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From Individual Decisions to Collective Decisions Changing the World

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Abstract. Decision making plays an important role in many situations. This applies both to individual decision making and collective decision making, although the scope of the consequences of a decision may be quite different for both cases. In the collective case a worldwide scale can be reached whereas in an individual situation often the scale is limited to personal life. Nevertheless, also collective decisions usually have a basis in decisions of individuals within a population. In this paper it is discussed how in certain cases individual decisions can indeed lead to collective decisions with a worldwide scope of consequences. Two mechanisms for this are considered in particular: influencer-driven social contagion within social networks and plasticity-driven evolution within biological populations.

Keywords: Collective decision · Contagion · Influencer · Evolution · Behavioural plasticity

1 Introduction

Decision making takes place both at an individual level and at a collective level. In principle, the scope of the consequences of a decision may be quite different for both cases. In a collective case a decision may affect people on a worldwide scale while in an individual situation often the scale is more limited to personal life. However, also to achieve collective decisions usually a basis of decisions of individuals is needed. The question is how big this basis has to be. In a democratic setting it is sometimes (mistakenly) believed that a majority is needed for collective decisions. In reality, due to certain mechanisms often a much smaller number of individual decisions already suffices to achieve in an emergent manner a collective decision with far-reaching consequences. In this paper two of such mechanisms are analysed in particular. One of them is the social mechanism of influencer-driven social contagion within social networks [5, 8] and the other one the biological mechanism of plasticity-driven evolution within biological populations [4, 6, 9, 11]. In both cases, through the mentioned mechanisms, decisions of a very few individuals already can grow out to emerging collective decisions with worldscale consequences.

In the paper, after a brief introduction of the modeling approach used (in Sect. 2), first (in Sect. 3) the social mechanism will be addressed, and next (in Sect. 4) the biological

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mechanism. In both cases it will be shown how due to these mechanisms that act as a kind of decision amplifier, decisions of a percentage of individuals as low as 5% of a population or even lower can already achieve a collective decision.

2 Adaptive Temporal-Causal Network Modeling

In the literature, mental, physical or biological processes often are described by pathways through causal networks, also called *causal pathways*. This causal perspective suggests that for modeling of such processes, causal network modeling approaches may be suitable. For the topic addressed here, a particular causal modeling approach is required that is able to model temporal dynamics and adaptivity of such causal pathways. The modeling approach based on reified adaptive temporal-causal networks, as presented in [10], fulfills such requirements and is used here. First a brief introduction to this adaptive causal modeling approach. A temporal-causal network is characterised by the following types of characteristics:

- **connectivity characteristics**: the *connections* from nodes (also called states) X to Y and their *weights* $\boldsymbol{\omega}_{X,Y}$; here states X have varying values $X(t)$ over time t
- **aggregation characteristics**: for each state Y, by a *combination function* $\mathbf{c}_Y(..)$ some form of aggregation is applied to the single causal impacts $\boldsymbol{\omega}_{X_i,Y}X_i(t)$ from each of its incoming connections from states X_1, …, X_k
- **timing characteristics**: each state Y has a *speed factor* $\boldsymbol{\eta}_Y$ indicating how fast it changes upon causal impact

The standard difference equations used for simulation and mathematical analysis incorporate these three types of network characteristics $\boldsymbol{\omega}_{X,Y}$, $\mathbf{c}_Y(..)$, $\boldsymbol{\eta}_Y$: for any state Y it holds

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

where X_1, …, X_k are the states from which Y gets incoming connections; this can also be expressed by an equivalent differential equation. These concepts enable to design and analyse causal networks with their dynamics by declarative mathematically defined relations. For example, for analysis of equilibria (i.e., when no change occurs: $\mathbf{d}Y/\mathbf{d}t = 0$), assuming a nonzero speed factor, based on (1), the criterion is

$$\mathbf{c}_Y(\boldsymbol{\omega}_{X_1,Y}X_1(t), \ldots, \boldsymbol{\omega}_{X_k,Y}X_k(t)) = Y(t) \qquad (2)$$

See [10], Ch 2 for more information on modeling and analysis based on temporal-causal networks.

This far adaptive causal networks, in which the network characteristics $\boldsymbol{\omega}_{X,Y}$, $\mathbf{c}_Y(..)$, $\boldsymbol{\eta}_Y$ may change over time, are not covered yet. However, recently it was found out that extending the approach by the notion of *network reification* makes this network modeling perspective becomes well-suitable to design *adaptive* networks as well, still by declarative mathematically defined relations. Reification [2] generally means making abstract things concrete; well-known examples are representing relations between objects by

objects themselves, and representing logical statements by numbers. Network reification works by adding the (this time adaptive) network characteristics $\boldsymbol{\omega}_{X,Y}$, $\mathbf{c}_Y(..)$, $\boldsymbol{\eta}_Y$ in a reified form to the network as nodes $\mathbf{W}_{X,Y}$, $\mathbf{C}_{i,Y}$, $\mathbf{H}_Y$ at a second level, called *adaptation level* or *reification level*, while the original network forms the *base level*. This introduces in the network modeling area ideas similar to metalevel architectures and metaprogramming as found in different other forms in a long standing tradition in AI: e.g., [1]. By this construction, an extended, reified network is obtained, which can be verified as being again a temporal-causal network [10], Ch 10. As for any temporal-causal network model, the dynamics of such a reified network is described in a declarative mathematical manner by the states and their connections, including those at the reification level and causal interlevel connections for the impact from one level to the other. But now these dynamics cover adaptivity of the base network. The construction can iteratively be applied to obtain multiple reification levels (*first-, second, third-, … order adaptation levels*) to model multiple orders of adaptation of a network.

A Matlab-translated form of a variant of difference equation (1) is part of the core of the Computational Reified Network Engine, where also (based on the role matrices that are made available in Matlab) an adequate administrative process of dynamically determining the right values of its variables for each state is taken care for. For more details on this approach to adaptive temporal-causal modeling and its applications, see [10].

3 A Social Mechanism for Decision Amplification

Social networks are used to model how different persons are connected to each other; in this case each node represents the state (for an opinion, belief, or emotion, for example) of one person, and the connections represent how strong they influence each other. Social contagion [5] is a form of dynamics within a social network where the activation level of each state is influenced by the states from which it has incoming connections. Not every node has the same influence in such a network, as it depends on the numbers and strengths of the connections. Nodes with a major influence are usually called *influencers*. Social networks and social contagion are generic concepts and mechanisms that can be applied to a wide variety of contexts, varying, for example, from sheep following an influencer sheep Boris for a place to go, to people following an influencer person for an opinion to go for (see Fig. 1), or even to how we transmit virus infections like for the Corona Covid-19 virus.

Being an influencer depends on a number of factors. Being a charming and funny person helps a lot. Knowing to accompany your interaction with positive emotions can make (e.g., in a retweeting context) the strength of propagation up to 60% higher [7]. And once someone is an influencer, the effect indeed can be decisive, especially when one is independent and not influenced by others. For example, in [8] the rise in veganism has been analysed, where it turned out that leaving out the biggest influencer would lead to a completely different pattern for the population: without the influencer the percentage of veganism-minded persons would stay 70% lower; see Fig. 2.

In the graphs shown in Figs. 3 and 4 it is analysed further how influencers affect the total pattern. The specified example network used is the fully connected network shown in Box 2.2 in [10], Ch 2. The combination function used is the scaled sum function:

$$\mathbf{ssum}_{\lambda}(V_1, \ldots, V_k) = \frac{V_1 + \ldots + V_k}{\lambda} \tag{3}$$

Here λ is a scaling factor. For this parameter, for each state Y a normalised value was used, which is the sum of the weights of the incoming connections for Y.

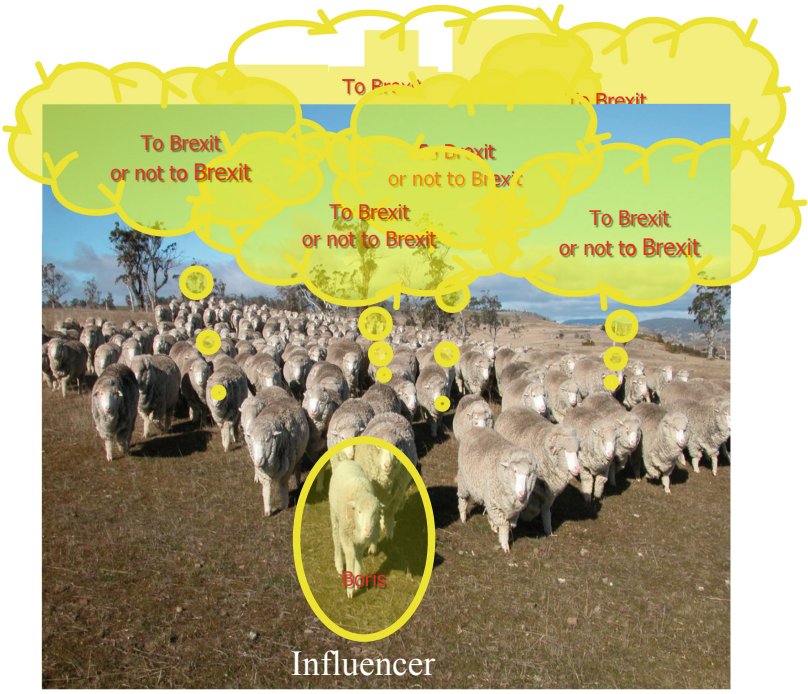

Fig. 1. The British To B or Not To B of this time

This makes the incoming aggregated impact on Y the weighted average of the values of the states from which the incoming connections are coming. If values would be used that are lower than the normalised values, than the patterns would go to 0 in an artificial manner, if higher values would be used they would go to 1 in an artificial manner. As shown in (1) above, the arguments V_1 to V_k in (3), but also in (4), (5) and (6) below are used for the single impacts from each of the causal connections from X_i to Y; these single impacts are calculated as the product $\omega_{X_i,Y}(t)X_i(t)$ of the connection weight $\omega_{X_i,Y}(t)$ and state value $X_i(t)$ at that time t.

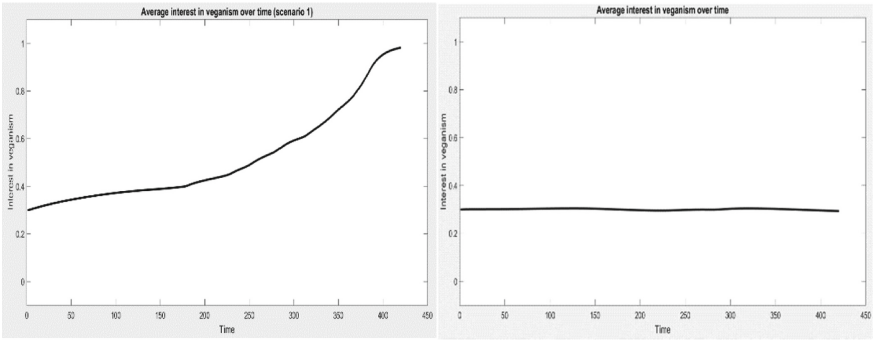

Fig. 2. The simulated rise of veganism-mindedness with influencer (left, approximating the real-world curve) or without the influencer (right). Here as a form of scaling the 1 at the vertical axis stands for the actual number of veganism-minded persons at the end time; these graphs were adopted from [8]

In Fig. 3 the difference is shown between a situation that everybody is influenced by the others and a situation where one (an influencer) is not influenced and has an arbitrary independent pattern, what then is followed by everybody. Here the ultimate level of the independent person is decisive for everyone. If that level would be 1, everybody would go there, or when 0 everybody to 0.

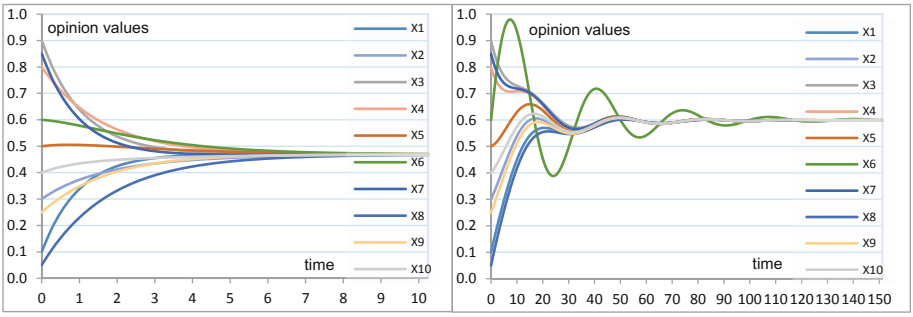

Fig. 3. Simulations for the example Social Network (a) Left graph: no influencer, everybody is influenced and ends up in some form of joint average value (b) Right graph: one person is influencer and is not influenced him/herself but instead makes an arbitrary independent pattern (the strongly fluctuating line in green), whereby everybody else is following.

In Fig. 4 it is shown what happens when there are two independent influencers (both not influenced by anyone, also not by each other). The left graph show that two opposite opinions of the two influencer make that everybody becomes divided. However, the two other graphs show that when the two influencers have similar opinions, everybody groups between them, wherever on the scale that is. The patterns found in Fig. 3 and 4 can be explained by the theorem below. First a definition.

Definition (Properties of Connectivity and Aggregation)

a) State Y is *reachable* from state X if there is a directed path from X to Y with nonzero connection weights and speed factors. The network is *strongly connected* if this holds for every X and Y.
b) A combination function $c_Y(..)$ is called *normalised* if for all Y it holds

$$c_Y(\omega_{X_1,Y}, \ldots, \omega_{X_k,Y}) = 1$$

where X_1,.., X_k are the states from which state Y gets incoming connections
c) A combination function $c_Y(..)$ is called *monotonically increasing* if

$$U_i \leq V_i \text{ for all } i \Rightarrow c_Y(U_1, \ldots, U_k) \leq c_Y(V_1, \ldots, V_k)$$

d) A combination function $c_Y(..)$ is called *strictly monotonically increasing* if

$$U_i \leq V_i \text{ for all } i, \text{ and } U_j < V_j \text{ for at least one } j \Rightarrow c_Y(U_1, \ldots, U_k) < c_Y(V_1, \ldots, V_k)$$

e) A combination function $c_Y(..)$ is *scalar-free* if $c_Y(\alpha V_1, \ldots, \alpha V_k) = \alpha\, c_Y(V_1, \ldots, V_k)$ for all $\alpha > 0$

Theorem
Suppose the network has normalised, scalar-free and strictly monotonic combination functions, then the following hold:

a) If the network is strongly connected, then in an equilibrium state all states have the same value.
b) Suppose the network has one or more independent states and the subnetwork without these independent states is strongly connected. If in an equilibrium state all independent states have equilibrium values V with $V_1 \leq V \leq V_2$, then all states have equilibrium values V with $V_1 \leq V \leq V_2$. In particular, when all independent states have the same equilibrium value V, then all states have this same equilibrium value V.

This theorem is Theorem 3 from [10], Ch 11, p. 266. The connectivity and combination functions used in Figs. 3 and 4 fulfil the conditions of the above Theorem 1. Here a) explains that in the left graph of Fig. 3 all curves go to one value as in this case the network is strongly connected, and b) for one independent state explains that in the right graph of Fig. 3 all curves follow the independent curve. Moreover, b) for two independent states explain the three graphs in Fig. 4 where all curves end up between the two constant ones.

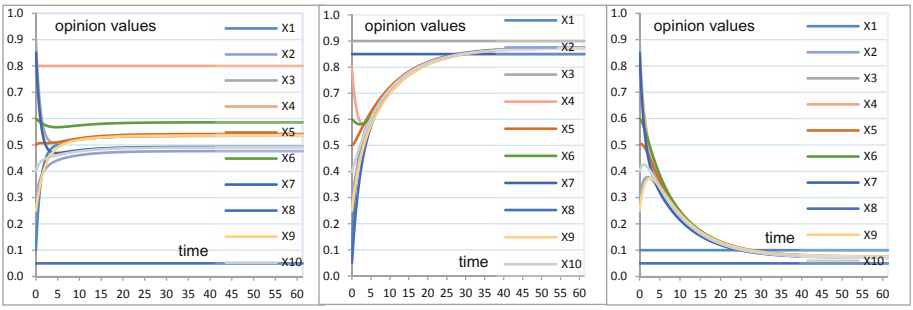

Fig. 4. How two independent influencer states affect all equilibrium values for the opinions of different persons. Left graph: the two constant lines for X_8 and X_4 at 0.05 and 0.8. Middle graph: the two constant lines for X_7 and X_{10} at 0.85 and 0.9. Right graph: the two constant lines for X_8 and X_1 at 0.05 and 0.1

4 A Biological Mechanism for Decision Amplification

From a biological perspective, an important factor in evolution for a population is a process of exploration of different options within a certain environmental context. Such an exploration can take place as a biological mechanism, for example, based on mutations and/or genetic reshuffling due to sexual reproduction. However, another important form of exploration on a much shorter time scale is based on individuals having during their lifetime some form of variation and plasticity in the decisions about their response to an environment, for example, a certain freedom to decide for the type of food to go for.

Plasticity Led Evolution

The *plasticity led evolution theory* assumes a main role of such plasticity for the direction of evolutionary processes; e.g., [4, 6, 9, 11]: plasticity can lead evolution so that genes may become followers rather than leaders in evolution. Two types of plasticity distinguished are *contextual plasticity* and *developmental plasticity*. The former type concerns a form of instantaneous response in relation to present stimuli from the environmental context. The latter type concerns adaptation of an individual organism to the environmental context taking into account stimuli over time. This may involve learning by changes in the causal pathways and connectivity in the brain or, in a wider developmental plasticity context, other changes in the organism's physical makeup that make the organism better suited for the environmental demands. Both types of plasticity are covered here.

Within the spectrum of plasticity, *behavioral plasticity* involves among others the ability to decide between different *behavioral options*; this will also be covered here as will be discussed further below, focusing on the (mental) decision making process in which prediction of effects of decision options and valuation of these predicted effects play an important role. An important assumption made here is that in a population of reasonable size in a contextually rich enough environment, different behavioral options do actually occur: it is assumed that for each option there are at least some individuals that decide for them.

In this section, the role of behavioral plasticity for evolution will be analysed computationally by using the adaptive temporal-causal modeling approach described in [10], covering different forms and orders of adaptation. This approach enables to model from a perspective of causality all kinds of adaptive processes in the real world in an integrative manner, including adaptive mental processes and adaptive biological processes. In particular, it is applied to the adaptive mental processes of decision making involved in behavioral plasticity, and the adaptive biological processes involved in optimising the biological effectiveness of the performance of such chosen behaviors for the given environmental context.

Historically, it has been debated from time to time whether and in how far what individual organisms decide to do during their lifetime has effect on the evolution of their species (plasticity-led evolution; e.g., [4]). For example, in [6] the idea is illustrated by the following simple scenario:

> 'Consider a finch population that finds itself on an island with larger seeds than previously encountered. Behavioral flexibility results in consumption of these seeds, but among the consumers, natural selection favours the larger individuals who can more easily crack large seeds. The net result is evolution of large size' [6], p. 1434

This scenario can be refined a bit more, by assuming that also smaller seeds are available, but they have a lower quality, and some individuals will decide to go for these smaller seeds and others will decide to go for the larger seeds. Based on the subpopulation of the individuals that decide for the larger seeds, for the population evolutionary development can take place in that direction. This evolutionary development would not take place if all individuals would decide for the bad smaller seeds. In this sense the decision of some of the individuals to go for the larger seeds drives the evolution.

In recent years, it has become more plausible that such effects indeed can be found, especially in cases of strongly changing environmental conditions (as we actually also experience nowadays). Plasticity can concern, for example, physiological processes, such as improving running performance over a lifetime in order to escape from new, faster enemies. But plasticity can also concern a choice for some behavior. Behavioral plasticity in particular, makes that potentially multiple options for behavior are available to an individual, and that within a given environmental context by each individual one of these options is decided for (a mental process) and then performed (a physiological process). So, within behavioral plasticity two subcategories can be distinguished: *mental behavioral plasticity* (plasticity for making decisions in the given environmental context) and *physiological behavioral plasticity* (plasticity for performing the decided behavior in the given environmental context).

These forms of plasticity can take place instantaneously (contextual behavioral plasticity), based on current stimuli, or over a longer time duration (developmental behavioral plasticity), after learning based on stimuli over a longer time period. Due to variation, in general within a population different individuals will make different decisions by (contextual and/or developmental) mental behavioral plasticity. Moreover, a given decision entails performing that particular behavior accordingly, so that also physiological optimisation of it may occur by (contextual and/or developmental) physiological behavioral plasticity. The latter type of optimisation will only take place when that behaviour is

actually decided for, so it has a dependence on the mental behavioural plasticity. When a behaviour is not decided for, no optimisation will take place and instead degradation may take place. But when the behaviour is decided for and subsequently by physiological behavioral plasticity, optimisation of that behaviour takes place within a lifetime, at the population level also *genetic optimisation* can start to take place, making the whole an evolutionary process.

The Designed Adaptive Network Model

The specific adaptive temporal-causal model used here is shown in conceptual graphical form in Fig. 5. For an explanation of the states, see Table 1. For easier understanding, the reified network model shown in the bottom part of Fig. 5 is accompanied by a more global overview of the different addressed processes as boxes in the upper part of Fig. 5. The model addresses a scenario similar to the one for a finch population quoted above, but in a more refined form involving both contextual and developmental plasticity and both mental behavioral plasticity and physiological behavioural plasticity.

The three main processes at the base level and their causal pathways are the following. They are distinguished as being individual-contributed (plasticity-based optimisation) or population-contributed (genetic optimisation) pathways. Note that for developmental plasticity and genetic optimisation both the base level and the adaptation level are involved, the latter of which is described after the base level.

Individual-Contributed Decision Making

The causal pathways for *individual-contributed decision making* for the two considered options address both contextual and developmental mental behavioural plasticity. At the base level they are modeled by the states srs_{c_i}, ps_{a_i}, and srs_{e_i} and their connections (indicated by the left box in base level of the upper part of Fig. 5). The context representation srs_{c_i} (which is based on the actual environmental context ws_{c_i}) induces (partial) activation of ps_{a_i}. Valuation of the options takes place by predicting the expected effect e_i of choice a_i by generating activation of srs_{e_i}, and using that to evaluate and modulate ps_{a_i}. So, the decision made (high activation level of ps_{a_i}) depends on the individual's connection from srs_{c_i} to ps_{a_i}, but also on the connections of the predictive valuation loop via srs_{e_i}.

Individual-Contributed Behavioral Performance

The causal pathways for *individual-contributed behavioral performance* (once a choice was made) address both contextual and developmental physiological behavioural plasticity. At the base level they are modeled by the states ps_{a_i} , and ws_{e_i} and their connections (indicated by the middle box in base level of the upper part of Fig. 5). This describes how the individual is able to perform the chosen behavioral option: a high level of ws_{e_i} is good performance, a low level bad performance; this depends on the individual's connection from ps_{a_i} to ws_{e_i}, together with the given influence of the environmental context ws_{c_i} on ws_{e_i} .

Population-Contributed Behavioral Performance

The causal pathways for *population-contributed behavioral performance* address genetic optimisation. At the base level they are also modeled by the states ps_{a_i} and ws_{e_i} in the

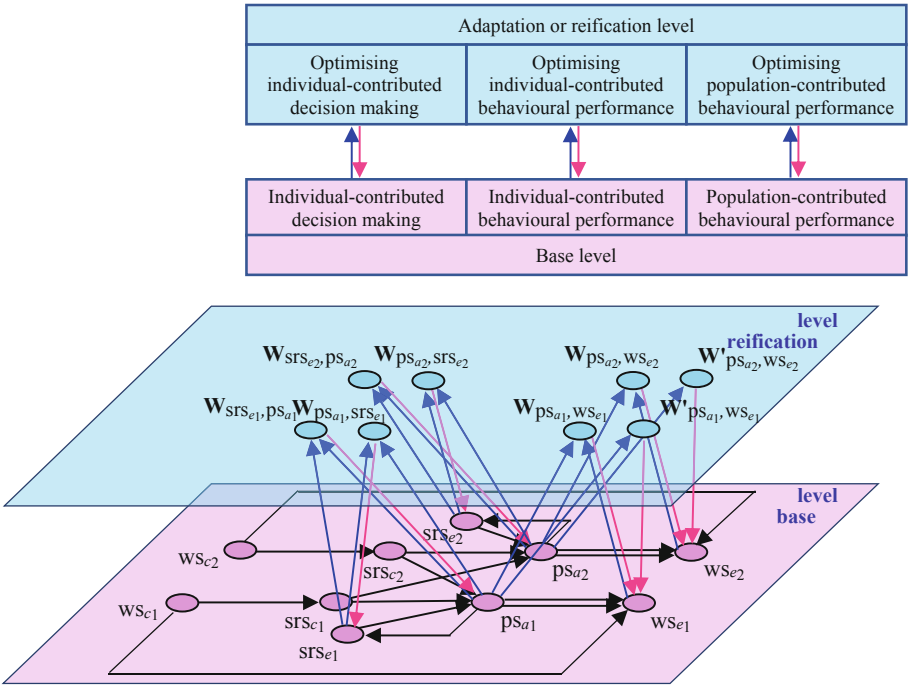

Fig. 5. The connectivity of the adaptive temporal-causal network model. Black arrows: intralevel connections for the base level processes. Blue arrows: upward interlevel connections informing the adaptation processes as a form of monitoring of the base level processes. Red arrows: downward interlevel connections effectuating the adaptations at the base level. The two arrows from ps_{ai} to ws_{ei} describe two contributions: one models individual-contributed causal pathways to behavioural performance and the other one population-contributed causal pathways to behavioural performance.

base network and their connections (indicated by the right box in base level of the upper part of Fig. 5). Note that there are two different connections from ps_{a_i} to ws_{e_i} which distinguish the individual-contributed causal pathways from the population-contributed causal pathways for the effect of a chosen behavior. In this way the two different causal pathways are distinguished, and aggregated by the combination function of ws_{e_i}.

How the Different Causal Pathways Become Adaptive

The different processes at the base level become adaptive by adding appropriate reification states at the adaptation level and connecting them in the right way to the states at the base level. In this way developmental plasticity and genetic optimisation are addressed.

The causal pathways for *individual-contributed decision making* become adaptive by (optimising) learning of the individual during its lifetime (developmental mental behavioral plasticity). As more often used for mental processes, this is modeled by applying hebbian learning [3], this time to the connections involved in the loops for evaluation based on effect prediction. At the adaptation level the connection weight reification states $\mathbf{W}ps_{a_i},srs_{e_i}$ and $\mathbf{W}srs_{e_i},ps_{a_i}$ take care of this, thereby applying to them the hebbian

Table 1. The states and their explanation

state nr	state name	explanation
X_1	ws_{c_1}	World state for environmental context c_1
X_2	ws_{c_2}	World state for environmental context c_2
X_3	srs_{c_1}	Sensory representation state for environmental context c_1
X_4	srs_{c_2}	Sensory representation state for environmental context c_2
X_5	ps_{a_1}	Preparation state for option a_1
X_6	ps_{a_2}	Preparation state for option a_2
X_7	srs_{e_1}	Sensory representation state for (predicted) effect e_1 of option a_1
X_8	srs_{e_2}	Sensory representation state for (predicted) effect e_2 of option a_2
X_9	ws_{e_1}	World state for (actual) effect e_1 of option a_1
X_{10}	ws_{e_2}	World state for (actual) effect e_2 of option a_2
X_{11}	$\mathbf{W}ps_{a_1},ws_{e_1}$	State for the weight of the individual-contributed connection from ps_{a_1} to ws_{e_1}
X_{12}	$\mathbf{W}ps_{a_2},ws_{e_2}$	State for the weight of the individual-contributed connection from ps_{a_2} to ws_{e_2}
X_{13}	$\mathbf{W}ps_{a_1},srs_{e_1}$	State for the weight of the individual-contributed connection from ps_{a_1} to srs_{e_1}
X_{14}	$\mathbf{W}ps_{a_2},srs_{e_2}$	State for the weight of the individual-contributed connection from ps_{a_2} to srs_{e_2}
X_{15}	$\mathbf{W}srs_{e_1},ps_{a_1}$	State for the weight of the individual-contributed connection from srs_{e_1} to ps_{a_1}
X_{16}	$\mathbf{W}srs_{e_2},ps_{a_2}$	State for the weight of the individual-contributed connection from srs_{e_2} to ps_{a_2}
X_{17}	$\mathbf{W'}ps_{a_1},ws_{e_1}$	State for the weight of the population-contributed connection from ps_{a_1} to ws_{e_1}
X_{18}	$\mathbf{W'}ps_{a_2},ws_{e_2}$	State for the weight of the population-contributed connection from ps_{a_2} to ws_{e_2}

learning combination function $\mathbf{hebb}_{\boldsymbol{\mu}}(.)$ defined below. The causal pathways for *individual-contributed behavioral performance* also become adaptive by (optimising) learning of the individual during its lifetime (developmental physiological behavioral plasticity). Also this was modeled by applying hebbian learning to the respective connections from ps_{a_i} to ws_{e_i}. This strengthens the connections in proportion to the activation levels of ps_{a_i} and ws_{e_i}. At the adaptation level, the connection weight reification states $\mathbf{W}ps_{a_i},ws_{e_i}$ take care for this, thereby applying the hebbian learning combination function $\mathbf{hebb}_{\boldsymbol{\mu}}(..)$ defined below.

The causal pathways for *population-contributed behavioral performance* become adaptive by genetic optimisation over generations. As this is assumed to be related to the choice to practice some behavior, this is modeled by applying state-connection modulation to the respective connections from ps_{a_i} to ws_{e_i}. This strengthens the connections in proportion to the activation level of ps_{a_i}, representing the choice. At the adaptation level the connection weight reification states $\mathbf{W}'ps_{a_i},ws_{e_i}$ take care for this, thereby applying the combination function $\mathbf{scm}_{\boldsymbol{\alpha}}(.)$ defined below.

The Combination Functions Used

For combination functions, within the modeling environment a library is available with 35 functions. For example, the logistic sum combination function $\mathbf{alogistic}_{\boldsymbol{\sigma},\boldsymbol{\tau}}(..)$ is available (with parameters $\boldsymbol{\sigma}$ for steepness and $\boldsymbol{\tau}$ for threshold) defined by

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

This is used for the base states in the presented model. In particular useful for reification levels is the combination function $\mathbf{hebb}_{\boldsymbol{\mu}}(..)$ for Hebbian learning [3] (where $\boldsymbol{\mu}$ is a

persistence factor), used by the individual-contributed optimisation states X_{11} to X_{16}, and **scm$_\alpha$(..)** (here α is a modulation factor) used by the population-contributed behavioral optimisation states X_{17} and X_{18}. They are defined by:

$$\mathbf{hebb}_\mu(V_1, V_2, W) = V_1 V_2 (1 - W) + \mu W \tag{5}$$

where V_1 is used for *X(t)*, V_2 for *Y(t)* and W for $\mathbf{W}_{X,Y}(t)$

$$\mathbf{scm}_\alpha(W, V) = W + \alpha V W (1 - W) \tag{6}$$

where V is used for *X(t)* in this case and W for $\mathbf{W}'_{X,Y}(t)$

Simulation Results

In Fig. 6 simulation results are shown. In this scenario, within environmental context c_2 it is decided to make a choice for a_2, whereas the previous choice within context c_1 was a_1. The switch of environmental context (at time 150) goes together with contextual plasticity for an instant switch from a_1 to a_2.

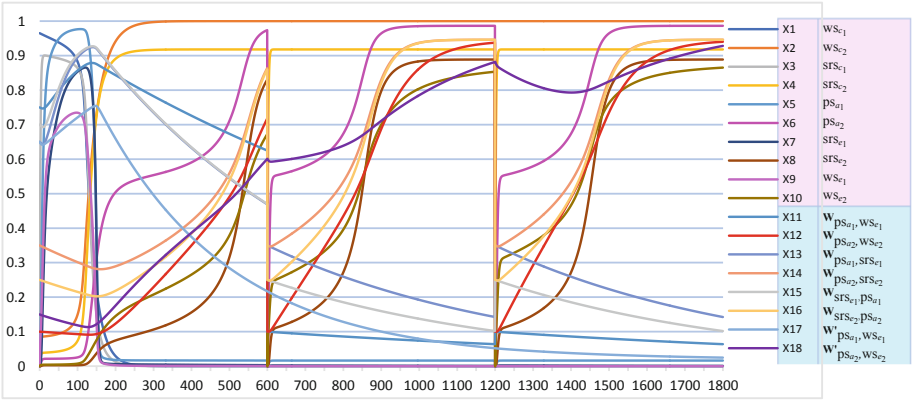

Fig. 6. The decision for behavioural option a_2 in the environmental context c_2

Note that the specific values used for the network characteristics can be found at URL https://www.researchgate.net/publication/340095095. In Fig. 6 it is shown how different generations are incorporated by repetitively resetting all effects learnt during lifetime (in this case shown at time 600 and at time 1200). Next, developmental plasticity during lifetime to optimise both the decision making and the behavioral performance for this choice takes place. Then, on the long term it leads to genetic optimisation over generations. The main observations for this scenario shown in Fig. 6 are shown in Table 2.

A similar simulation was made for the other choice a_1. It was found that for both choices a_1 and a_2 a form of adaptation to the new environmental context takes place, but they are of different quality: after time point 1600, for choice a_2 the individual health level became higher than 0.8 and for choice a_1 it stayed lower than 0.5. This can be quantified by at least 60% difference in health after time point 1600.

Table 2. Main observations for the scenario in Fig. 6

Time periods	Main patterns for states and causal connections
0-100 Environmental context c_1	• context representation X_3 (light grey line) for c_1 up to level 0.9 • X_5 (blue line) for choice for a_1 up to level 0.98 • effect X_9 (dark grey line) of performance for a_1 up to level 0.75
	• plasticity-determined causal pathway for decision making for a_1 via ps_{a1} and srs_{e1} (grey line for X_{13} and X_{15}) up to 0.95 • plasticity-determined causal pathway for performance for a_1 via connection from ps_{a1} to ws_{e1} (dark blue line for X_{11}) up to 0.94 • genetics-determined causal pathway for performance for a_1 via connection X_{17} from ps_{a1} to ws_{e1} (light blue line) gradually up to above 0.65
100-150 Switch to environmental context c_2	• context representation X_3 (light grey line) for c_1 drops to 0 • X_5 (blue line) for choice for a_1 drops to 0 • effect X_9 (dark grey line) of performance for a_1 drops to 0
	• not much change in connections
150-200 Contextual plasticity: instant individual response	• context representation X_4 (orange line) for c_2 up to level 0.9 • X_6 (pink line) for choice for a_2 up to level 0.5 • effect X_{10} (red line) of performance for a_2 up to level 0.15
	• not much change in connections
150-500 Developmental plasticity: inidividual-contributed adaptation during lifetime	• X_6 (pink line) for choice for a_2 up to level 0.92 • effect X_{10} (red line) of performance for a_2 up to 0.53
	• plasticity-determined causal pathway for decision making for a_2 via ps_{a2} and srs_{e2} (light brown and yellow line for X_{14} and X_{16}) up to around 0.75 • plasticity-determined causal pathway for performance for a_2 via connection X_{12} (green line) up to 0.79 • genetics-determined causal pathway for performance for a_2 via connection X_{18} (purple line) stays below 0.3 • plasticity-determined causal pathways for decision making and performance for a_1 (X_{11}, X_{13}, and X_{15}) all degrade to levels below 0.45
150-1600 Genetic optimisation: population-contributed adaptation	• X_6 (pink line) for choice for a_2 reaches level 0.98 (pink line) • effect X_{10} (red line) of performance for a_2 up to 0.82
	• genetics-determined causal pathway for performance for a_2 (purple line for X_{18}) gradually rises to above 0.76 • genetics-determined causal pathway for performance for a_1 (light blue line for X_{17}) degrades to below 0.2

As formulated above, within the population in actual situations there both are individuals that make choice a_1 and individuals that make choice a_2. As the individuals going for choice a_2 fit better to environmental context c_2 and therefore gain a better health, by natural selection over time their numbers can be expected to outperform the numbers of the other individuals that go for a_1. For example, the 60% better health can be translated into getting 60% more offspring per time unit. This has been modeled by assuming an overall example population consisting of two subpopulations: Subpopulation 1 formed by individuals going for choice a_1, and Subpopulation 2 with individuals going for choice a_2. Figure 7 shows the outcome for the relative sizes S_1 and S_2 of these two subpopulations over time, as modeled by the following discrete difference equations where the step from t to $t + 1$ indicates a periodic production of offspring, which in actual time may depend on the considered species:

$$S_1(t+1) = \frac{S_1(t) + \gamma_1 S_1(t)}{S_1(t) + \gamma_1 S_1(t) + S_2(t) + \gamma_2 S_2(t)} \qquad S_2(t+1) = \frac{S_2(t) + \gamma_2 S_2(t)}{S_1(t) + \gamma_1 S_1(t) + S_2(t) + \gamma_2 S_2(t)} \quad (7)$$

Here, the factor for offspring per period for Subpopulation 1 was set on $\gamma_1 = 0.5$ and for Subpopulation 2 60% higher, so $\gamma_2 = 0.8$. Initial sizes of Subpopulations 1 and 2 were set at 95% (0.95 for S_1) and 5% (0.05 for S_2). The outcome shown in Fig. 7 indeed shows the turn evolution takes based on part of the individuals' choice for a_2. If these individuals would not have been capable of deciding for that choice a_2, evolution would have taken the turn to optimisation of performance in relation to choice for a_1, which in this case is clearly suboptimal compared to the optimisation of performance for the other choice for a_2.

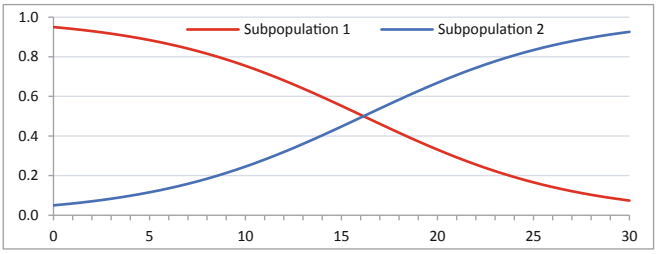

Fig. 7. Development of the two subpopulations over generations

5 Discussion

Decision making covers both individual decision making and collective decision making, but the scope of the consequences of a decision may be quite different for both cases. Collective decisions usually have a basis in decisions of individuals within a population. In this paper it was discussed how in certain cases individual decisions can lead to collective decisions with a worldwide scope of consequences. Two mechanisms for this were considered in particular: influencer-driven social contagion within social networks (e.g., [5, 8]) and plasticity-driven evolution within biological populations (e.g., [4, 6, 9, 11]). In both cases it was shown how in an emergent manner decisions by a very low number of individuals can generate a collective decision for the whole population.

In the first case it was shown that by social contagion one or two influencers can make a major part of the individuals go for similar decisions, as, for example, illustrated for political issues such as the Brexit issue. In the second case it was also shown that if only a very low percentage of individuals makes some decision, after some generations this can lead to practically the whole population making the same decision.

References

1. Demers, F.N., Malenfant, J.: Reflection in logic, functional and object oriented programming: a short comparative study. In: IJCAI95 Workshop on Reflection and Meta-Level Architecture and their Application in AI, pp. 29–38 (1995)
2. Galton, A.: Operators vs. arguments: the ins and outs of reification. Synthese **150**, 415–441 (2006)
3. Hebb, D.O.: The Organization of Behavior: A Neuropsychological Theory. Wiley, New York (1949)
4. Levisa, N.A., Pfennig, D.W.: Plasticity-led evolution: evaluating the key prediction of frequency-dependent adaptation. Proc. R. Soc. B **286**, 20182754 (2019)
5. Levy, D.A., Nail, P.R.: Contagion: a theoretical and empirical review and reconceptualization. Genetic Soc. General Psychol. Monographs **119**(2), 233–284 (1993)
6. Price, T.D., Qvarnstrom, A., Irwin, D.E.: The role of phenotypic plasticity in driving genetic evolution. Proc. R. Soc. B **270**, 1433–1440 (2003). https://doi.org/10.1098/rspb.2003.2372
7. Schoenmaker, R., Treur, J., Vetter, B.: A temporal-causal network model for the effect of emotional charge on information sharing. Biologically Inspired Cogn. Archit. **26**, 136–144 (2018)
8. Sijm, M., Exel, C., Treur, J.: Using a temporal-causal network model for computational analysis of the effect of social media influencers on the worldwide interest in Veganism. In: Yang, X-S., Sherratt, S., Dey, N., Joshi, A. (eds.) Proceedings ICICT 2019. Advances in Intelligent Systems and Computing, vol. 1027, pp. 129–140. Springer Publishers, New York (2020)
9. Snell-Rood, E.C.: An overview of the evolutionary causes and consequences of behavioural plasticity. Animal Behav. **85**(5), 1004–1011 (2013)
10. Treur, J.: Network-Oriented Modeling for Adaptive Networks: Designing Higher-Order Adaptive Biological, Mental and Social Network Models, Springer-Nature, Cham (2020)
11. Ulle, T., Feiner, N., Radersma, R., Jackson, I.S.C., Rago, A.: Developmental plasticity and evolutionary explanations. Evol. Dev. **22**(1/2), 47–55 (2019)