how the adaptive network-oriented modeling approach presented in this paper exploits them.

One of the common elements in different disciplines is the use of *causal relations* to describe processes (i.e. describing when one factor affects another factor). Just a few examples are how opinions or emotions (or even viral infections) of persons influence each other (social science, social neuroscience), how certain events may lead to stress within a person (psychology), how stress can lead to many mental and physical disorders (neuroscience, psychology, medicine), and how

pathogens can negatively affect someone's health (biology, medicine). Next to causal relations, often some form of *dynamics* of processes over time is addressed in different disciplines. For example, how will a viral infection spread within the whole population (social Science, medicine), how does stress lead to certain disorders (neuroscience, psychology, medicine), how is the human body fighting against a pathogen after an infection (biology, medicine). Describing such dynamics can make use of networks of causal relations, which often contain internal cycles. In many cases, also a form of *adaptiveness* is considered as a kind of extended form of dynamics. For example, how does a human body adapt to infections (biology, medicine), how do relations between humans adapt due to circumstances (social science), and how does the brain adapt to long-term stress (neuroscience, psychology).

Other common elements in different disciplines concern the use of concepts and techniques from formal sciences. An important one is *mathematical formalization*. Historically, the important role of mathematical formalization within physics has become well-known. It has led to the foundations for important generic concepts and areas within mathematics, such as calculus and differential equations by Descartes [2], Leibniz [3], Newton [4]. Later on, these have been applied in many other disciplines. Accordingly, in more recent times, mathematical formalizations are also playing an important role in other disciplines. This concerns, for example, mathematical descriptions of biochemical processes within a cell addressed in biology and chemistry [5–7], of contagion processes addressed within social science and biology [8–10], and of processes within the brain addressed within neuroscience [11]; these examples all use concepts and techniques from the areas mentioned above of mathematics.

Nowadays, mathematical formalizations often go hand in hand with *computational analysis and simulation* from computational science within the formal science called informatics or computer science. For this, simulated *in silico* versions of processes in reality, can be obtained by executing mathematical descriptions of these processes on a computational device as dynamical systems, often specified by differential equations [12, 13]. The outcomes of such simulated processes for different inputs or parameters can be analyzed using mathematical techniques as well. For example, the processes within a cell are simulated based on a mathematical description of the cell's biochemistry within biology [5–7], the processes within the brain are simulated based on a mathematical description of these brain processes within neuroscience [11], and the spread of a virus is simulated based on a mathematical description of the effects of the characteristics of the contact network within social science or biology [8–10]. Also, more and more concepts and techniques are applied from artificial intelligence (AI), which can be viewed as an area within informatics or computer science.

Within this paper, the focus is on the adaptive dynamic network-oriented modeling approach described in [14, 15]; this approach covers all of the common elements mentioned above. By some examples in different sections, it is illustrated how this approach supports unification and integration for different disciplines. The considered approach is a network-oriented modeling approach based on adaptive

dynamic *causal networks*, a relatively new branch in the longstanding causal modeling area within AI, e.g. [16–18]. Due to this causal perspective, it fits well with all scientific disciplines where causal relations are considered, which are practically all scientific disciplines. Within these causal networks, the temporal *dynamics* of causal effects is addressed, too.

By addressing *adaptiveness* through self-models within the network based on the notion of network reification, the modeling approach also belongs to another longstanding area within AI, metalevel architectures, and metaprogramming [19–25]. The use of self-models provides a modeling technique by which adaptation can be modeled easily and also iteratively. This is of good help when modeling in scientific domains where multiple levels of adaptivity are important. Examples of this addressed here in some detail are:

- adaptive social processes for bonding based on homophily or similarity [26] within social science (Sect. 3).
- plasticity and metaplasticity for emotion (dys)regulation [27–30] within neuroscience and psychology and complex adaptive disorders [30] in medicine (Sect. 4).
- different levels of evolutionary adaptation [31] in biology and medicine (Sect. 5).

The adaptive network-oriented modeling approach has a solid basis in *formal sciences*. Being network-oriented, the approach belongs to the relatively new formal discipline called network science [14]. Moreover, the basic modeling constructs are just declarative relations and functions, which are elementary concepts within mathematics. Based on them, in a canonical manner, difference and differential equations are defined mathematically, which are also declarative (temporal) notions within mathematics. Via them, mathematical analysis of considered network models can be performed to analyses emerging behavior, for example, concerning emerging equilibria and monotonicity.

The mathematical formalizations of adaptive networks, as mentioned, can easily be used to conduct *computational simulation experiments* from a computational science perspective, which is an area within the formal science indicated by informatics or computer science. A dedicated software environment is available to support this [15, Chap. 9]. The outcomes of such simulation experiments for different inputs and parameters can be analyzed in relation to mathematical analysis, thus supporting the verification of implemented models.

Based on the above, the chapter is structured as follows. In Sect. 2, the adaptive network-oriented modeling approach is briefly explained. In Sect. 3, a first example network model is presented for the social science discipline addressing the adaptive process of bonding in relation to similarity (homophily) of persons. Next, in Sect. 4, an example model is presented for the neuroscience, psychology, and medicine disciplines. This model addresses the role of emotion (dys)regulation in handling stress and medical therapy for that. Section 5 presents an example of evolutionary

processes within biology and medicine concerning pregnancy. Finally, Sect. 6 is a discussion.

2 Modeling by Adaptive Networks

Network-oriented modeling uses some form of network structure as basic architecture. In this Section, such a modeling approach is briefly introduced for temporal-causal network models, which can be nonadaptive or adaptive.

2.1 Dynamic Modeling by Temporal-Causal Network Models

For the networks used here, the interpretation of connections is based on causality and dynamics. The nodes in a network represent states (or state variables) varying in time. The connections define causal relationships that show how each state affects other states over time. The type of network used has been called a *temporal-causal network* [32]. They represent by mathematical relations, and function states X and connections $X \rightarrow Y$ between them for (causal) impacts; here, states X have values $X(t)$ that usually change over time t . More precisely, the following notions from the defining network structure characteristics of a temporal-causal network model:

Connectivity of the Network

- *connection weights* $\omega_{X,Y} \in [-1, 1]$ for each connection from a state X to a state Y .

Aggregation of Multiple Impacts on a Given State in the Network

- *basic combination functions* $\mathbf{c}_j(\cdot)$, $j = 1, \dots, m$ for aggregation, selected for the whole network model from an available combination function library. This is done by specifying $\mathbf{mcf} = [k_1, \dots, k_m]$, where k_j refers to the number combination function $\mathbf{c}_j(\cdot)$ has within that library;
- for each state Y , *combination function weights* $\gamma_{j,Y}$ for the basic combination functions $\mathbf{c}_j(\cdot)$, $j = 1, \dots, m$ are used to indicate by a weighted average of the functions $\mathbf{c}_j(\cdot)$, $j = 1, \dots, m$ the aggregation of incoming single causal impacts $\omega_{X_i,Y} X_i(t)$ of the states $X_1, \dots, X_k$ from which Y gets incoming connections;
- for each state Y and combination function $\mathbf{c}_j(\cdot)$, $\pi_{i,j,Y}$ is used to identify *combination function parameters*.

Timing in the Network

- for each state Y , there is a *speed factor* $\eta_Y \geq 0$.

The above defined concepts define in a canonical manner an associated numerical representation of the network model [32, Chap. 2], in difference or differential equation format which can be used for simulation and mathematical analysis:

$$\begin{aligned} Y(t + \Delta t) &= Y(t) + \eta_Y [c_Y(\omega_{X_1,Y} X_1(t), \dots, \omega_{X_k,Y} X_k(t)) - Y(t)] \Delta t \\ dY(t)/dt &= \eta_Y [c_Y(\omega_{X_1,Y} X_1(t), \dots, \omega_{X_k,Y} X_k(t)) - Y(t)] \end{aligned} \quad (1)$$

where $X_1, \dots, X_k$ are the states from which state Y gets incoming base connections (Table 1).

Here the overall combination function $c_Y(\dots)$ for state Y is the weighted average of the basic combination functions $c_j(\dots)$ by the specified weights $\gamma_{j,Y}$ for Y :

$$c_Y(V_1, \dots, V_k) = \frac{\gamma_{1,Y} c_1(V_1, \dots, V_k) + \dots + \gamma_{m,Y} c_m(V_1, \dots, V_k)}{\gamma_{1,Y} + \dots + \gamma_{m,Y}} \quad (2)$$

Such equations are hidden in the dedicated software environment [15, Chap. 9] that has been developed. This software environment is freely downloadable at <https://www.researchgate.net/project/Network-Oriented-Modeling-Software>.

Table 1 Numerical representations of a temporal-causal network in terms of the network characteristics

Concepts	Numerical representation	Explanation
State values over time t	$Y(t)$	At each time point t each state Y in the model has a real number value, usually in $[0, 1]$
Single causal impact	$\text{impact}_{X,Y}(t) = \omega_{X,Y} X(t)$	At t state X with connection to state Y has an impact on Y , using weight $\omega_{X,Y}$
Aggregating multiple impacts	$\text{aggimpact}_Y(t) = c_Y(\text{impact}_{X_1,Y}(t), \dots, \text{impact}_{X_k,Y}(t))$ $c_Y(\omega_{X_1,Y} X_1(t), \dots, \omega_{X_k,Y} X_k(t))$	The aggregated causal impact of multiple states X_i on Y at t , is determined using combination function $c_Y(\dots)$ applied to the single causal impacts $\omega_{X_i,Y} X_i(t)$, where $X_1, \dots, X_k$ are the states from which state Y gets incoming connections
Timing of the causal effect	$Y(t + \Delta t) = Y(t) + \eta_Y [\text{aggimpact}_Y(t) - Y(t)] \Delta t$ $= Y(t) + \eta_Y [c_Y(\omega_{X_1,Y} X_1(t), \dots, \omega_{X_k,Y} X_k(t)) - Y(t)] \Delta t$	The causal impact on Y is exerted over time gradually, using speed factor η_Y

Making the parameters $\pi_{1,j,Y}$, $\pi_{2,j,Y}$ of the combination functions $c_{j,Y}(\cdot)$ explicit, this becomes

$$c_Y(\pi_{1,1,Y}, \pi_{2,1,Y}, \dots, \pi_{1,m,Y}, \pi_{2,m,Y}, V_1, \dots, V_k) = \frac{\gamma_{1,Y} c_1(\pi_{1,1,Y}, \pi_{2,1,Y}, V_1, \dots, V_k) + \dots + \gamma_{m,Y} c_m(\pi_{1,m,Y}, \pi_{2,m,Y}, V_1, \dots, V_k)}{\gamma_{1,Y} + \dots + \gamma_{m,Y}} \quad (3)$$

There are many different approaches possible to address the issue of aggregating multiple impacts by combination functions. Therefore, for this aggregation, a combination function library with a number of basic combination functions (currently more than 40) is available, while also own-defined functions can be added. Examples of basic combination functions from this library can be found in Table 2.

Based on the canonical equation in (1), the following criterion for mathematical analysis can easily be derived.

Table 2 Examples of basic combination functions from the library

Combination function	Notation	Formula	Parameters
Identity	id (V)	V	
Complemental identity	compid (V)	$1 - V$	
Scaled sum	ssum $_{\lambda}(V_1, \dots, V_k)$	$\frac{V_1 + \dots + V_k}{\lambda}$	Scaling factor $\lambda > 0$
Simple logistic	slogistic $_{\sigma, \tau}(V_1, \dots, V_k)$	$\frac{1}{1 + e^{-\sigma(V_1 + \dots + V_k - \tau)}}$	Steepness $\sigma > 0$ Excitability threshold τ
Advanced logistic	alogistic $_{\sigma, \tau}(V_1, \dots, V_k)$	$\left[\frac{1}{1 + e^{-\sigma(V_1 + \dots + V_k - \tau)}} - \frac{1}{1 + e^{\sigma\tau}} \right]$	Steepness $\sigma > 0$ Excitability threshold τ
Scaled maximum	smax $_{\lambda}(V_1, \dots, V_k)$	$\frac{\min(V_1, \dots, V_k)}{\lambda}$	Scaling factor $\lambda > 0$
Scaled minimum	smin $_{\lambda}(V_1, \dots, V_k)$	$\frac{\max(V_1, \dots, V_k)}{\lambda}$	Scaling factor $\lambda > 0$
Euclidean	eucl $_{n, \lambda}(V_1, \dots, V_k)$	$\sqrt[n]{\frac{V_1^n + \dots + V_k^n}{\lambda}}$	Order $n > 0$ Scaling factor $\lambda > 0$
Scaled geometric mean	sgeomean $_{\lambda}(V_1, \dots, V_k)$	$\sqrt[k]{\frac{V_1 \cdot \dots \cdot V_k}{\lambda}}$	Scaling factor $\lambda > 0$

2.2 A Criterion for Stationary Points and Equilibria

State Y has a *stationary point* at time t if $dY(t)/dt = 0$ at t . The network has an *equilibrium* at t if all states have a stationary point at t . From (1). It directly follows that in a temporal-causal network model, state Y is stationary at t if and only if

$$\eta_Y = 0 \quad \text{or} \quad \text{aggimpact}_Y(t) = Y(t) \\ \text{where} \quad \text{aggimpact}_Y(t) = c_Y(\omega_{X_1,Y}X_1(t), \dots, \omega_{X_k,Y}X_k(t)) \quad (4)$$

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

The network model is in equilibrium when for all states, Y of the network the Eq. (4) holds (also called an *equilibrium equation* then).

Note that considering (3), this criterion (4) is expressed purely in terms of the network structure characteristics $\eta_Y, \omega_{X,Y}, \gamma_{i,Y}, \pi_{i,j,Y}$. Therefore, it is a very simple and easily applicable criterion to analyze a network's stationary points and its emerging equilibrium behavior, just based on the network's structure characteristics. This can be used, for example, to verify whether an implemented network model is correct with respect to the network structure. By a variant of this criterion (4), also monotonicity can be analyzed, just by replacing the equality sign in the equation by an inequality sign $<, \leq, \text{ or } >, \geq$, thus obtaining an inequality relation.

2.3 Adaptive Dynamic Modeling by Adaptive Temporal-Causal Network Models

In Sect. 2.1, the characteristics $\omega_{X,Y}, \gamma_{i,Y}, \pi_{i,j,Y}, \eta_Y$ of the network structure were assumed to be static, which works fine for modeling nonadaptive networks. However, to model adaptive processes as often occur in the real world by adaptive networks, some or all of these characteristics also have to change over time. In general, adaptive networks are usually modeled in a hybrid manner by considering two types of models that interact with each other: a *network model* for the dynamics within the base network and an *adaptation model* for the dynamics of the network characteristics of the base network.

The latter dynamic model is usually specified in a format outside the network-oriented modeling perspective as used for the base network itself. It is most often specified in the form of some procedural or algorithmic programming specification closely related to the code used to program the network adaptation process. This non-network dynamic adaptation model interacts intensively with the dynamic model for the internal network dynamics of the base network. The hybrid approach for adaptive networks was also followed earlier in [32, 33]. In that case, for each new adaptation principle, a new piece of software had to be added to the modeling environment. These limitations of the hybrid approach to adaptive networks have led to the motivation to develop the alternative approach described below.

Recently it has been found out how adaptive networks can be modeled in a more transparent network-oriented modeling manner by one overall network extending the base network by a *self-model* based on so-called *reification states* or *self-model states* [14, 15, 34]. This step has been called *network reification*, and the obtained network a *self-modeling network* or *reified network*. It has been shown in [15] how network reification can be used to explicitly represent many kinds of well-known (e.g. from cognitive neuroscience and social science) adaptation principles for networks in a declarative, transparent, and unified manner. Examples of such adaptation principles include, among others, principles for Hebbian learning (to model plasticity in the brain or mental processes and for bonding based on homophily (to model adaptive social networks). Using self-models, writing procedural or algorithmic specifications, and programming code, as usually applied for network adaptation by the hybrid approach, is not needed anymore. Both the dynamics of the states within the base network and the dynamics of the network structure are specified in a declarative manner by mathematical relations and functions and executed, not by two different interacting software components as in the hybrid case, but by the same generic computational reified network engine as described in more detail in [15, Chaps. 9 and 10].

More specifically, network reification for a temporal-causal network means that for each state Y of the base network, for the adaptive ones among the network structure characteristics $\omega_{X,Y}$, $\gamma_{i,Y}$, $\pi_{i,j,Y}$, η_Y , additional self-model states $\mathbf{W}_{X,Y}$, $\mathbf{C}_{i,Y}$, $\mathbf{P}_{i,j,Y}$, $\mathbf{H}_Y$ (also called reification states) are introduced (see the blue upper plane in Fig. 1):

(a) Connectivity characteristics reification

- Self-model states $W_{X_i,Y}$ are added, representing adaptive connection weights $\omega_{X_i,Y}$.

Fig. 1 Network reification for temporal-causal networks: downward connections from self-model states to base network states (Adapted from [15, Chap. 3, Fig. 3.3])

(b) Aggregation characteristics reification

- Self-model states $\mathbf{C}_{j,Y}$ are added representing adaptive combination function weights $\gamma_{j,Y}$; and
- Self-model states $\mathbf{P}_{i,j,Y}$ are added representing adaptive combination function parameters $\pi_{i,j,Y}$.

(c) Timing characteristics reification

- Self-model states $\mathbf{H}_Y$ are added representing adaptive speed factors η_Y
The notations $\mathbf{W}_{X,Y}$, $\mathbf{C}_{i,Y}$, $\mathbf{P}_{i,j,Y}$, $\mathbf{H}_Y$ for the self-model states indicate the specific *roles* they play with respect to base state Y . These roles are the basis for the specification of a reified network according to *role matrices* **mcw**, **mcfw**, **mcfp**, **ms**, as explained in detail in Sect. 4.2:
- Connection weight role **W** role matrix **mcw**
The self-model states $\mathbf{W}_{X_i,Y}$ for states X_i from which Y gets an incoming connection play the role of providing the adaptive values for the *connection weights* for Y .
- Combination function weight role **C** role matrix **mcfw**
The self-model states $\mathbf{C}_{j,Y}$ play the role of providing the adaptive values for the *combination function weights* for the chosen combination functions for Y .
- Combination function parameter role **P** role matrix **mcfp**
The self-model states $\mathbf{P}_{i,j,Y}$ play the role of providing the adaptive values for the *combination function parameters* for the chosen combination functions for Y .
- Speed factor role **H** role matrix **ms**
The self-model states $\mathbf{H}_Y$ play the role of providing the adaptive value for the *speed factor* for Y .

For a nonadaptive network characteristic from $\omega_{X,Y}$, $\gamma_{j,Y}(\cdot)$, $\pi_{i,j,Y}$, η_Y , the role is played by the network characteristic itself.

The first step is the inclusion of self-model states for some of the characteristics of the base network structure to obtain an extended network. As a next step, the dynamics of the self-model states themselves and their impact on base state Y are described by additional network structure of the extended network. The outgoing impact of the self-model states $\mathbf{W}_{X,Y}$, $\mathbf{C}_{j,Y}$, $\mathbf{P}_{i,j,Y}$, $\mathbf{H}_Y$ on the concerning base state Y is indicated in Fig. 1 by the pink downward arrows. Their effect on Y is that within the above Eq. (1), in computations actually some of the static characteristics $\omega_{X,Y}$, $\gamma_{j,Y}$, $\pi_{i,j,Y}$, η_Y are replaced by the corresponding adaptive characteristics $\mathbf{W}_{X,Y}(t)$, $\mathbf{C}_{j,Y}(t)$, $\mathbf{P}_{i,j,Y}(t)$, $\mathbf{H}_Y(t)$ at the considered time point t . For shortness leaving out the t , as an adaptive variant of (1) and (3) this becomes:

$$dY/dt = H_Y \left[\frac{C_{1,Y} c_1 (P_{1,1,Y}, P_{2,1,Y}, W_{X_1,Y} X_1, \dots, W_{X_k,Y} X_k) + \dots + C_{m,Y} c_m (P_{1,m,Y}, P_{2,m,Y}, W_{X_1,Y} X_1, \dots, W_{X_k,Y} X_k)}{C_{1,Y} + \dots + C_{m,Y}} - Y \right] \quad (5)$$

However, note that this (5) is by itself not in standard temporal-causal network format, as that format requires constant speed factors η_Y , combination function weights $\gamma_{i,Y}$, combination function parameter values $\pi_{i,j,Y}$, and connection weights $\omega_{X,Y}$, and apparently in (4), the H_Y , $C_{i,Y}$, $P_{i,j,Y}$, $W_{X,Y}$ that replace them are not constant. Nevertheless, with a little more effort, the additional network structure within the reified network can still be (re)written in standard temporal-causal network format (with static characteristics η_Y^* , $\gamma_{j,Y}^*$, $\pi_{i,j,Y}^*$, and $\omega_{X,Y}^*$) [15, Chaps. 9 and 10].

As in this way, the resulting reified network is still a temporal-causal network. It can be handled as any temporal-causal network. In particular, mathematical analysis and verification can be applied to it as described for nonadaptive networks [35] and [32, Chap. 12]. It can also be reified itself and thus provide second- and higher-order reified networks to cover networks that are second- or higher-order adaptive. Moreover, the universal combination function and universal difference equation derived in [15, Chap. 10] applies to all states at all levels, enabling a quite compact and uniform computational reified network engine, as described in [15, Chap. 9].

So, by this construction, a reified network structure is obtained that explicitly represents the adaptive characteristics of the base network structure as self-model by some of its states. By its own network characteristics, a reified network represents how the base network adapts over time. This is how certain adaptation principles are specified that describes how exactly the specific base network structure characteristics change due to the adaptation process. To this end, like any other state, the dynamics of reification states themselves are specified by the three general types of network structure characteristics *connectivity* (a), *aggregation* (b), and *timing* (c), also mentioned above:

(a) Connectivity for the self-model states in the reified network

For the self-model states, their *connectivity* in terms of their incoming and outgoing connections has two different functions:

- **Effectuating its special effect from its specific role**

The *outgoing downward causal connections* (the pink downward arrows in Fig. 1) from the self-model states $W_{X,Y}$, $C_{i,Y}$, $P_{i,j,Y}$, H_Y to state Y represent the specific causal impact (its *special effect* from its specific *role*) each of these self-model states has on Y . These downward causal impacts are standard per

role, and make that the adaptive values $\mathbf{W}_{X,Y}(t)$, $\mathbf{C}_{i,Y}(t)$, $\mathbf{P}_{i,j,Y}(t)$, $\mathbf{H}_Y(t)$ are used for the adaptive characteristics of the base network as in (5).

- **Indicating the input for the adaptation principle specified in (b)**

The *incoming upward or leveled causal connections* (blue and black arrows) to a self-model state are used to specify the *input needed for the particular adaptation principle* that is addressed.

(b) **Aggregation of multiple impacts for the self-model states in the reified network**

For the self-model states, their aggregation characteristics have one main aim:

- **Expressing the adaptation principle by a (declarative) mathematical function**

For the *aggregation* of the incoming causal impacts for a self-model state, provided as indicated in (a), a specific combination function is chosen *to express the adaptation principle* in a declarative mathematical manner. For example, the mathematical function definition (7) in Sect. 4.1 used for the self-model states $\mathbf{W}_{X_i,Y}$ expresses a Hebbian Learning adaptation principle by the function $\mathbf{hebb}_{\mu}(\cdot)$ in a declarative manner.

(c) **Timing for the self-model states in the reified network**

For the self-model states, their timing characteristics have one main aim:

- **Expressing the adaptation timing in a declarative manner by a number**

Finally, like any other state, self-model states have their own *timing* in terms of speed factors. Here these speed factors are used as the *means to express the adaptation speed*.

3 A First-Order Adaptive Social Network for Bonding by Homophily

Within social science, adaptive network models are often used to study the emerging behavior of the coevolution of social contagion [36, 37] and bonding by homophily [26, 38]. In this Section, this process is analyzed both by simulation and by mathematical analysis. Using the approach described in Sect. 2, social contagion is modeled by the base level and bonding by homophily by the first reification level, using a state $\mathbf{W}_{X,Y}$ for the strength of any connection from base state X to base state Y . In particular, it is analyzed how emerging communities based on the coevolution of social contagion and bonding by homophily can be related to characteristics of the reified adaptive network's structure characteristics. The homophily adaptation

principle, “*birds of a feather flock together*” [26], expresses how being “birds of a feather” (or being alike or being similar) strengthens the connection between two persons [26, 38]. Social contagion suggests that the stronger the tie between two persons, the more they will become alike [36, 37]. So, this provides an interesting causal cycle, often indicated by the term coevolution.

Below, the first relevant characteristics of the reified network and the incorporated homophily adaptation principle are identified, such as a *tipping point* τ for homophily. This is the extent τ of similarity between the two persons where being attracted (extent of dissimilarity lower than τ) turns into being repelled (extent of dissimilarity higher than τ), and conversely. This tipping point plays a determining role in the emergence of communities. Specific characteristics of the reified network structure make all connection weights to converge to 0 (for states in different emerging communities) or 1 (for states within one emerging community). The reference [15, Chap. 15] describes the proofs of the results.

As adaptation principles are specified by the network characteristics of their reification, particularly the combination functions of the reification states for the adaptive connection weights, properties of them are described as properties of such homophily combination functions. In this Section, they are often called homophily functions.

3.1 Design of a First-Order Adaptive Social Network Model for Bonding by Homophily

The coevolution of social contagion and bonding by homophily is illustrated by a first-order adaptive social network model with connectivity, as shown in Fig. 2. This example of social network has ten base states X_i depicted in the pink plane for the base level in and Fig. 2 90 connection weight self-model states W_{X_i, Y_i} for all i, j with $i \neq j$. Only two examples are depicted in the blue plane for the first reification level in Fig. 2.

The following combination functions were used:

- For social contagion between the base states X_i , the normalized scaled sum functions $\mathbf{ssum}_\lambda(\cdot)$ from Table 2 is used for the self-model states for the connection weights, the simple linear homophily function $\mathbf{slhomo}_{\alpha, \tau}(V_1, V_2, W)$ with tipping point $\tau = 0.1$ and modulation factor α is expressed as $\mathbf{slhomo}_{\alpha, \tau}(V_1, V_2, W) = W + \alpha (\tau - |V_1 - V_2|)(1 - W)W$; and
- The speed factors for all base states Y are $\eta_Y = 0.1$ and for all self-model states for the connection weights, the (adaptation) speed factor is $\eta_{W_{X,Y}} = 0.4$.

Fig. 2 The example adaptive social network model: base level and one example self-model state W_{X_1, X_4} for the weight w_{X_1, X_4} of the connection from X_1 to X_4

3.2 Simulation for the First-Order Adaptive Network Model for Bonding by Homophily

In Fig. 3, examples of simulations are shown for modulation factors α varying from 5 to 15. These examples all concern an initially fully connected social network with initial values $W_{X,Y}(0)$ for the self-model states for the connection weights, as shown in Table 3. As can be seen, no initial values W of weights for the connection between distinct states are 0. The initial values for base states X_1 to X_{10} were 0.1, 0.3, 0.9, 0.8, 0.5, 0.6, 0.85, 0.05, 0.25, and 0.4, respectively.

The graphs show how clusters emerge and, more specifically, how their emergence depends on one of the aggregation characteristics of the adaptive network: the chosen homophily parameter α . The graphs at the left-hand side of Fig. 3 show how the base states in the simulations end up in an equilibrium state (shown for $t = 100$ and $\Delta t = 0.05$), i.e. all base state values become constant. On the right-hand side of the Fig. 3 are matrices with the final connection weights (shown for $t = 500$ and $\Delta t = 0.25$) with an accuracy of 3 digits (0 means < 0.001 , 1 means > 0.999). All show a pattern of emerging clustering. To explore different clustering, the homophily modulation factor α varied from 5 to 15. The emerging clusters are listed in Table 4.

3.3 Mathematical Analysis of the Emerging Equilibrium Behavior for Bonding by Homophily

From a wider perspective, an analysis is presented of the behavior of an adaptive network emerging as a result of social contagion and its coevolution with bonding

Fig. 3 Simulations for the example network for bonding by homophily with modulation factor $\alpha = 5, 11, 11.4$, and 15 . Left-hand side: the graphs of the state values and their clustering up to $t = 100$. Right-hand side: the final connection weights at $t = 500$, showing the same clustering

by homophily. Below are plausible assumptions for a homophily function $c(V_1, V_2, W)$ for the self-model states $\mathbf{W}_{X,Y}$ for $\omega_{X,Y}$. Note that in the current Section, all connection weights are assumed to be nonnegative, which means $\mathbf{W}_{X,Y}(t) \geq 0$ for all t .

Table 3 Connection matrix for the initial connection weights in the example

From to	X ₁	X ₂	X ₃	X ₄	X ₅	X ₆	X ₇	X ₈	X ₉	X ₁₀
X ₁		0.1	0.2	0.1	0.2	0.15	0.1	0.25	0.25	0.1
X ₂	0.25		0.25	0.2	0.1	0.2	0.15	0.25	0.25	0.25
X ₃	0.1	0.25		0.1	0.2	0.15	0.1	0.25	0.1	0.15
X ₄	0.25	0.15	0.25		0.15	0.8	0.25	0.15	0.25	0.25
X ₅	0.25	0.2	0.1	0.2		0.25	0.2	0.1	0.2	0.15
X ₆	0.25	0.1	0.25	0.25	0.25		0.1	0.25	0.25	0.1
X ₇	0.2	0.1	0.2	0.15	0.2	0.2		0.2	0.15	0.25
X ₈	0.1	0.25	0.1	0.25	0.05	0.15	0.25		0.1	0.25
X ₉	0.25	0.15	0.25	0.15	0.2	0.1	0.2	0.15		0.15
X ₁₀	0.2	0.25	0.2	0.2	0.1	0.2	0.15	0.8	0.2	

Definition 1 (*Tipping point and symmetric*)

- (a) The function $c(V_1, V_2, W): [0, 1] \times [0, 1] \times [0, 1] \rightarrow [0, 1]$ has *tipping point* τ for V_1 and V_2 if for all W with $0 < W < 1$ and all V_1, V_2 it holds

- (i) $c(V_1, V_2, W) > W$ if $|V_1 - V_2| < \tau$
- (ii) $c(V_1, V_2, W) = W$ if $|V_1 - V_2| = \tau$
- (iii) $c(V_1, V_2, W) < W$ if $|V_1 - V_2| > \tau$

- (b) The function $c(V_1, V_2, W)$ has a *strict tipping point* τ if it has tipping point τ and in addition it holds:

- (i) If $|V_1 - V_2| < \tau$ then $c(V_1, V_2, 0) > 0$
- (ii) If $|V_1 - V_2| > \tau$ then $c(V_1, V_2, 1) < 1$

- (c) A function $c(V_1, V_2, W)$ is called *symmetric* in V_1 and V_2 if $c(V_1, V_2, W) = c(V_2, V_1, W)$.

It is quite simple to obtain functions with a tipping point τ or with a strict tipping point τ based on the following proposition.

Proposition 1 Here, D is a variable used to represent $|V_1 - V_2|$. Suppose, for any function $d(\tau, D)$ it holds for all $D \geq 0$:

$$\begin{aligned} d(\tau, D) &> 0 \text{ if } D < \tau \\ d(\tau, D) &< 0 \text{ if } D > \tau \end{aligned}$$

Then the following hold:

(a) For any $\alpha > 0$ the function

$$c(V_1, V_2, W) = W + \alpha W(1 - W)d(\tau, |V_1 - V_2|)$$

is symmetric in V_1 and V_2 and satisfies the tipping point condition, but not strict.

(b) For any $\alpha > 0$ the function

$$\begin{aligned} c(V_1, V_2, W) = & W + \alpha \text{Pos}(d(\tau, |V_1 - V_2|))(1 - W) \\ & - \alpha \text{Pos}(-d(\tau, |V_1 - V_2|))W \end{aligned}$$

with $\text{Pos}(x) = (|x|+x)/2$ is symmetric in V_1 and V_2 and satisfies the strict tipping point condition.

Proposition 1 can easily be applied to linear or quadratic functions $d(\tau, D) = \tau - D$ or $d(\tau, D) = \tau^2 - D^2$ or to similar functions $d(\tau, D) = \tau^n - D^n$ of any degree n in D , thus obtaining homophily functions (with non-strict tipping point or strict tipping point) of any order (Table 5).

Table 5 Examples of basic homophily combination functions from the library

Combination function	Notation	Formula	Parameters
Simple linear homophily function	$\mathbf{slhomo}_{\alpha, \tau}(V_1, V_2, W)$	$W + \alpha (\tau - V_1 - V_2)(1 - W)W$	Modulation factor α Tipping point τ
Simple quadratic homophily function	$\mathbf{sqhomo}_{\tau, \alpha}(V_1, V_2, W)$	$W + \alpha W(1 - W)(\tau^2 - D^2)$	
Advanced linear homophily function	$\mathbf{alhomo}_{\tau, \alpha}(V_1, V_2, W)$	$W + \text{Pos}(\alpha (\tau - V_1 - V_2))(1 - W) - \text{Pos}(-\alpha (\tau - V_1 - V_2))W$	
Advanced quadratic homophily function	$\mathbf{aqhomo}_{\tau, \alpha}(V_1, V_2, W)$	$W + \text{Pos}(\alpha (\tau^2 - (V_1 - V_2)^2))(1 - W) - \text{Pos}(-\alpha (\tau^2 - (V_1 - V_2)^2))W$	Modulation factor α Tipping point τ
Simple n th order homophily function	$\mathbf{shomo}_{\tau, \alpha}^n(V_1, V_2, W)$	$W + \alpha W(1 - W)(\tau^n - D^n)$	
Advanced n th order homophily function	$\mathbf{ahomo}_{\tau, \alpha}^n(V_1, V_2, W)$	$W + \text{Pos}(\alpha (\tau^n - (V_1 - V_2)^n))(1 - W) - \text{Pos}(-\alpha (\tau^n - (V_1 - V_2)^n))W$	Order n

Next, it is found out how the properties of the network (and adaptation) defined above (such as a tipping point τ), actually affect the emerging equilibrium behavior. Theorem 1 below presents some of the basic results found for the relation between the emerging equilibrium values for base states and those for the self-model states for the related connection weights. This is obtained by applying criterion (4) from Sect. 2 to an equilibrium state and, in particular, to the self-model state for bonding by homophily that has a homophily function $c(V_1, V_2, W)$ as a combination function. For such an equilibrium state, criterion (4) is equivalent to

$$c(V_1, V_2, W) = W \quad (6)$$

Note that if $c(\cdot)$ has tipping point τ , then by definition 1a) it easily follows that (6) is equivalent to

$$|V_1 - V_2| = \tau \quad \text{or} \quad W = 0 \quad \text{or} \quad W = 1$$

If, moreover, a strict tipping point τ is assumed for $c(\cdot)$, then from Theorem 1b) by contraposition it easily follows that (6) is equivalent to

$$|V_1 - V_2| = \tau \quad \text{or} \quad W = 0 \text{ and } |V_1 - V_2| \geq \tau \quad \text{or} \quad W = 1 \text{ and } |V_1 - V_2| \leq \tau$$

This indicates how the distance of the equilibrium values of the two states relates to the equilibrium value of their connections. The following theorem is mainly based on the above analysis [15, Chap. 13].

Theorem 1 Suppose the function $c(V_1, V_2, W)$ has tipping point τ for V_1 and V_2 . Then, in any emerging attracting equilibrium, the following hold:

- All base states X, Y with values V_1, V_2 at distance $\neq \tau$ have connection weight $W = 0$ or $W = 1$;
- All base states X, Y with values V_1, V_2 at distance $< \tau$ have connection weight $W = 1$, if initial connection weight $W > 0$ or the function $c(V_1, V_2, W)$ has strict tipping point τ for V_1 and V_2 ;
- All base states X, Y with values V_1, V_2 at a distance $> \tau$ have connection weight $W = 0$, if the initial connection weight $W < 1$ or the function $c(V_1, V_2, W)$ has a strict tipping point τ for V_1 and V_2 ; and
- For all base states X, Y with connection weight $0 < W < 1$, their values V_1, V_2 differ exactly τ . ■

Note that no specific assumption is made on the aggregation characteristics for the social contagion of the base states in terms of properties of the combination functions for it. However, when also some assumptions are made for social contagion of the base states, more refined results can be found. The following notions are needed for this.

Definition 2 (*Connectivity and aggregation properties*) Let $c(V_1, \dots, V_k)$ be a function

- $c(\cdot)$ is *strictly monotonically increasing* if $U_i \leq V_i$ for all i , and $U_j < V_j$ for at least one $j \Rightarrow c(U_1, \dots, U_k) < c(V_1, \dots, V_k)$;
- $c(\cdot)$ is *scalar-free* if $c(\alpha V_1, \dots, \alpha V_k) = \alpha c(V_1, \dots, V_k)$ for all $\alpha > 0$;
- $c_Y(\cdot)$ is *normalized* in the network if for each state Y it holds $c_Y(\omega_{X_1,Y}, \dots, \omega_{X_k,Y}) = 1$, where; and
- $X_1, \dots, X_k$ are the states from which Y gets incoming connections.

A network is called *weakly symmetric* if for all states X, Y , it holds $\omega_{X,Y} > 0$ if $\omega_{Y,X} > 0$. Based on this, the following is obtained.

Theorem 2 Suppose the network is weakly symmetric, and the combination functions for the social contagion for the base states are strictly monotonically increasing, normalized, and scalar-free. Moreover, suppose $c(V_1, V_2, W)$ has a tipping point τ and is symmetric in V_1 and V_2 . Then the following hold for any emerging attracting equilibrium state:

- (a) All base states X, Y with distinct values $V_1 \neq V_2$ have connection weight $W = 0$
- (b) All base states X, Y with equal values $V_1 = V_2$ have connection weight $W = 1$ if the initial connection weight $W > 0$ or the function $c(V_1, V_2, W)$ has a strict tipping point τ for V_1 and V_2 . ■

Theorem 3 (Emerging clusters) This theorem is applicable to emerging communities or clusters and distances between different equilibrium values for the base states. Suppose the network is weakly symmetric, the combination function for the states is strictly monotonically increasing, scalar-free, and normalized, and the combination function $c(V_1, V_2, W)$ for the connections uses a strict tipping point τ and is symmetric in V_1 and V_2 . Then for any emerging attracting equilibrium state, the following hold:

- (a) A partition of the set of base states into disjoint subsets (clusters) $C_1, \dots, C_p$ occurs such that:
 - For each cluster C_i the values for all the base states in C_i are equal: $V_1 = V_2$ for all $X, Y \in C_i$;
 - Every cluster C_i forms a fully connected network with weights 1: connection weight $W = 1$ for all $X, Y \in C_i$;
 - Every two base states in two different clusters C_i have connection weight 0: $i \neq j, X \in C_i$ and $Y \in C_j$ imply $W = 0$; and
 - Any two distinct values $V_1 \neq V_2$ of base states X, Y have distance $\geq \tau$.

- Therefore, there are at most $p \leq 1 + 1/\tau$ clusters C_i and base state values V .
- (b) The condition that $c(V_1, V_2, W)$ has a strict tipping point τ for a) can be weakened to having a non-strict tipping point τ if no initial values $W = 0$ are used for connection weights. ■

These theorems are illustrated by the example of adaptive social networks and the simulations shown in Fig. 3. It turns out that all connection weights converge to 0 or 1. This is consistent with that what Theorems 2 and 3 predicted. Moreover, it can be seen that clusters with distance less than 0.1 do not occur, in accordance with Theorem 3. For example, in the step from $\alpha = 5$ to $\alpha = 11$, one of the clusters splits in two, where the two emerging clusters have distance >0.1 . Between $\alpha = 11$ and $\alpha = 11.4$, there is another (discontinuous) jump like that. For $\alpha = 11$, after some time of ‘hesitation’ between time point 20 and time point 40, states X_5 and X_6 finally ‘decide’ to join X_3 , X_4 , and X_7 to form one cluster of five states still. However, increasing aggregation characteristic α very slightly, for $\alpha = 11.4$, they suddenly stay separate, not just at a small distance from the cluster of X_3 , X_4 and X_7 , but immediately with a distance more than $\tau = 0.1$: this new cluster $\{X_5, X_6\}$ converges to state value 0.683 whereas the other cluster $\{X_3, X_4, X_7\}$ converges to state value 0.796, which is a difference of 0.113, just above the tipping point $\tau = 0.1$. This is exactly as Theorem 3 predicts. This would correspond with a difference of 0.113, just above the tipping point $\tau = 0.1$, as exactly predicted by Theorem 3. Note that for higher values of α , no more than these four clusters occur, and for $\alpha = 1$, the ten states together form just one cluster.

It is discussed in [15, Chap. 6] how a second-order adaptive network model can be obtained by making the tipping point parameter τ of the homophily function adaptive so that it becomes dependent on the number or strength of the already available connections.

4 A Second-Order Adaptive Network for Stress Regulation Dysfunction

In this Section, an example of a second-order adaptive network model is described for a medical context. In such a second-order adaptive network, the base network has its own internal dynamics, but it also uses first-order adaptation principles. Moreover, these first-order adaptation principles themselves change based on second-order adaptation principles.

4.1 Design of the Adaptive Network Model Showing Plasticity and Metaplasticity

Neural or mental networks equipped with a Hebbian learning mechanism [39–41] can adapt connection weights over time. From a neuroscientific perspective, this is usually considered an important form of *plasticity*. The principle is in a simplified form, sometimes formulated as “what fires together, wires together” [42, 43].

In some circumstances, an organism should learn (and change) fast, but in other circumstances, it is better to stay stable and persist what has been learned in the past, referred to as the *Plasticity Versus Stability Conundrum* [44, p. 773]. To control this, the brain uses a type of (second-order) adaptation called *metaplasticity*. It has become an important focus of study within cognitive neuroscience. Various studies such as [30, 44–46] show how the adaptation of synapses (as described, for example, by Hebbian learning) is modulated by accelerating the adaptation process or decelerating or even blocking it. Among the reported factors affecting synaptic plasticity in such a way are stimulus exposure, activation, previous experiences, and stress. In [30], the focus is on the role of stress in reducing or blocking plasticity. Many mental and physical disorders are stress-related and are hard to overcome due to poor or even blocked plasticity that comes with stress. Garcia [30] describes the negative role of stress-related metaplasticity for this, which often leads to a patient being locked in a disorder by that negative pattern. However, he also shows that by some form of therapy, this negative cycle might be broken:

At the cellular level, evidence has emerged indicating neuronal atrophy and cell loss in response to stress and in depression. At the molecular level, it has been suggested that these cellular deficiencies, mostly detected in the hippocampus, result from a decrease in the expression of brain-derived neurotrophic factor (BDNF) associated with elevation of glucocorticoids [30, p. 629].

...modifications in the threshold for synaptic plasticity that enhances cognitive function is referred here to as ‘positive’ metaplasticity. In contrast, changes in the threshold for synaptic plasticity that yield impairment of cognitive functions, for example (..) in response to stress (..), is referred to as ‘negative’ metaplasticity [30, pp. 630–631].

In summary, depressive-like behavior in animals and human depression are associated with high plasma levels of glucocorticoids that produce ‘negative’ metaplasticity in limbic structures (...). This stress-related metaplasticity impairs performance on certain hippocampal-dependent tasks. Antidepressant treatments act by increasing expression of BDNF in the hippocampus. This antidepressant effect can trigger, in turn, the suppression of stress-related metaplasticity in hippocampal-hypothalamic pathways thus restoring physiological levels of glucocorticoids [30, p. 634].

Based on this literature, a second-order adaptive reified network model for plasticity and metaplasticity has been designed with connectivity, as shown in Fig. 4. Table 6 displays the explanations of the states, too. Here, blocked plasticity for emotion regulation is modeled, which leads to emotion dysregulation and its often-found consequences in the form of a mental or physical disorder [27–30].

In the base network, s is a stressful stimulus leading to stressful, emotional response ps_b and stress feeling fs_b . State cs_b performs stress regulation by its

Fig. 4 Connectivity of the second-order adaptive network model for plasticity and metaplasticity of emotion regulation with base level (lower plane, pink), first reification level (middle plane, blue) and second reification level (upper plane, purple), and upward connections (blue) and downward connections (red) defining interlevel relations

negative outgoing connection, as soon as it is activated. However, to get cs_b activated, the connections from fs_b and ps_b to cs_b play an important role, and such connections have to be learned and maintained, which may not happen if that plasticity is out of order. Many disorders, both physical and mental, originate from such poor functioning stress regulation. In the example, for the sake of simplicity, we focus on the connection from fs_b to cs_b . The learning process for this connection is modeled by a Hebbian learning principle represented by first-order self-model state $\mathbf{W}_{fs_b, cs_b}$. This state models a Hebbian learning adaptation principle defined as a combination function by

$$\text{hebb}_{\mu}(V_1, V_2, W) = V_1 V_2 (1 - W) + \mu W \quad (7)$$

where μ is the persistence parameter, variable W is used for the weight of the considered connection and variables V_1, V_2 for the activation values of the two connected states.

However, whether or not learning takes place depends on metaplasticity, modeled here by second-order self-model states $\mathbf{H}_{\mathbf{W}_{fs_b, cs_b}}$ and $\mathbf{M}_{\mathbf{W}_{fs_b, cs_b}}$ for learning speed and persistence, respectively. Note that the $\mathbf{M}$ -state is an aggregation self-model state for a combination function parameter: persistence parameter μ for the combination function $\text{hebb}_{\mu}(\cdot)$ for first-order adaptation state, and $\mathbf{W}_{fs_b, cs_b}$ for Hebbian learning of the connection from fs_b to cs_b . In principle, the learning will start to work or accelerate when the external stimulus s is sensed through sensor state ss_s . As discussed above, whether or not and to which extent learning is controlled by a

form of metaplasticity, for example, affected by stimulus exposure. As noted in [46, p. 2], “*adaptation accelerates with stimulus exposure.*”

However, as shown in the above quotations by Garcia [30], stress creates a negative metaplasticity effect, which corresponds to low values for these second-order self-model states. For example, a value close to 0 for learning speed representation $H_{W_{fs_b, cs_b}}$ practically blocks the learning, and value around 0.5 for persistence representation $M_{W_{fs_b, cs_b}}$ makes that every time unit, around 50% of the learned effect is lost, which is a dramatic effect if no additional learning takes place.

For most of the states in the designed network model, for the aggregation characteristics **alogistic** _{σ, τ} ($\cdot$) is used as a combination function (Table 2). The only exceptions are W_{fs_b, cs_b} , which uses **hebb** _{μ} ($\cdot$) defined by (7), and therapy state ts_b , which is an external input modeled by **stepmod** _{ρ, δ} ($\cdot$), which defines an (independent) activation after *duration* δ and *repetition* of the cycle after a time period ρ .

In Figs. 5 and 6, all network characteristics for the designed adaptive network model are specified in the form of role matrices. Role matrix mb in Fig. 1 specifies the *base connectivity* characteristics. On each row, for the given state, it indicates from which states at the same or a lower level it gets an incoming connection (the black and blue arrows in Fig. 4). Note that for some of the states, a connection from the state itself occurs. The latter applies to all (first- and second-order) self-model states, as shown in mb.

As an example, in the second row, it is indicated that state X_2 (= srs_s) only has one incoming base connection from state X_1 (= ss_s). As another example, the seventh row indicates that state X_7 (= W_{fs_b, cs_b}) has incoming base connections from X_4 (= fs_b), X_5 (= cs_b), X_7 (= W_{fs_b, cs_b}) itself, and in that order. This order is important as the Hebbian combination function **hebb** _{μ} ($\cdot$) used is not symmetric in its arguments. Note that the more informative state names such as ss_s , and so on, in each of the role matrices depicted in Figs. 1 and 2 are not part of the specification but are just added for human understanding. Similarly, the other types of role matrices were defined (Figs. 1 and 2): role matrices mcw for connection weights; mcfw for

mb base connectivity					mcw connection weights					ms speed factors	
		1	2	3	4			1	2	3	4
X_1	ss_s	X_1				X_1	ss_s	1			
X_2	srs_s	X_1				X_2	srs_s	1			
X_3	ps_b	X_2	X_4			X_3	ps_b	1			
X_4	fs_b	X_3	X_5			X_4	fs_b	1	-1		
X_5	cs_b	X_3	X_4	X_6		X_5	cs_b	0.5	X_7	1	
X_6	ts_b	X_6				X_6	ts_b	1			
X_7	W_{fs_b, cs_b}	X_4	X_5	X_7		X_7	W_{fs_b, cs_b}	1	1	1	
X_8	Hw_{fs_b, cs_b}	X_4	X_5	X_7	X_8	X_8	Hw_{fs_b, cs_b}	-0.5	1	0.1	1
X_9	Mw_{fs_b, cs_b}	X_4	X_5	X_7	X_9	X_9	Mw_{fs_b, cs_b}	-0.15	0.5	0.05	1
X_1	ss_s					X_1	ss_s				0.5
X_2	srs_s					X_2	srs_s				0.5
X_3	ps_b					X_3	ps_b				0.2
X_4	fs_b					X_4	fs_b				0.02
X_5	cs_b					X_5	cs_b				0.2
X_6	ts_b					X_6	ts_b				4
X_7	W_{fs_b, cs_b}					X_7	W_{fs_b, cs_b}				X_8
X_8	Hw_{fs_b, cs_b}					X_8	Hw_{fs_b, cs_b}				0.1
X_9	Mw_{fs_b, cs_b}					X_9	Mw_{fs_b, cs_b}				0.02

Fig. 5 Specification of the *connectivity characteristics* and *timing characteristics* of the second-order adaptive example network model by role matrices mb, mcw, and ms

mcfw				mcfp								
combination		1	2	3	function		1		2		3	
							alogistic		hebb		stepmod	
function weights		alog- istic	hebb	step- mod	parameter		σ	τ	μ		ρ	δ
X_1	ss_s	1			X_1	ss_s	5	0.2				
X_2	srs_s	1			X_2	srs_s	5	0.2				
X_3	ps_b	1			X_3	ps_b	5	0.2				
X_4	fs_b	1			X_4	fs_b	5	0.4				
X_5	cs_b	1			X_5	cs_b	5	0.7				
X_6	ts_b			1	X_6	ts_b					200	100
X_7	W_{fs_b,cs_b}		1		X_7	W_{fs_b,cs_b}			X_9			
X_8	Hw_{fs_b,cs_b}	1			X_8	Hw_{fs_b,cs_b}	5	0.9				
X_9	Mw_{fs_b,cs_b}	1			X_9	Mw_{fs_b,cs_b}	5	0.6				

Fig. 6 Specification of the *aggregation characteristics* of the second-order adaptive example network model by role matrices mcfw and mcwfp (Adapted from [15, Chap. 7, Fig. 7.1])

combination function weights; mcfp for combination function parameters; and ms for speed factor roles.

Within each role matrix, for an adaptive network characteristic, entries in red cells indicate a reference to the name of another state that, as a form of reification, represents that characteristic. In contrast, entries in green cells indicate fixed values for nonadaptive characteristics. In this way, the red cells represent the pink downward connections from the self-model states in pictures, as shown in Fig. 4, with their specific roles **W**, **H**, **C**, **P** indicated by the type of role matrix. The type of role matrix in which connections are represented defines the roles of the self-model states. For example, in mcw, the X_7 in the red cell in the row for X_5 defines that the connection weight for X_5 ($= cs_b$) from X_4 ($= fs_b$) is adaptive with value represented by X_7 ($= W_{fs_b,cs_b}$). Similarly, role matrix ms indicates (in red) that X_8 represents the adaptive speed factor of X_7 , and (in green) that the speed factors of all other states have fixed values.

In Fig. 6, the role matrices **mcfw** and **mcfp** are shown for aggregation characteristics in terms of combination function weights and parameters, respectively. Matrix mcfp is a 3D matrix with the first dimension for the states, second dimension for the (two) combination function parameters, and third dimension for the combination functions. For example, in Fig. 2, the name X_9 in the red cell in role matrix mcfp indicates that the value of the persistence parameter μ for X_7 ($= W_{fs_b,cs_b}$) is adaptive and is represented by the value of state X_9 ($= M_{W_{fs_b,cs_b}}$). In contrast, the 5 in the first green cell of mcfp for X_5 indicates the static value of the steepness of the logistic function for X_5 ($= cs_b$).

For this example network model, the selection of combination functions from the library for the network is specified by **mcf** = [2 3 35], being **alogistic** _{σ,τ} (..),

$\text{hebb}_\mu(\cdot)$, $\text{stepmod}_{\rho,\delta}(\cdot)$ respectively. So, in terms of (2) for this network it holds $c_1(\cdot) = \text{alogistic}_{\sigma,\tau}(\cdot)$, $c_2(\cdot) = \text{hebb}_\mu(\cdot)$, $c_3(\cdot) = \text{stepmod}_{\rho,\delta}(\cdot)$.

4.2 Simulations for the Adaptive Network Model Showing Plasticity and Metaplasticity

Some simulation experiments have been performed using the dedicated software environment for reified network models described in [15, Chap. 9]. In particular, a scenario is shown here in which the focus was on the effect of the stress level of fs_b on plasticity [30]. In Fig. 5, simulation results are shown for the characteristics in Figs. 1 and 2. Here a person is considered who for some time (before time 0) has had a stressful life so that the initial values already reflect a high-stress level. Initial values are shown in Table 7.

The graph in Fig. 7 shows the activation levels of all states, including how the weight of the connection from fs_b to cs_b represented by W_{fs_b,cs_b} is learned, and what speed and persistence values are applied for that, represented by $H_{W_{fs_b,cs_b}}$ and $M_{W_{fs_b,cs_b}}$. As can be seen, in the first phase until time 100, a high-stress level represented by fs_b (the red line) leads to maintaining low values of $H_{W_{fs_b,cs_b}}$ (the brown line starting at 0.05) and $M_{W_{fs_b,cs_b}}$ (the grey line starting at 0.6). Therefore learning is practically blocked in this phase (the blue line for W_{fs_b,cs_b} starting at 0.1 stays low). In the next phase, from time 100 to time 200, therapy is applied, represented by ts_b , that boosts the activation level of cs_b , which in turn reduces the stress level represented by fs_b . This also increases the speed and persistence of the Hebbian learning (second-order states $H_{W_{fs_b,cs_b}}$ and $M_{W_{fs_b,cs_b}}$ increase to 1 and 0.9, respectively), and because of this, now indeed learning takes place. In the last phase, from time 200 to time 300, the therapy has finished, but due to the therapy, the stress regulation mechanism has been unlocked, now it can keep the stress levels low without external help.

4.3 Mathematical Analysis of the Emerging Equilibrium Behavior for the Example Network

Mathematical analysis and verification have been performed based on Sect. 2, criterion (4). After continuing the simulation until $t = 1000$ without any further intervention, the values shown in Table 8 were obtained while the pattern stabilizes.

So, the feeling state fs_b stabilizes at 0.37 and the control state at 0.64. This is in contrast to the phase before therapy indicated by $t = 100$ where the feeling state was

Table 7 Initial values for the simulation

ss_s	srs_s	ps_b	fs_b	cs_b	ts_b	W_{fs_b,cs_b}	$H_{W_{fs_b,cs_b}}$	$M_{W_{fs_b,cs_b}}$
1	0.8	0.7	0.7	0.2	0.1	0.1	0.05	0.6

Fig. 7 Simulations for the second-order adaptive example network model. The first phase (time 0 to 100): a high-stress level while metaplasticity blocks plasticity due to which emotion regulation does not function properly. The second phase (time 100 to 200): a therapy artificially boosts emotion regulation, making metaplasticity unblock the plasticity, making emotion regulation stronger. The third phase (time 200 to 300): due to the unblocked plasticity, the emotion regulation is now able to learn further and keep the stress level low without therapy. (Adapted from [15, Chap. 7, Fig. 7.4])

0.72 and the control state was 0.38. The connection from feeling state to control state now has weight 0.87, which before the therapy was 0.16. All this indicates that a well-functioning emotion regulation mechanism was obtained, unlike before the therapy. Another big difference is that the learning speed was only 0.002 before the therapy, which indicates that the learning was blocked, whereas now it has become 0.95, so now plasticity is there. For verification of the network model, some further elementary mathematical analysis has been performed. Such analysis for the learning process goes as follows;

$$\begin{aligned}
 W &= \text{hebb}_{\mu}(V_1, V_2, W) = V_1 V_2 (1 - W) + \mu W && \Leftrightarrow \\
 W &= V_1 V_2 - V_1 V_2 W + \mu W && \Leftrightarrow \\
 W &= \frac{V_1 V_2}{1 - \mu + V_1 V_2} && (8)
 \end{aligned}$$

In the above situation at $t = 1000$ the simulated values are:

$$\begin{aligned}
 V_1 &= fs_b(1000) = 0.3728337 \\
 V_2 &= cs_b(1000) = 0.6376211 \\
 W &= W_{fs_b, cs_b}(1000) = 0.870346678 \\
 \mu &= M_{W_{fs_b, cs_b}}(1000) = 0.964586502
 \end{aligned}$$

Substituting these values in the right-hand side of (8) provides 0.8703467786 (in 10 digits). Comparing this to the value 0.8703466777 of W at time 1000 in 10 digits

Table 8 Values in the simulation at $\tau = 1000$

State	ss_b	srs_b	ps_b	fs_b	cs_b	ts_b	W_{fs_b,cs_b}	$H_{W_{fs_b,cs_b}}$	$M_{W_{fs_b,cs_b}}$
$\tau = 1000$	0.9717402	0.9717402	0.9955412	0.3728337	0.6376211	0	0.8703467	0.9490621	0.9645865
$\tau = 100$	0.9717402	0.9717402	0.9992254	0.7229209	0.3786141	0	0.1626221	0.0019257	0.5104981

shows a difference of 1.006×10^{-7} . This is reasonably small, and so can be considered as some evidence that the implemented network model is correct in comparison to the adaptive model's specified network characteristics. As another verification, the value of $X_8 (= H_{W_{fs_b, cs_b}})$ has been checked compared to its aggregated impact. The aggregated impact for X_8 at time 1000 is:

$$\begin{aligned} \text{aggimpact}_{X_8}(t) &= \text{alogistic}_{5,0.9}(-0.18642, 0.637621, 0.087035, 0.949062) \\ &= \text{alogistic}_{5,0.9}(1.487301) \\ &= 0.949062128 \end{aligned}$$

The difference of this with the value 0.949062090 of X_8 at $t = 1000$ is 3.7×10^{-8} . As this is also small, this provides another evidence that the implemented network model is correct in comparison to the model's specified network characteristics.

4.4 Further Mathematical Analysis of Hebbian Learning

In this Section, it is discussed more generally how aggregation for the connection weight self-model state can be defined by a specific class of combination functions for Hebbian learning, and it will be analyzed what equilibrium values can emerge for the learned connections. The following plausible assumptions are made for this class of Hebbian learning functions. They were adopted from [15, Chap. 14].

Definition 3 (*Hebbian learning function*) A function $c: [0, 1] \times [0, 1] \times [0, 1] \rightarrow [0, 1]$ is called a *Hebbian learning function* with *persistence parameter* μ if the following hold:

- (a) $c(V_1, V_2, W)$ is a monotonically increasing function of V_1 and V_2
- (b) $c(V_1, V_2, W) - \mu W$ is a monotonically decreasing function of W
- (c) $c(V_1, V_2, W) \geq \mu W$
- (d) $c(V_1, V_2, W) = \mu W$ if and only if one of V_1 and V_2 is 0 (or both), or $W = 1$

A Hebbian learning function is called a *fully persistent* Hebbian learning function if $\mu = 1$.

Definition 4 (*Variable separation*) The Hebbian learning function $c(V_1, V_2, W)$ with persistence parameter μ *enables variable separation* by functions $cs: [0, 1] \times [0, 1] \rightarrow [0, 1]$ monotonically increasing and $cc: [0, 1] \rightarrow [0, 1]$ monotonically decreasing if $c(V_1, V_2, W) = cs(V_1, V_2) cc(W) + \mu W$, where $cs(V_1, V_2) = 0$ if and only if one of V_1, V_2 is 0, and $cc(1) = 0$ and $cc(W) > 0$ when $W < 1$.

The function $cs(V_1, V_2)$ is called the *state factor* and the function $cc(W)$ the *connection factor*.

Theorem 4 Assume in a reified temporal-causal network the Hebbian learning combination function $c(V_1, V_2, W)$ with persistence parameter μ enables variable separation by state factor $cs(V_1, V_2)$ and connection factor $cc(W)$, then:

- (a) For $\mu < 1$ the function h_μ defined for $W \in [0, 1)$ by

$$h_\mu(W) = \frac{(1 - \mu)W}{cc(W)}$$

is strictly monotonically increasing and has an inverse g_μ .

- (b) In any emerging attracting equilibrium state, it holds

- (i) If $\mu < 1$ then $W = g_\mu(cs(V_1, V_2)) < 1$
- (ii) If $\mu = 1$ then $V_1 = 0$ or $V_2 = 0$ or $W = 1$ ■

As an example, the specific class of Hebbian learning functions enabling variable separation with a linear connection function $cc(W) = 1 - W$ is considered. Then

$$h_\mu(W) = \frac{(1 - \mu)W}{cc(W)} = \frac{(1 - \mu)W}{1 - W} \quad (9)$$

and the inverse $g_\mu(W')$ of $h_\mu(W)$ can easily be determined from (9) algebraically:

$$g_\mu(W') = \frac{W'}{W' + (1 - \mu)} \quad (10)$$

Substitute $W' = cs(V_1, V_2)$ in (18) and it is obtained:

$$W = g_\mu(cs(V_1, V_2)) = \frac{cs(V_1, V_2)}{(1 - \mu) + cs(V_1, V_2)} \quad (11)$$

This generalizes what was found in Sect. 4.3, which was based on the case $cs(V_1, V_2) = V_1 V_2$.

5 A Fourth-Order Adaptive Evolutionary Biological Network

The third adaptive network model addressed here is an example of a fourth-order adaptive network model for the evolutionary biological domain, in particular, concerning pregnancy. Viewed from a distance, an evolutionary process is an adaptation process that is changing the physical world by creating new causal pathways that often change or block existing causal pathways, based on circumstances. From a causal modeling perspective, this can be described as (depending

on certain circumstances) changing some of the causal connections in such existing causal pathways from high to 0 or very low, or conversely. As these new causal pathways usually depend on specific circumstances, they make that an organism can operate in a more context-sensitive manner than before: it becomes more fine-tuned to what can happen. Selection pressure makes that for given circumstances, organisms with more favorable causal pathways for these circumstances become more dominant within a population. In this way, this leads to a shift in the average pathways within the population. In [31, 47], it is suggested that the following evolutionary levels of adaptation can be distinguished for pathogens, defense (immune) systems, and disgust during the first trimester of pregnancy: “... *one form of disgust, pathogen disgust, functions in part as a third-order adaptation, as disease-avoidance responses are up-regulated in a manner that compensates for the increases in vulnerability to pathogens that accompany pregnancy and preparation for implantation—changes that are themselves a second-order adaptation addressing the conflict between maternal immune defenses and the parasitic behavior of the half-foreign conceptus ...*” [47].

Fessler et al. [31, 47] sketch three adaptation levels, which occur on top of the ecological adaptation level of the development of pathogens that affect health. It makes a total of four adaptation levels that can be distinguished. At each level, new causal pathways were added that modulate the causal pathway of the previous level by modifying it if certain circumstances occur:

- First-order adaptation.
Pathogens occur, with causal pathways negatively affecting the causal pathways for good health;
- Second-order adaptation.
An internal defense system occurs, with causal pathways which negatively affect the causal pathways used by pathogens as soon as they occur;
- Third-order adaptation.
For pregnancy, causal pathways are added to make during first-trimester pregnancy the defense system's causal pathways less strong as the half-foreign conceptus might easily be identified as a kind of parasite and attacked; and
- Fourth-order adaptation.
Disgust during (first-trimester) pregnancy adds causal pathways by which potential pathogens in the external world are avoided to reduce risks of entering pathogens while the internal defense system is low functioning. This strengthens the overall defense system by strengthening the external defense system (the behavioral immune system) by which the pathogens are addressed outside the body. This makes the causal pathway from (first trimester) pregnancy to suppress the causal pathways of the overall defense system less strong as the external component of the defense system is strengthened by disgust.

5.1 Design of the Fourth-Order Adaptive Network Model for Evolutionary Adaptation

A fourth-order reified adaptive network for these evolutionary processes has been designed. Levels of adaptation are created where the causal pathways at one adaptation level are adapted by the causal pathways at the next level. The general pattern then becomes in a simple form, as shown in the connectivity picture depicted in Fig. 8. For an explanation of the states, see Table 9. Here, the causal pathways addressed are modeled by the (upward) causal relations from base states s_1 to s_4 to certain states in the four non-base planes that play a role as connection weight self-model state for the connection of the previous causal pathway. For example, by the causal pathway from s_1 to W_{s_5, e_1} , the occurrence s_1 of pathogens has a negative effect on the causal pathway leading to good health e_1 . In turn, by the causal pathway from s_2 to $W_{s_1, W_{s_5, e_1}}$, the occurrence s_2 of a defense system has a negative effect on the pathogen's causal pathway from s_1 to W_{s_5, e_1} , thus making that the causal pathway to good health can function better.

Similarly, the other two adaptation levels triggered by s_3 (pregnancy) and s_4 (disgust) modify the previous causal pathways. To get the suppression effect on the self-model states aimed for, the occurrence of a high level for any of the s_i , $i = 1, 2, 3, 4$ has to result in a low level of the targeted self-model state, whereas a low level

Fig. 8 Connectivity of the fourth-order adaptive example network model for evolutionary processes (Adapted from [15, Chap. 8, Fig. 8.2])

of this s_i should result in a high level of that self-model state. This is exactly what the complemental identity function $\text{compid}(\cdot)$ does. Recall that it is defined by (Table 2):

$$\text{compid}(V) = 1 - V \quad (12)$$

Therefore, this combination function is used here for all self-model states X_7 to X_{10} .

Figure 9 shows the role matrices for this adaptive network model. As in Sect. 4, each role matrix has a format in which in each row for the indicated state, it is specified which other states (red cells) or values (green cells) affect it, according to the role of that matrix. In particular, in role matrix mcw , the red cells indicate which states X_i play the role of the self-model states for the weights of the connection indicated in that cell in mb .

5.2 Simulation for the Fourth-Order Adaptive Network for Evolutionary Adaptation

Simulations have been performed using the dedicated software environment for reified network models described in [15, Chap. 9]. The scenario considered here focuses on a time period in which for a population, subsequently, pathogens occur, a defense system against them is developed, pregnancy occurs, and disgust (in the first trimester of pregnancy) occurs. So, in the simulation, the four orders of adaptation show up:

Adaptation 1 Pathogens are introduced	first-order adaptation
Adaptation 2 Defense system is developed	second-order adaptation
Adaptation 3 Pregnancy	third-order adaptation
Adaptation 4 Disgust	fourth-order adaptation

Figure 10 summarizes the simulation results for this scenario. The red line in Fig. 10 indicates the health level. Before adaptation 1, health is good. After adaptation 1, health becomes bad. After adaptation 2, health becomes good again. After adaptation 3, health becomes worse again. Finally, after adaptation 4, health becomes better again.

5.3 Mathematical Equilibrium Analysis for the Example of an Evolutionary Network

Again criterion (6) is used here to analyze the state values in an equilibrium. The states X_1 to X_5 are independent states as they have no incoming connections from other states. They can be handled separately. States X_7 to X_{10} depend on the independent states X_1 to X_4 , respectively; they have a single incoming arrow with the complemental identity function $\text{compid}(\cdot)$ as combination function $c_V(V)$ for

mb base connectivity 1			mcw connection weights 1		
X_1	s_1	X_1	X_1	s_1	1
X_2	s_2	X_2	X_2	s_2	1
X_3	s_3	X_3	X_3	s_3	1
X_4	s_4	X_4	X_4	s_4	1
X_5	s_5	X_5	X_5	s_5	1
X_6	e_1	X_5	X_6	e_1	X_7
X_7	W_{s_5, e_1}	X_1	X_7	W_{s_5, e_1}	X_8
X_8	$W_{s_1, W_{s_5, e_1}}$	X_2	X_8	$W_{s_1, W_{s_5, e_1}}$	X_9
X_9	$W_{s_2, W_{s_1, W_{s_5, e_1}}}$	X_3	X_9	$W_{s_2, W_{s_1, W_{s_5, e_1}}}$	X_{10}
X_{10}	$W_{s_3, W_{s_2, W_{s_1, W_{s_5, e_1}}}}$	X_4	X_{10}	$W_{s_3, W_{s_2, W_{s_1, W_{s_5, e_1}}}}$	1

mcfw combination function weights				mcfp combination function parameters			
			1 2				1 2
			alogis comp				alogistic compid
			tic id				parameter
							1 2 1 2
							σ τ
X_1	s_1		1	X_1	s_1		18 0.2
X_2	s_2		1	X_2	s_2		18 0.2
X_3	s_3		1	X_3	s_3		18 0.2
X_4	s_4		1	X_4	s_4		18 0.2
X_5	s_5		1	X_5	s_5		18 0.2
X_6	e_1		1	X_6	e_1		8 0.5
X_7	W_{s_5, e_1}		1	X_7	W_{s_5, e_1}		
X_8	$W_{s_1, W_{s_5, e_1}}$		1	X_8	$W_{s_1, W_{s_5, e_1}}$		
X_9	$W_{s_2, W_{s_1, W_{s_5, e_1}}}$		1	X_9	$W_{s_2, W_{s_1, W_{s_5, e_1}}}$		
X_{10}	$W_{s_3, W_{s_2, W_{s_1, W_{s_5, e_1}}}}$		1	X_{10}	$W_{s_3, W_{s_2, W_{s_1, W_{s_5, e_1}}}}$		

ms speed factors 1			iv initial values 1		
X_1	s_1	0.08	X_1	s_1	0.2
X_2	s_2	0.05	X_2	s_2	0.1
X_3	s_3	0.015	X_3	s_3	0.11
X_4	s_4	0.008	X_4	s_4	0.1
X_5	s_5	0.2	X_5	s_5	0.5
X_6	e_1	0.5	X_6	e_1	0
X_7	W_{s_5, e_1}	0.05	X_7	W_{s_5, e_1}	0.8
X_8	$W_{s_1, W_{s_5, e_1}}$	0.05	X_8	$W_{s_1, W_{s_5, e_1}}$	0.8
X_9	$W_{s_2, W_{s_1, W_{s_5, e_1}}}$	0.004	X_9	$W_{s_2, W_{s_1, W_{s_5, e_1}}}$	0.8
X_{10}	$W_{s_3, W_{s_2, W_{s_1, W_{s_5, e_1}}}}$	0.004	X_{10}	$W_{s_3, W_{s_2, W_{s_1, W_{s_5, e_1}}}}$	0.8

Fig. 9 Role matrices for the fourth-order adaptive example network model

Fig. 10 Simulations for the fourth-order adaptive example network model with pathogens, internal defense system, pregnancy, and disgust occurring

Y . Then for state X_j depending on state X_i based on criterion (6), the equilibrium equation for X_j is,

$$\begin{aligned} \text{compid}(W_{X_i, X_j} X_i) &= X_j \\ 1 - W_{X_i, X_j} X_i &= X_j \end{aligned} \quad (13)$$

where W_{X_i, X_j} is also one of the X_k , actually for $k = j + 1$. So, the equation becomes

$$1 - X_{j+1} X_i = X_j$$

From this, the following equilibrium equations are found for X_7 to X_{10} .

$$\begin{aligned} X_7 &= 1 - X_8 X_1 \\ X_8 &= 1 - X_9 X_2 \\ X_9 &= 1 - X_{10} X_3 \\ X_{10} &= 1 - X_4 \end{aligned} \quad (14)$$

State X_6 depends on independent state X_5 , but also on the connection weight represented by X_7 . Based on criterion (6), the equilibrium equation for X_6 is

$$X_6 = \text{alogistic}_{8,0.5}(X_7 X_5) \quad (15)$$

After some algebraic rewriting, the following solutions in terms of X_1 to X_5 of the equilibrium equations (14) and (15) result.

$$\begin{aligned}
X_{10} &= 1 - X_4 \\
X_9 &= 1 - X_3 + X_4 X_3 \\
X_8 &= 1 - X_2 + X_3 X_2 - X_4 X_3 X_2 \\
X_7 &= 1 - X_1 + X_2 X_1 - X_3 X_2 X_1 + X_4 X_3 X_2 X_1 \\
X_6 &= \text{alogistic}_{8,0.5}((1 - X_1 + X_2 X_1 - X_3 X_2 X_1 + X_4 X_3 X_2 X_1) X_5)
\end{aligned} \tag{16}$$

For the nonzero independent states among X_1 to X_5 the equilibrium equation is

$$X_i = \text{alogistic}_{18,0.2}(X_i) \tag{17}$$

and the solution value for this Eq. (17) is numerically approximated by 0.999999427374 [15, Chap. 3, Sect. 3.7.4]. Therefore, for the analysis X_1 , X_2 , X_3 , X_4 , and X_5 , all have been assigned equilibrium value 0.999999427374. From these, the equilibrium values for the other states have been determined using Eqs. (16). All of these analyzed values are in the column under analysis in Table 10.

For verification for the scenario shown in Fig. 10, these analyzed equilibrium values have been compared to the approximate simulated equilibrium values that have been determined by running the model with $\Delta t = 0.1$ until $t = 3000$; see the column under simulation in Table 10. The deviations (the last column in Table 10) are in the order of 10^{-4} to 10^{-12} , which is fair and gives evidence that the implemented network model is correct in relation to the model's specified network characteristics.

6 Conclusion

In this paper, it has been illustrated how a network-oriented modeling approach based on adaptive temporal-causal networks can be used to model adaptive processes in different domains. The approach uses mathematical relations and functions as declarative building blocks incorporated in a canonical manner in a standard temporal-causal network format. A dedicated software environment is available, including a combination function library for aggregation of multiple impacts within a temporal-causal network, with more than 35 already predefined basic combination functions. This software environment makes design and simulation for such network models relatively easy. In [48], it has been shown how for any first-order differential equation, a combination function can be found so that the standard temporal-causal network format using this combination function is equivalent to that differential equation. Therefore, the standard temporal-causal network format in principle has a scope of applicability as wide as all (state-determined) dynamical systems or all sets of first-order differential equations [12, 13].

The network format is an adequate means to model in particular complex processes with a highly interactive and cyclic nature, and the approach to adaptive networks discussed here is especially useful for complex adaptive processes, as also has been illustrated here by the three main examples of the first-, second- and fourth-order adaptive processes, respectively.

For example, in social contexts, it is clear that an intense interaction between persons based on their mutual and often interrelated and adaptive cyclic relationships takes place. In the domain of neuroscience, the structures and mechanisms found also show that many parts in the brain are connected by connections that are part of adaptive cyclic paths, and such cycles and their adaptation play an important role in many mental processes (e.g. [49]). For example, usually, mental states trigger emotions, and these emotions, in turn, affect these and other mental states. Even leaving adaptation aside, for this type of circular effect, different views on causality and modeling are required, compared to the traditional views in the modeling of mental processes. For example, recall the quotation from Scherer [1] at the beginning of this chapter, which was stated for emotions and appraisal but has much wider applicability: *“What is the role of causality in the mechanisms suggested here? Because of the constant recursivity of the process, the widespread notion of linear causality (a single cause for a single effect) cannot be applied to these mechanisms. Appraisal is a process with constantly changing results over very short periods of time and, in turn, constantly changing driving effects on subsystem synchronization (and, consequently, on the type of emotion). (...) there is no simple, unidirectional sense of causality ...”* [1, p. 3470].

Similarly, from the angle of psychiatry, it is put forward that dynamics based on networks of causal relations play an important role in many mental disorders; for example: *“... instead of being effects of a common cause, psychiatric symptoms have been argued to cause each other. For instance, if one thinks that other people can read one’s mind (delusion), this may generate extreme suspicion (paranoia); this paranoia can lead one to avoid other people (social isolation), which, because one is no longer exposed to corrective actions of the social environment, may serve to sustain and exacerbate the relevant delusions. In this way, symptoms may form feedback loops that lead the person to spiral down into the state of prolonged symptom activation that we phenomenologically recognize as a mental disorder”* [50, pp. 5–6]. Also, *“Disorders with a cyclic pattern (e.g. bipolar disorder) may be accommodated in models for which the stable state is a cycle rather than a fixed point”* [50, p. 11].

Another example was a network perspective for medicine, which addresses adaptive chronic pain circuitry, as can be found in [51, 52]. Another example is [53], where a migraine is addressed from a disrupted plasticity perspective for the circuitry involved, a form of metaplasticity or second-order adaptation, like in Sect. 4.

For modeling the cell’s metabolic and life processes as biochemical networks within biology, Westerhoff et al. [6, 7] also point at the importance of such structures, *“the dynamic biochemical networks of life”*, for example: *“Living organisms persist by virtue of complex interactions among many components*

organized into dynamic, environment-responsive networks that span multiple scales and dimensions. Biological networks constitute a type of information and communication technology (ICT): they receive information from the outside and inside of cells, integrate and interpret this information, and then activate a response. Biological networks enable molecules within cells, and even cells themselves, to communicate with each other and their environment” [7, p. 1].

The network-oriented perspective provides a form of unification so that different types of processes become comparable, and we can, for example, compare the processes underlying human intelligence and behavior to the processes underlying bacterial behavior, as described in more detail in [5, 7]. For example: “We have become accustomed to associating brain activity—particularly activity of the human brain—with a phenomenon we call “intelligence.” Yet, four billion years of evolution could have selected networks with topologies and dynamics that confer traits analogous to this intelligence, even though they were outside the intercellular networks of the brain. Here, we explore how macromolecular networks in microbes confer intelligent characteristics, such as memory, anticipation, adaptation and reflection and we review current understanding of how network organization reflects the type of intelligence required for the environments in which they were selected. We propose that, if we were to leave terms such as “human” and “brain” out of the defining features of “intelligence,” all forms of life—from microbes to humans—exhibit some or all characteristics consistent with “intelligence”” [7, p. 1]. The emphasis in this quote is on how not only in the brain, but even in the smallest life forms, network structure, organization, dynamics, and adaptation occur to realize many if not all aspects of intelligence.

Hofstadter [54] emphasizes that for human intelligence and consciousness, not only different levels are important, but also that the ‘hierarchical’ structure formed by these levels is actually cyclic and is what he calls a Strange Loop: “My belief is that the explanations of “emergent” phenomena in our brains—for instance, ideas, hopes, images, analogies, and finally consciousness and free will—are based on a kind of Strange Loop, an interaction between levels in which the top level reaches back down towards the bottom level (...) In other words, a self-reinforcing “resonance” between different levels (...) The self comes into being at the moment it has the power to reflect itself” [54, p. 704].

He uses as one of his illustrations of this idea, artwork of Maurits Cornelis Escher (1898–1972). Escher was a famous Dutch graphic artist who made mathematically-inspired woodcuts, lithographs, and mezzotints. For example, in *Ascending and Descending* from 1960 as shown in Artwork, two lines of persons are seen, one of which walks in circles upstairs all the time and one downstairs, paradoxically both all the time after one cycle returning to the same level. The network-oriented modeling approach based on network reification or self-modeling covers this notion as well. Examples of higher-order adaptive network models applying this notion of Strange Loop can be found in [15, Chap. 8] and in [55].

The above cases indicate that networks can provide a unifying perspective over different disciplines. Such a unifying perspective of networks can be seen in many other cases as well. For example, by social contagion, someone can be a big

influencer for many others via social media, and in the same way, a flock of sheep following a leader sheep can be described by social contagion in a similar network structure, where the leading sheep is the big influencer. In such cases, usually, forms of causality and dynamics are important, and often also some form of adaptation. This means that a more traditional, static approach to causal modeling as in [17, 18] has limitations in the sense that the dynamic, cyclic, and interactive character described above is beyond the scope of such an approach, as is adaptation. To overcome this limitation, by adding a temporal dimension, a systematic approach to dynamics in causal modeling was obtained (based on temporal-causal network models) in [32], and by adding the notion of self-modeling by network reification, a systematic approach to adaptation (based on adaptive temporal-causal network models) in [15], as well. All this is supported by a dedicated software environment. Thus, a quite effective and efficient modeling approach is obtained, which is a relatively straightforward manner that enables the design of scientifically justified computational models for all kinds of human characteristics, grounded in human-directed disciplines such as neuroscience, cognitive science, and social sciences.

The examples in Sects. 3 to 5 illustrate how, based on the obtained approach, the required dynamics and adaptation within these different disciplines are indeed covered. These are just a few examples, while more can be found in [15, 32], and still many more in other papers, for example, available at https://www.researchgate.net/profile/Jan_Treur/research. This extensive collection of already available computational models covers many human characteristics such as autonomy and using goals, emotions and their regulation, decision making, mindreading, empathy, self-interpretation, and ownership and responsibility for actions. By adopting, improving, and refining such computational models, they can provide a good basis for the future development of human-like robots and other smart artifacts with human-like behavior.

Core Messages

- Causality and dynamics are central concepts in many disciplines.
- Also, often adaptiveness plays an important role, which can occur in different orders.
- The described (multi-order) adaptive network modeling approach addresses these.
- For social sciences, processes of bonding based on similarity have been described by adaptive social network models.
- For cognitive neuroscience, the concepts plasticity and metaplasticity have been identified as crucial for (first- and second-order) adaptiveness and, for example, the role this plays in stress-related disorders, where metaplasticity controls under which circumstances plasticity will occur.
- For biology, examples of evolutionary processes have been addressed by multi-order adaptive network models for the causal pathways involved.

- These are just a few illustrations of how the described modeling approach can and has been used to obtain computational models for a wide variety of human characteristics, which then can be applied to make future robots and other smart artifacts more human-like.

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