

Rationality for Adaptive Collective Decision Making Based on Emotion-Related Valuing and Contagion

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Abstract. In this paper it is explored how adaptive collective decision making can be evaluated with respect to learning speed and rationality of the decisions. A collective decision model is presented based on interacting adaptive agents that learn from their experiences by a Hebbian learning mechanism. The decision making process makes use of emotion-related valuing of decision options on the one hand based on predictive loops through feeling states, and on the other hand based on contagion. The resulting collective decision making process is analysed from the perspective of learning speed and rationality. Simulation results and the extent of rationality of the model over time are presented and analysed. It is shown how the collectiveness amplifies both learning speed and rationality of the decisions.

Keywords: collective decision making, adaptive agent model, emotion-related valuing, Hebbian learning, rationality.

1 Introduction

Usually adaptive agents base their decisions on experiences with their decisions made earlier, in relation to the valuation and feeling for the effects of decision options. In this way they are tuned to the environment and become more rational over time with respect to the world characteristics; e.g., [24]. Collective decision making in groups of agents involves such individual decision making processes, but also mutual contagion processes. In recent years some of the mechanisms underlying such social processes have been described in the area of Social Neuroscience (e.g., [9]). Two main concepts in these processes are mirror neurons and internal simulation. Mirror neurons are neurons that are not only active to prepare for a certain action or body change, but also when somebody else who is performing or tending to perform this action or body change is observed (e.g., [15], [21]). Internal simulation is mental processing that copies processes that (may) take place externally. Two different examples of internal simulation are (1) predicting the effects of a prepared action (e.g., [2], [13], [17]), and (2) copying another individual's mental processes (e.g., [6], [7], [11]).

More specifically, in an individual decision making process, before a decision option is chosen, by an internal simulation the expected effects of the option are predicted. For these predicted effects valuations are made, in relation to the affective state associated to this effect (e.g., [1], [6], [8], [16], [18]). To achieve a collective decision, in addition these feelings and valuations for a considered option by different agents affect each other as a form of contagion. By this contagion, a decision option can get a shared positive valuation and feeling, which can be the basis of a common decision. To analyse the effect of the collectiveness on the decision process and its rationality is the focus of the current paper. The decision making within the agent model involving valuing of the decision options by predictive valuation through feeling states, is adopted from [3], [24]; this decision model is based on cognitive and neurological literature such as [6, 8, 14, 15, 16, 17, 18, 19, 20]). The adaptation model used is based on Hebbian learning (cf. [10, 12]).

In this paper, first in Section 2 the multi-agent model is introduced. Section 3 presents the adaptation model based on Hebbian learning. Simulation results are presented in Section 4. Section 5 describes tests of the learning speeds for some of the generated simulation traces and compares this with predictions made on the basis of the mathematical analysis. In Section 6 two measures for rationality used are presented, and the adaptive joint decision making models are evaluated based on these measures. Finally, Section 7 is a discussion. An appendix presents a mathematical analysis addressing equilibria, and the comparison of the learning speed for the cases with and without contagion.

2 The Basic Model for the Agents and their Contagion

This section describes the basic agent model used. Part of this model has been adopted from [24], which addresses especially the single agent case. The contagion mechanism has been added to this, and was adopted from [14].

Decision making based on emotion-related valuing In this part of the model emotional responses triggered by the environment play a role; see Fig. 1 for an overview. More specifically, it is assumed that responses in relation to a sensory representation state of a world stimulus w roughly proceed according to the following causal chain for a *body loop* (based on elements from [4, 7, 8]):

sensory representation → preparation for bodily response → body state modification → sensing body state → sensory representation of body state → induced feeling

In addition, an *as-if body loop* uses a direct causal relation

preparation for bodily response → sensory representation of body state

as a shortcut in the causal chain; cf. [7]. This can be considered a prediction of the action effect by internal simulation (e.g., [13]). The resulting induced feeling provides an emotion-related valuation of this prediction (cf. [1], [6], [8], [16], [18], [20], [22], [23]). If the level of the feeling (which is assumed positive here) is high, a positive valuation is obtained. This valuation has a reinforcing effect on the preparation state. Therefore the body loop (or as-if body loop) is extended to a recursive (as-if) body loop by assuming that the preparation of the bodily response is also affected by the level of the induced feeling:

induced feeling → preparation for the bodily response

Such recursion is also suggested in [8], pp. 91-92. Through this recursive loop a high valuation will strengthen activation of the preparation, which can lead to a high activation level as a basis for a decision for the option.

The role of contagion in a collective decision Within the collective decision making model an additional mechanism for contagion has been incorporated, based on mirroring of the preparation states (adopted from [14]). An important element is the contagion strength γ_{BA} from person B to person A . This indicates the strength by which a preparation state S (for an option b_i) of A is affected by the corresponding preparation state S of B . It depends on characteristics of the two persons: how expressive B is, how open A is, and how strong the connection from B to A is. In the model it is defined by

$$\gamma_{BA} = \varepsilon_B \alpha_{BA} \delta_A$$

Here, ε_B is the *expressiveness* of B , δ_A the *openness* of A , and α_{BA} the *channel strength* from B to A . Note the labels in Fig. 1 for these concepts. The level q_{SA} of preparation state S in agent A (with values in the interval $[0, 1]$) over time is determined as follows. The overall contagion strength γ_A from the rest of the group towards agent A is $\gamma_A = \sum_{B \neq A} \gamma_{BA}$. The aggregated impact q_{SA}^* of all these agents upon state S of agent A is the following weighted average:

$$q_{SA}^*(t) = \sum_{B \neq A} \gamma_{BA} q_{SB}(t) / \gamma_A$$

This is an additional external impact on the preparation state S of A , which has to be combined with the impact from the internal emotion-related valuing process. Note that for the case that there is only one other agent, this expression for $q_{SA}^*(t)$ can be simplified to $q_{SB}(t)$.

Informally described theories in scientific disciplines, for example, in biological or neurological contexts, often are formulated in terms of causal relationships or in terms of dynamical systems. To adequately formalise such a theory the hybrid dynamic modelling language LEADSTO has been developed that subsumes qualitative and quantitative causal relationships, and dynamical systems; cf. [4]. This language has been proven successful in a number of contexts, varying from biochemical processes that make up the dynamics of cell behaviour to neurological and cognitive processes e.g. [4], [5]. Within LEADSTO the *dynamic property* or temporal relation $a \rightarrow_D b$ denotes that when a state property a occurs, then after a certain time delay (which for each relation instance can be specified as any positive real number D), state property b will occur. Below, this D will be taken as the time step Δt . In LEADSTO both logical and numerical calculations can be specified in an integrated manner, and a dedicated software environment is available to support specification and simulation. A formal specification of the model in LEADSTO can be found in current section.

An overview of the multi-agent model is depicted in Fig. 1. This picture also shows representations from the detailed specifications explained below. However, note that the precise numerical relations are not expressed in this picture, but in the detailed specifications below, through local properties LP0 to LP11. Note that the effector state for b_i combined with the (stochastic) effectiveness of executing b_i in the world (indicated by the *world characteristic* or *effectiveness rate* λ_i between 0 and 1) activates the sensor state for b_i via the body loop as described above. By a recursive as-if body loop each of the preparations for b_i generates a level of feeling for b_i which is considered a valuation of the prediction of the action effect by internal simulation. This in turn affects the level of the related action preparation for b_i . Dynamic interaction within these loops results in equilibrium for the strength of the preparation and of the feeling, and depending on these values, the action is actually activated with a certain intensity. The specific strengths of the connections from the sensory representation to the preparations, and within the recursive as-if body loops can be innate, or are acquired during lifetime. The computational model is based on such neurological notions as valuing in relation to feeling, body loop and as-if body loop, as briefly discussed above. In this paper the considered adaptation mechanisms for the model is based on Hebbian learning (Section 3). The detailed specification of the basic model is presented below, starting with how the world state is sensed.

LP0 Sensing a world state

If world state property w occurs of level V
then the sensor state for w will have level V .

world_state(W, V) → sensor_state(W, V)

From the sensor state a sensory representation of the world state is generated by dynamic property LP1.

LP1 Generating a sensory representation for a sensed world state

If the sensor state for world state property w has level V ,
then the sensory representation for w will have level V .

$$\text{sensor_state}(W, V) \rightarrow \text{srs}(W, V)$$

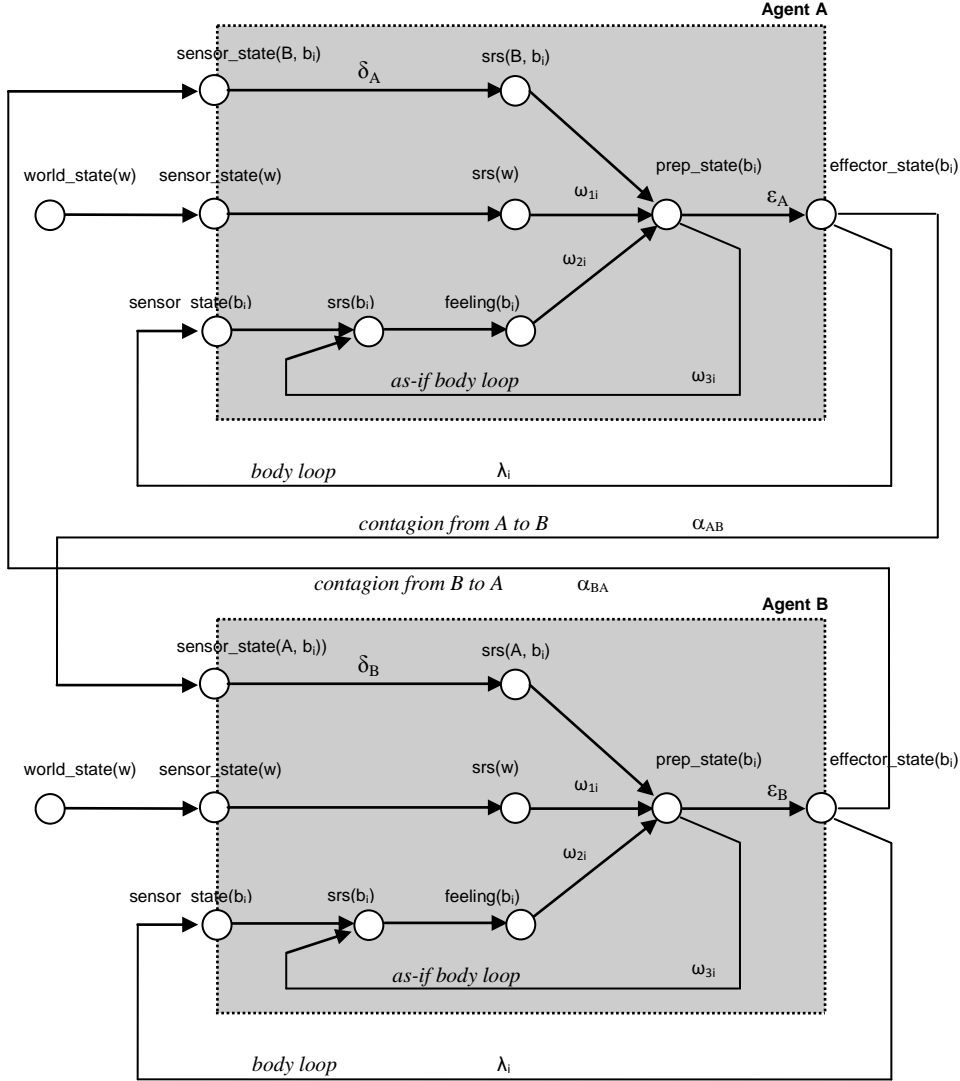


Fig. 1. Overview of the multi-agent model: two agents

The combination function h to combine two inputs which activates a subsequent state is used along with the threshold function th to keep the resultant value in the interval $[0, 1]$ as follows:

$$h(\sigma, \tau, V_1, V_2, \omega_1, \omega_2) = th(\sigma, \tau, \omega_1 V_1 + \omega_2 V_2)$$

where V_1 and V_2 are the current activation level of the states and ω_1 and ω_2 are the connection strength of the link between the states; here

$$th(\sigma, \tau, V) = \left(\frac{1}{1+e^{-\sigma(V-\tau)}} - \frac{1}{1+e^{\sigma\tau}} \right) * (1 + e^{-\sigma\tau})$$

where σ is the steepness and τ is the threshold of the given function. Alternatively for higher value of σ , following threshold function might be used.

$$th(\sigma, \tau, V) = \frac{1}{1+e^{-\sigma(V-\tau)}}$$

Dynamic property LP2 describes the generation of the preparation state from the sensory representation of the world state and the feeling thereby taking into account mutual inhibition. For the case of three agents the combination function is defined as:

$$g(\sigma, \tau, V_1, V_2, V_3, V_4, \omega_1, \omega_2, \theta_{ji}, \theta_{ki}) = th(\sigma, \tau, \omega_1 V_1 + \omega_2 V_2 + \theta_{ji} V_3 + \theta_{ki} V_4)$$

where θ_{mi} is the strength of the inhibition link from preparation state for b_m to preparation state for b_i (which have negative values).

LP2 From sensory representation and feeling to preparation of a body state with mutual inhibition

If a sensory representation for w with level V occurs
 and the feeling associated with body state b_i has level V_i and the preparation state for each b_m has level U_m
 and ω_{1i} is the strength of the connection from sensory representation for w to preparation for b_i
 and ω_{2i} is the strength of the connection from feeling of b_i to preparation for b_i
 and θ_{mi} is for each m the strength of the inhibiting connection from preparation state for b_m to preparation state for b_i
 and σ_i is the steepness value for preparation of b_i and τ_i is the threshold value for preparation of b_i
 and γ_1 is the person's flexibility for bodily responses and $j \neq i, k \neq i, j \neq k$
 then after Δt the preparation state for body state b_i will have level $U_i + \gamma_1 (g(\sigma_i, \tau_i, V, V_i, U_j, U_k, \omega_{1i}, \omega_{2i}, \theta_{ji}, \theta_{ki}) - U_i) \Delta t$

srs(w, V) & feeling(b_i, V_i) & \wedge_m preparation_state(b_m, U_m) &
 has_connection_strength(srs(w), preparation(b_i), ω_{1i}) &
 has_connection_strength(feeling(b_i), preparation(b_i), ω_{2i}) &
 $\wedge_{m \neq i}$ has_connection_strength(preparation_state(b_m), preparation(b_i), θ_{mi}) &
 has_steepness(preparation_state(b_i), σ_i) & has_threshold(preparation_state(b_i), τ_i) & $j \neq i, k \neq i, j \neq k$
 \rightarrow preparation($b_i, U_i + \gamma_1 (g(\sigma_i, \tau_i, V, V_i, U_j, U_k, \omega_{1i}, \omega_{2i}, \theta_{ji}, \theta_{ki}) - U_i) \Delta t$)

Dynamic property LP3 describes the generation of sensory representation of a body state from the respective preparation state and sensory state.

LP3 From preparation and sensor state to sensory representation of a body state

If preparation state for b_i has level X_i and sensor state for b_i has level V_i
 and the sensory representation for b_i has level U_i
 and ω_{3i} is the strength of the connection from preparation state for b_i to sensory representation for b_i
 and σ_i is the steepness value for sensory representation of b_i and τ_i is the threshold value for sensory representation of b_i
 and γ_2 is the person's flexibility for bodily responses
 then after Δt the sensory representation for body state b_i will have
 level $U_i + \gamma_2 (h(\sigma_i, \tau_i, X_i, V_i, \omega_{3i}, I) - U_i) \Delta t$.

prep_state(b_i, X_i) & sensor_state(b_i, V_i) & srs(b_i, U_i) &
 has_connection_strength(preparation_state(b_i), srs(b_i), ω_{3i}) &
 has_steepness(srs(b_i), σ_i) & has_threshold(srs(b_i), τ_i) &
 \rightarrow srs($b_i, U_i + \gamma_2 (h(\sigma_i, \tau_i, X_i, V_i, \omega_{3i}, I) - U_i) \Delta t$)

Dynamic property LP4 describes how the feeling is generated from the sensory representation of the body state.

LP4 From sensory representation of a body state to feeling

If the sensory representation for body state b_i has level V ,
 then b_i will be felt with level V .
 srs(b_i, V) \rightarrow feeling(b_i, V)

LP5 describes how an effector state is generated from respective preparation state, thereby taking into account the expressiveness ε_A .

LP5 From preparation to effector state

If the preparation state for b_i has level V , and ε_A is the expressiveness
 then the effector state for body state b_i will have level $\varepsilon_A V$.
 prep_state(b_i, V) \rightarrow effector_state($b_i, \varepsilon_A V$)

Dynamic property LP6 describes how a sensor state is generated from an effector state.

LP6 From effector state to sensor state of a body state

If the effector state for b_i has level V_i , and λ_i is world characteristics/ recommendation for the option b_i
 then the sensor state for body state b_i will have level $\lambda_i V_i$
 effecor_state(b_i, V_i) &
 has_contribution(effecor_state(b_i), sensor_state(b_i), λ_i) &
 \rightarrow sensor_state($b_i, \lambda_i V_i$)

The following two properties model the contagion mechanism, first in LP7 from effector state of one agent to a sensor state of another agent (taking into account the channel strength), and next the further internal processing in the form of a sensory representation (taking into account the openness δ_A).

LP7 From effector state to sensor state of another agent (contagion)

If the effector state for b_i has level V_i , and α_{BA} is the channel strength
 then the sensor state for body state b_i will have level $\alpha_{BA} V_i$
 effecor_state(b_i, V_i) &
 has_contribution(effecor_state(b_i), sensor_state(b_i), α_{BA}) &
 \rightarrow sensor_state($B, b_i, \alpha_{BA} V_i$)

LP8 Generating a sensory representation for a sensed state of another agent

If the sensor state for state property b_i of agent B has level V ,
 then the sensory representation for state property b_i of agent B will have level $\delta_A V$.
 sensor_state(B, b_i, V) \rightarrow srs($B, b_i, \delta_A V$)

For the case studies addressed three options are assumed available in the world for the agent and the objective is to see how rationally agents make joint decisions using the given adaptation model (under static as well as stochastic world characteristics).

3 An Adaptation Model by Hebbian Learning

From a Hebbian perspective [12], strengthening of a connection over time may take place when both nodes are often active simultaneously ('neurons that fire together wire together'). The principle goes back to Hebb [12], but has recently gained enhanced interest (e.g., [10]). In the adaptive computational model three connections that play a role in decision making can be adapted based on a Hebbian learning principle. More specifically, for such a connection from node i to node j its strength ω_{ij} is adapted using the following *Hebbian learning rule*, taking into account a maximal connection strength 1 , a *learning rate* η , and an *extinction rate* ζ (usually taken small):

$$\frac{d\omega_{ij}(t)}{dt} = \eta a_i(t)a_j(t)(1 - \omega_{ij}(t)) - \zeta\omega_{ij}(t) = \eta a_i(t)a_j(t) - (\eta a_i(t)a_j(t) + \zeta) \omega_{ij}(t)$$

Here $a_i(t)$ and $a_j(t)$ are the activation levels of node i and j at time t and $\omega_{ij}(t)$ is the strength of the connection from node i to node j at time t . A similar Hebbian learning rule can be found in [10], p. 406. By the factor $1 - \omega_{ij}(t)$ the learning rule keeps the level of $\omega_{ij}(t)$ bounded by 1 (which could be replaced by any other positive number); Hebbian learning without such a bound usually provides instability. When the extinction rate is relatively low, the upward changes during learning are proportional to both $a_i(t)$ and $a_j(t)$; maximal learning takes place when both are 1 . Whenever one of $a_i(t)$ and $a_j(t)$ is 0 (or close to 0) extinction takes over, and ω_{ij} slowly decreases. This learning principle has been applied for example to the connection from sensory representation of w to the preparation state for b_i in Fig. 1, according to the following instantiation of the general learning rule above:

$$\frac{d\omega_{1iA}(t)}{dt} = \eta srs(w)(t) prep_state(b_i)(t)(1 - \omega_{1iA}(t)) - \zeta\omega_{1iA}(t)$$

In this section the basic agent model described in Section 2 is extended with a Hebbian learning approach. The connection strength of the different links are updated according to the local properties LP9 to LP11 given below.

LP9 Hebbian learning for connection from sensory representation of stimulus to preparation of b_i

If the connection from sensory representation of w to preparation of b_i has strength ω_{1i} and the sensory representation for w has level V and the preparation of b_i has level V_i and the learning rate from sensory representation of w to preparation of b_i is η and the extinction rate from sensory representation of w to preparation of b_i is ζ

then after Δt the connection from sensory representation of w to preparation of b_i will have strength $\omega_{1i} + (\eta V V_i (1 - \omega_{1i}) - \zeta \omega_{1i}) \Delta t$.
 has_connection_strength(srs(w), preparation(b_i), ω_{1i}) & srs(w, V) & preparation(b_i, V_i) &
 has_learning_rate(srs(w), preparation(b_i), η) & has_extinction_rate(srs(w), preparation(b_i), ζ)
 → has_connection_strength(w, b_i, $\omega_{1i} + (\eta V V_i (1 - \omega_{1i}) - \zeta \omega_{1i}) \Delta t$)

LP10 Hebbian learning for connection from feeling to preparation of b_i

If the connection from feeling b_i to preparation of b_i has strength ω_{2i} and the feeling for b_i has level V_i and the preparation of b_i has level U_i and the learning rate from feeling of b_i to preparation of b_i is η and the extinction rate from feeling of b_i to preparation of b_i is ζ

then after Δt the connection from feeling of b_i to preparation of b_i will have strength $\omega_{2i} + (\eta V_i U_i (1 - \omega_{2i}) - \zeta \omega_{2i}) \Delta t$.
 has_connection_strength(feeling(b_i), preparation(b_i), ω_{2i}) & feeling(b_i, V_i) & preparation(b_i, U_i) & has_learning_rate(feeling(b_i),
 preparation(b_i), η) &
 has_extinction_rate(feeling(b_i), preparation(b_i), ζ)
 → has_connection_strength(feeling(b_i), preparation(b_i), $\omega_{2i} + (\eta V_i U_i (1 - \omega_{2i}) - \zeta \omega_{2i}) \Delta t$)

LP11 Hebbian learning for connection from preparation to sensory representation of b_i

If the connection from preparation of b_i to sensory representation of b_i has strength ω_{3i} and the preparation of b_i has level V_i and the sensory representation of b_i has level U_i and the learning rate from preparation of b_i to sensory representation of b_i is η and the extinction rate from preparation of b_i to sensory representation of b_i is ζ

then after Δt the connection from preparation to sensory representation of b_i will have strength $\omega_{3i} + (\eta V_i U_i (1 - \omega_{3i}) - \zeta \omega_{3i}) \Delta t$.
 has_connection_strength(preparation(b_i), srs(b_i), ω_{3i}) & preparation(b_i, V_i) & srs(b_i, U_i) &
 has_learning_rate(preparation(b_i), srs(b_i), η) & has_extinction_rate(preparation(b_i), srs(b_i), ζ)
 → has_connection_strength(preparation(b_i), srs(b_i), $\omega_{3i} + (\eta V_i U_i (1 - \omega_{3i}) - \zeta \omega_{3i}) \Delta t$)

4 Simulation Results

In this section some of the simulation results, performed using numerical software, are described in detail. Moreover, learning of the connections was done either one at a time ω_{1i} (A), ω_{2i} (B), ω_{3i} (C), or for multiple connections simultaneously (ABC). Due to space limitation only the graphs for (B) where ω_{2i} is learned are shown here. Results for the rationality factors are presented in Section 7. For all simulation results shown in Figs. 2 to 21, time is on the horizontal axis and the vertical axis shows the activation level of the different states. The step size for all simulations is $\Delta t = 1$. Fig. 2 shows simulation results for the model under stochastic world characteristics. To simulate a stochastic world, probability distribution functions (PDF) were defined for the world characteristic parameters λ_i according to a normal distribution. Using these PDFs, the λ_i were generated at random, thereby limiting the values to the interval $[0, 1]$

with modi $\mu_1=0.9$, $\mu_2=0.2$ and $\mu_3=0.1$ for the λ_i , respectively. Furthermore the standard deviation for all λ_i was taken 0.1 . Other parameters were set as follows:

- learning rate $\eta = 0.04$, extinction rate $\zeta = 0.0015$
- fixed values $\omega_{1i} = \omega_{3i} = 0.8$
- speed factors $\gamma_1 = 0.05$, $\gamma_2 = 1$, $\gamma_3 = 0.05$
- steepness $\sigma = 2$, threshold $\tau = 1.2$ for each of the preparation states for b_i
- steepness $\sigma = 10$, threshold $\tau = 0.3$ for each of the sensory representation states of b_i

For the presented results all of the mutual inhibiting connections between the preparation states have strength $\theta_{ij} = -0.2$. Figures 2 and 3 show the effect of inhibition on the output of the model. For the initial 80 time units the stimulus w is kept 1 and for next 170 time units it is kept 0 and the same sequence of activation and deactivation for the stimulus w is repeated for the rest of the simulation. Due to inhibition the differences between the higher and lower values of effector states for different world characteristics λ_i are increased by suppressing the lower ones.

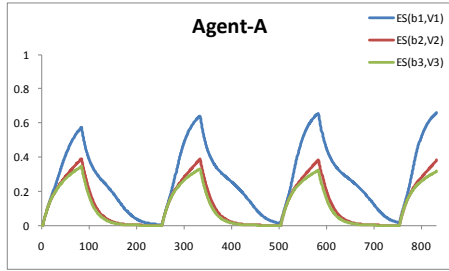


Fig. 2. Model without Inhibition

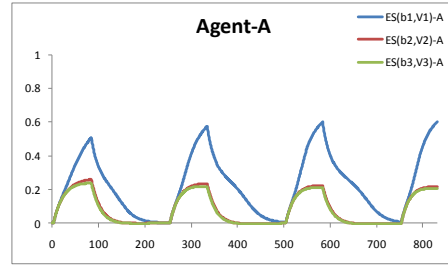


Fig. 3. Model with Inhibition $\theta_{ij} = -0.2$

To study the effect of contagion a number of scenarios were simulated; they are presented one by one with a brief description. In the scenarios only results with inhibition are presented. Note that in all scenarios the two agents deal with the same world, i.e., the world characteristics λ_i are the same.

Scenario 1: The simplest scenario is for two agents having no mutual contagion with the same parameter setting (see Fig. 4 and Fig. 5). For this particular case $\varepsilon_A = \varepsilon_B = \delta_A = \delta_B = 1$, and $\alpha_{AB} = \alpha_{BA} = 0$. So both agents individually go through the same learning process but in total isolation.

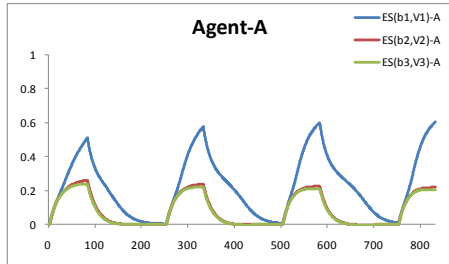


Fig. 4. Decision levels of Agent A

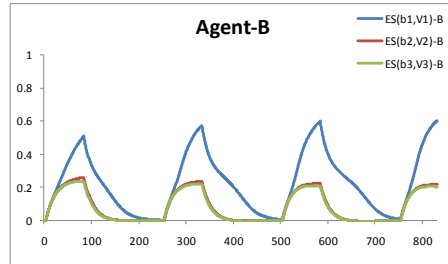


Fig. 5. Decision levels of Agent B

Scenario 2: This is an extension of the first scenario by just including contagion. In this experiment the situation is addressed in which both agents are fully expressive, completely open and the channel strength among them is maximal: $\varepsilon_A = \varepsilon_B = \delta_A = \delta_B = \alpha_{AB} = \alpha_{BA} = 1$. The results show that in comparison to scenario 1, the decisions are amplified; see Fig. 6 and Fig. 7. As both agents have the same characteristics, they show to the same development.

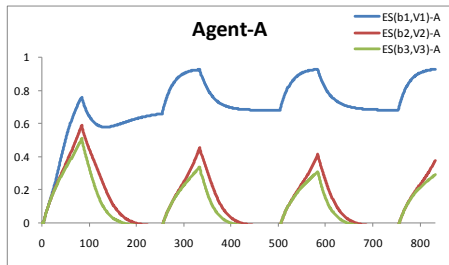


Fig. 6. $\varepsilon_A = \delta_A = \alpha_{AB} = 1$

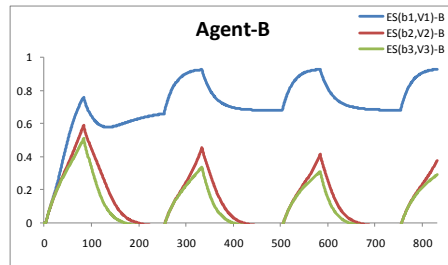


Fig. 7. $\varepsilon_B = \delta_B = \alpha_{BA} = 1$

Scenario 3: The next scenario is a situation in which both agents have a different characteristic: the connection strength between world state and sensory state of the agents is different: 1 for A and 0.8 for B (maybe due to different positions in the world). Moreover for this scenario there is no contagion between them. Due to their difference, and their isolation they develop differently (see Fig. 8 and Fig. 9).

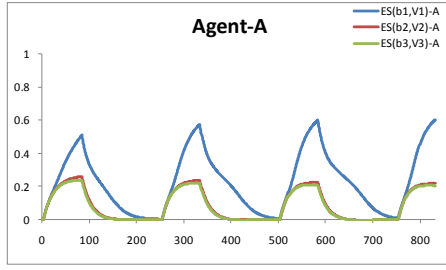


Fig. 8. $\epsilon_A = \delta_A = 1$, $\alpha_{AB} = 0$, $\omega = 1$

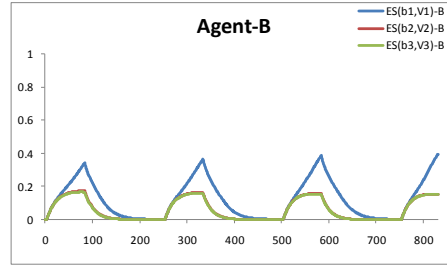


Fig. 9. $\epsilon_B = \delta_B = 1$, $\alpha_{BA} = 0$, $\omega = 0.8$

Scenario 4: This scenario adds contagion to scenario 3. It can be observed that these agents start with different decision levels, but over the time a consensus is developed and the differences become practically 0; see Fig. 10 and Fig. 11.

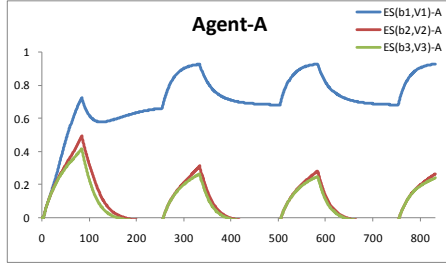


Fig. 10. $\epsilon_A = \delta_A = \alpha_{AB} = 1$, $\omega = 1$

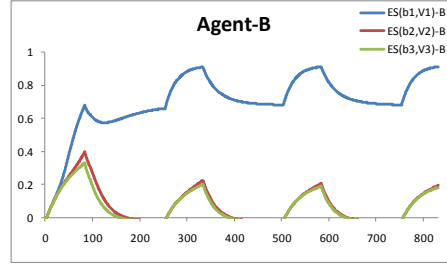


Fig. 11. $\epsilon_B = \delta_B = \alpha_{BA} = 1$, $\omega = 0.8$

Scenario 5: In this scenario the effect of the channel strength is studied between the agents having differences in decision levels initially. To simulate this situation α_{AB} is taken 0.5 and α_{BA} is taken 0.4, which means the decisions of agent A would have more impact on B than conversely. This is indeed reflected in Fig. 12 and Fig. 13.

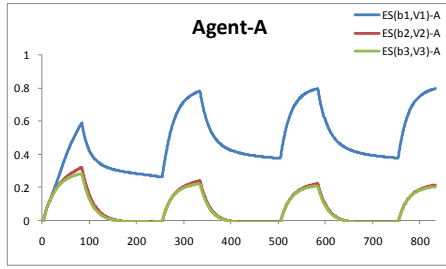


Fig. 12. $\epsilon_A = \delta_A = 1$, $\alpha_{AB} = 0.5$, $\omega = 1$

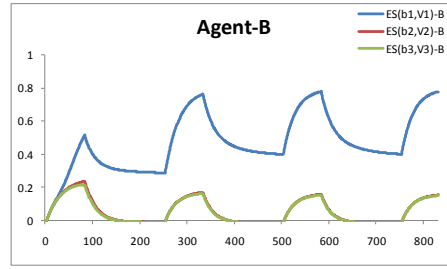


Fig. 13. $\epsilon_B = \delta_B = 1$, $\alpha_{BA} = 0.4$, $\omega = 0.8$

Scenario 6: In this scenario the channel strengths which are used in previous scenario are swapped. The objective of this swapping was to see what happened if an agent has a lower decision value but has a strong channel strength to the other agent having a higher value. It is observed that to some extent the final decision value is relatively lower than in the previous scenario: the person with lower value has more influence on the collective decision; see Fig. 14 and Fig. 15.

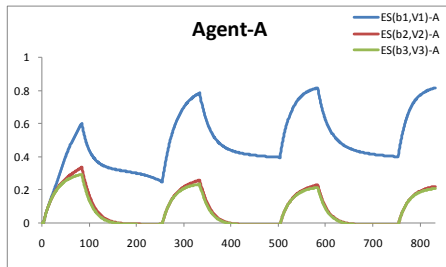


Fig. 14. $\epsilon_A = \delta_A = 1$, $\alpha_{AB} = 0.4$, $\omega = 1$

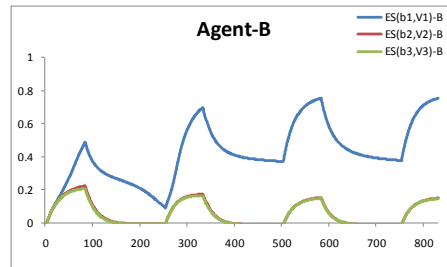


Fig. 15. $\epsilon_B = \delta_B = 1$, $\alpha_{BA} = 0.5$, $\omega = 0.8$

Scenario 7: Fig. 16 and Fig. 17 show the results of a scenario where agent A is assumed less expressive ($\epsilon_A = 0.5$) and more open ($\delta_A = 1$). Furthermore, agent B is more expressive ($\epsilon_B = 1$) and less open ($\delta_B = 0.5$). According to these parameter settings, agent A has influence from agent B but A itself is not expressive enough to amplify its own decision values. Hence its main decision value remains lower than agent B by almost 50%, whereas B shows higher values as they are amplified due to the higher expressiveness (see Fig. 16 and Fig. 17)

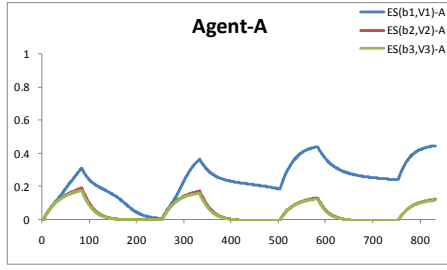


Fig. 16. $\varepsilon_A=0.5$, $\delta_A=1$, $\alpha_{AB}=1$, $\omega=1$

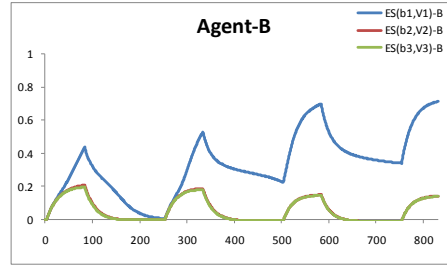


Fig. 17. $\varepsilon_B=1$, $\delta_B=0.5$, $\alpha_{BA}=1$, $\omega=0.8$

Scenario 8: The last scenario presented here displays the situation in which the expressiveness for both agents are same i.e. $\varepsilon_A = \varepsilon_B = 1$ but they have different channel strengths and different openness: $\delta_A = 0.2$, $\alpha_{BA} = 0.6$ and $\delta_B = 0.6$, $\alpha_{AB} = 0.4$. With these settings one observes better consensus among two agents even though their openness is different and they have different channel strength; this is because a weaker connection is compensated by a higher openness and vice versa; see Fig. 18 and Fig. 19.

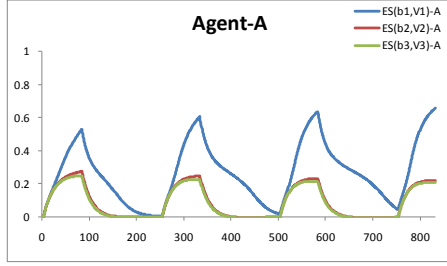


Fig. 18. $\varepsilon_A=1$, $\delta_A=0.2$, $\alpha_{AB}=0.4$, $\omega=1$

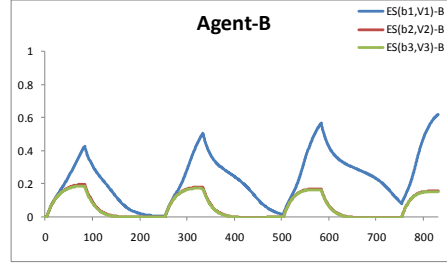


Fig. 19. $\varepsilon_B=1$, $\delta_B=0.6$, $\alpha_{BA}=0.6$, $\omega=0.8$

The results presented in Fig. 20 and Fig. 21 are obtained from the simulation results for scenario 5. The values for the parameters are $\varepsilon_A = \delta_A = 1$, $\alpha_{AB} = 0.5$, $\omega = 1$ and $\varepsilon_B = \delta_B = 1$, $\alpha_{BA} = 0.5$, $\omega = 0.8$ whereas the parameter setting without contagion are $\varepsilon_A = \delta_A = 1$, $\alpha_{AB} = 0$, $\omega = 1$ and $\varepsilon_B = \delta_B = 1$, $\alpha_{BA} = 0$, $\omega = 0.8$. It can be observed for the given results that due to contagion a consensus can evolve between agents having different initial decision values about some specific situation.

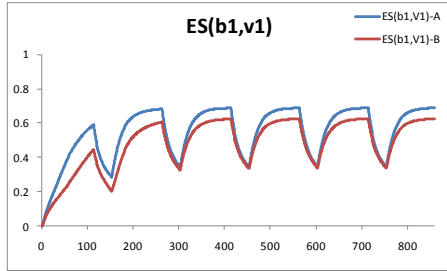


Fig. 20. Without Contagion

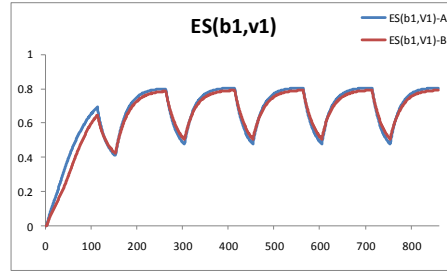


Fig. 21. With Contagion

5 Analysis Based on Simulation Results

As an additional test for the difference in learning speed for the cases with and without contagion, and of the correctness of the mathematical analysis presented above, the current section conducts an analysis based on the simulation results shown in Section 4. In particular, the focus is on the estimated learning speed of ω_{2iA} , and on the ratio between the ω_{2iA} for the case with and without contagion.

In Figure 22, the behaviour of ω_{2iA} is shown for scenario 1 (depicted in Fig. 4 and 5) and scenario 2 (depicted in Fig. 6 and 7)¹. Recall from Section 4 that the parameter settings of these two scenarios are identical, except for the fact that no contagion ($\gamma_{AB} = \gamma_{BA} = 0$) takes place in scenario 1 and full contagion ($\gamma_{AB} = \gamma_{BA} = 1$) takes place in scenario 2. As can be observed in Fig. 22, one of the effects of the full contagion is that (after the first 30 time points, in which the model needs to settle) the learning speed of that scenario is immediately much higher than that of the scenario without contagion (as seen from the steeper curve in the most relevant time period between time points 30 and 80). After time point 80, the learning speed cannot increase much for the scenario with contagion (since it is already close to 1), whereas for the scenario without contagion it gradually increases during the periods when the stimulus w is 1.

This learning speed of ω_{2iA} is visualised explicitly in Figure 23 (for scenario 1) and 24 (for scenario 2), by means of the red lines. In addition, the blue lines show how closely these curves are approximated by the approximation presented at the end of Section 5. In order to generate these estimations, σ_2 was set to 0.45 for scenario 1, and to 0.35 for scenario 2. For both scenarios, σ_7 was set to 0.6. As shown in the figures, the approximation of the speed factor is

¹ As in Section 5, the focus is on the highest option. Therefore, ω_{2iA} should be read as ω_{21A} here.

generally quite accurate for both cases (with average errors of 0.00126 and 0.00134 , respectively), except for the fact that the transitions between the periods with and without stimulus are more abrupt.

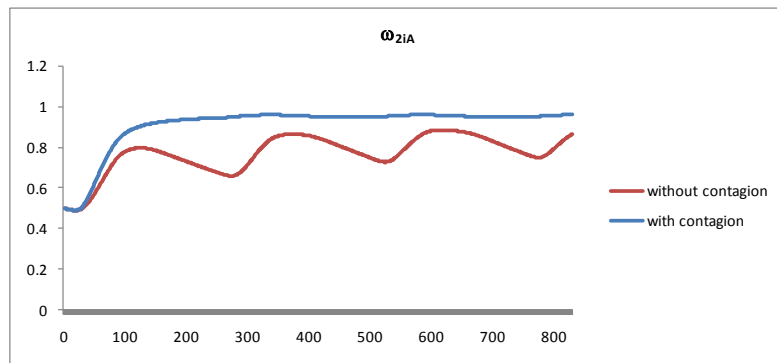


Fig. 22. Change of ω_{2iA} during scenario 1 (without contagion) and 2 (with contagion)

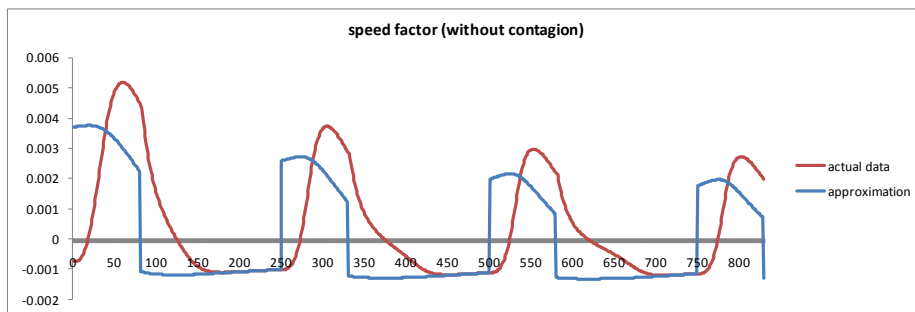


Fig. 23. Change of speed factor of ω_{2iA} during scenario 1 (without contagion)

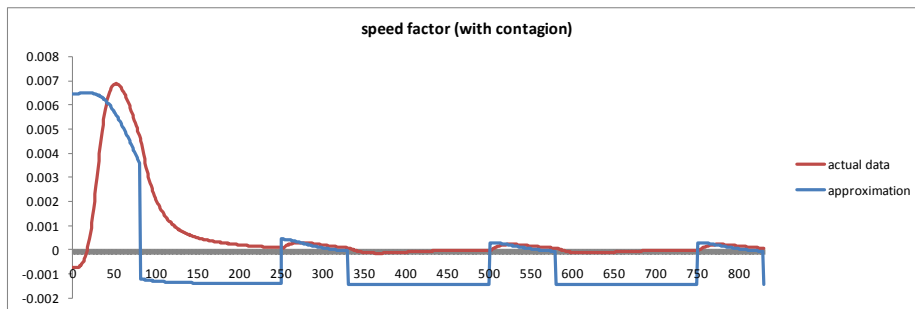


Fig. 24. Change of speed factor of ω_{2iA} during scenario 2 (with contagion)

Finally, also the ratio between the speed factors of ω_{2iA} without and with contagion was calculated using the approach presented in Section 5. Some (partial) results of this are shown in Figure 25.

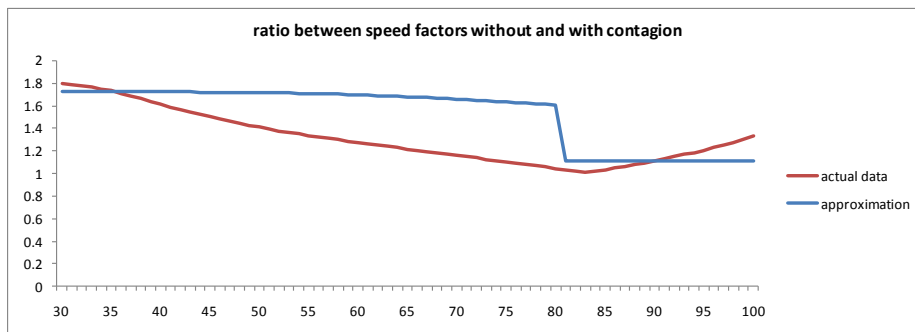


Fig. 25. Ratio between speed factors of ω_{2iA} in scenario 1 (without) and 2 (with contagion)

Note that this figure only shows a fragment of the scenario which lasts from time point 30 to 100. The reason for this is that after this period, there are a number of instances where the speed factor without contagion becomes 0, which leads to unreliable results (because of division by 0). Nevertheless, Figure 25 clearly shows that the estimated ratio between the speed factors comes close (but again, with more abrupt transitions) to the actual ratio for these scenarios

(average error 0.257). This provides additional evidence that the approximations of the ratio between learning speed with and without contagion (as presented at the end of Section 5) are accurate. So, both the approximation from Section 5 and the analysis of simulation results here show that in the most relevant time period the ratio between learning speed with contagion and without contagion is from about 1.7 to about 1.2. This shows the amplifying effect of the contagion.

6 Evaluating the Models on Rationality

In the simulation experiments it was shown that the agent model behaves rationally in different scenarios. The results of these scenarios were assessed with respect to the extent of their rationality only in a rather informal manner. In the current section the rationality is determined more formally by two methods developed earlier: by one rationality measure based on a discrete scale and another one based on a continuous scale.

Method 1 (Discrete Rationality Measure)

The first method presented is based on the following point of departure: *an agent which has the same respective order of effector state activation levels for the different options compared to the order of world characteristics λ_i will be considered highly rational*. More specifically, the following formula is used to determine the irrationality factor IF .

$$IF = \sum_{i=1}^n |\text{rank}(es_i) - \text{rank}(\lambda_i)|$$

where n is the number of options available. To calculate the discrete rationality factor DRF , the maximum possible irrationality factor $MaxIF$ can be determined as follows.

$$MaxIF = \frac{n(n+1)}{2} - \text{ceiling}\left(\frac{n}{2}\right)$$

Here $\text{ceiling}(x)$ is the first integer higher than x . Note that $MaxIF$ is approximately $\frac{1}{2}n^2$. As a higher IF means lower rationality, the discrete rationality factor DRF is calculated as:

$$DRF = 1 - \frac{IF}{MaxIF}$$

Method 2 (Continuous Rationality Measure)

The second method presented is based on the following point of departure: *an agent which receives high benefit will be a highly rational agent*. In this method to calculate the continuous rationality factor CRF , to account for the effort spent in performing actions, the effector state values ES_i are normalised as follows.

$$nES_i = \frac{ES_i}{\sum_{i=1}^n ES_i}$$

with n is the number of options available. Based on this, the continuous rationality factor CRF is determined as follows, with $Max(\lambda_i)$ the maximal value of the different λ_i .

$$CRF = \frac{\sum_{i=1}^n nES_i \lambda_i}{Max(\lambda_i)}$$

This method enables to measure to which extent the agent is behaving rationally in a continuous manner.

A number of simulations were carried out to study the rationality for the different scenarios: with contagion and without contagion. Due to space limitation only the results for scenario 5 is given here. Fig. 26 and Fig 27 present the results for agent A, for both measures of rationality.

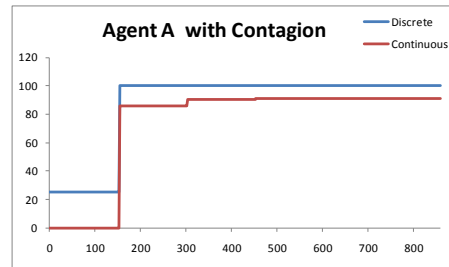
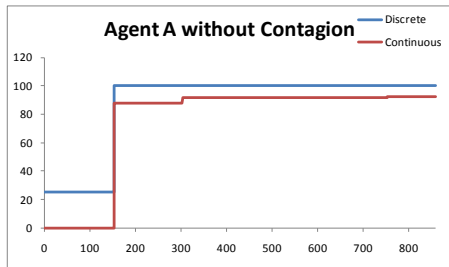


Fig. 26. A's rationality over time: no contagion Fig. 27. A's rationality over time: contagion

Fig. 28 and Fig. 29 show the results for agent B. It is clear from the given results that even though there is no significance improvement of the rationality over time for agent A due to contagion, for agent B rationality improved significantly due to contagion. Thus one can say that consensus is developed with improved rationality in collective decision making (see Fig. 28 and Fig. 29).

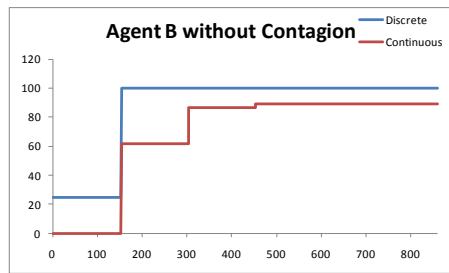


Fig. 28. B's rationality over time: no contagion

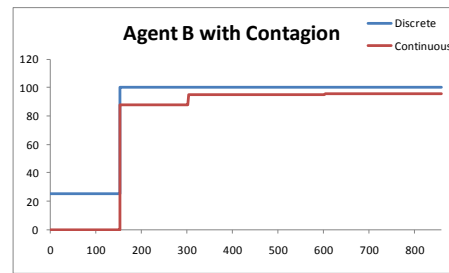


Fig. 29. B's rationality over time: contagion

7 Discussion

The presented adaptive collective decision model is based on interacting adaptive agents that learn from their experiences by a Hebbian learning mechanism (cf. [10], [12]). Within each agent, the decision making process makes use of (1) emotion-related valuing of decision options by internal simulation, and (2) social contagion processes. The internal simulations are based on predictive loops through feeling states, inspired by literature on the neurological basis of valuing; e.g., [1], [6], [8], [16], [18], [20], [22], [23].

The resulting collective decision making process was analysed on learning speed, and on rationality of the collective decisions after a major world change. This was done by a mathematical analysis providing approximated learning speeds, and based on analysis of simulation results. By both types of analysis it was shown how the contagion amplifies the learning speed, and therefore strengthens the rationality of the decision making, in particular after changes of the world characteristics.

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Appendix: Mathematical Analysis

In this section a mathematical analysis is made of the equilibria and learning speed of the adaptive model, and in particular how for the multi-agent case the learning speed depends on the extent of contagion between the agents. For the sake of simplicity the analysis is done for two agents, and the focus is on the highest option. First an approximation of the states involved is made.

Approximation of internal states

The following contributions to preparation state p_A are used as an approximation for the impacts of other states on p_A :

$\omega_{1iA} s$	impact from $srs(w)$ via the connection strength ω_{1iA}
$\omega_{2iA} f_A$	impact through feeling state f_A
$\gamma_{BA} p_B$	impact from preparation state of B via the contagion from B

Note that here the mutual inhibition is neglected, as it is assumed that i is the highest option and suppresses the other options $j \neq i$ so that they are low and do not provide much suppression of the option i . Furthermore, the following contributions to f_A are used as an approximation for these impacts of other states on f_A :

$\omega_{3iA} p_A$	impact through the as-if body loop
$\lambda_i \varepsilon_A p_A$	impact through the body loop

Moreover, it is assumed that the value combined from these two impacts directly affects f_A in a linear form (approximating the threshold function) with steepness σ_1 (e.g., set on 0.6):

$$f_A = \sigma_1 [\omega_{3iA} p_A + \lambda_i \varepsilon_A p_A] = \sigma_1 [\omega_{3iA} + \lambda_i \varepsilon_A] p_A \quad (1)$$

Note that this type of linear approximation is more accurate when the steepness of the approximated threshold function is relatively low.

Similarly, it is assumed that the value combined from the three impacts on p_A directly affects p_A in a linear form with steepness σ_2 (e.g., around 0.4):

$$\begin{aligned} p_A &= \sigma_2 [\omega_{1iA} s + \omega_{2iA} f_A + \gamma_{BA} p_B] \\ &= \sigma_2 [\omega_{1iA} s + \omega_{2iA} \sigma_1 [\omega_{3iA} + \lambda_i \varepsilon_A] p_A + \gamma_{BA} p_B] \end{aligned}$$

Note that the parameters σ_i also depend on the maximal range of the expected incoming combined values on which the threshold function is applied. Therefore for σ_2 slightly different values can be used for cases without contagion (maximal range [0, 2] based on feeling and stimulus as input; e.g., $\sigma_2 = 0.45$), and cases with contagion (maximal range [0, 3] based on feeling, stimulus and contagion as input; e.g., $\sigma_2 = 0.35$).

The above expression for p_A can be rewritten as follows:

$$p_A = \alpha_A [\omega_{1iA} s + \gamma_{BA} p_B]$$

where

$$\alpha_A = \sigma_2 / [1 - \sigma_1 \sigma_2 \omega_{2iA} [\omega_{3iA} + \lambda_i \varepsilon_A]]$$

A similar equation can be obtained for the other agent's preparation:

$$p_B = \alpha_B [\omega_{1iB} s + \gamma_{AB} p_A]$$

These two linear equations in p_A and p_B can be solved, providing an expression of p_A in terms of the connection strengths:

$$p_A = \mu_{AB} s [\omega_{1iA} + \alpha_B \gamma_{BA} \omega_{1iB}] \quad (2)$$

where

$$\mu_{AB} = \alpha_A / [1 - \alpha_A \alpha_B \gamma_{AB} \gamma_{BA}]$$

Note that when $\gamma_{BA} = 0$, it holds $\mu_{AB} = \alpha_A$. Similarly the other preparation state p_B can be expressed in terms of the connection strengths:

$$p_B = \mu_{BA} s [\omega_{1iB} + \alpha_A \gamma_{AB} \omega_{1iA}] \quad (3)$$

Estimation of equilibria: ω_{1iA}

Given the estimations for the states obtained above, the equilibria can be analysed. For example, from the differential equation

$$\frac{d\omega_{1iA}(t)}{dt} = \eta s p_{iA}(t) (1 - \omega_{1iA}(t)) - \zeta \omega_{1iA}(t)$$

and (2) above it follows that an equilibrium for ω_{1iA} has to satisfy

$$\eta s p_{iA} (1 - \omega_{1iA}) = \zeta \omega_{1iA}$$

$$\eta s \mu_{AB} s [\omega_{1iA} + \alpha_B \gamma_{BA} \omega_{1iB}] (1 - \omega_{1iA}) = \zeta \omega_{1iA}$$

In particular, for the symmetric case where the two agents have exactly the same parameter values, this can be simplified further, assuming $\omega_{1iB} = \omega_{1iA} \neq 0$, and $\mu_{AB} = \mu_{BA}$ $\alpha_B = \alpha_A$, $\gamma_{AB} = \gamma_{BA}$:

$$\eta s \mu_{AB} s [\omega_{1iA} + \alpha_B \gamma_{BA} \omega_{1iA}] (1 - \omega_{1iA}) = \zeta \omega_{1iA}$$

$$\eta s \mu_{AB} s [1 + \alpha_B \gamma_{BA}] (1 - \omega_{1iA}) = \zeta$$

$$1 - \omega_{1iA} = \zeta / \eta s \mu_{AB} s [1 + \alpha_B \gamma_{BA}]$$

$$\omega_{1iA} = 1 - \zeta / \eta s \mu_{AB} s [1 + \alpha_B \gamma_{BA}]$$

$$\omega_{1iA} = 1 - (\zeta / \eta) [1 - \alpha_A \alpha_B \gamma_{AB} \gamma_{BA}] / \alpha_A s^2 [1 + \alpha_B \gamma_{BA}]$$

$$\omega_{1iA} = 1 - (\zeta / \eta) [1 - \alpha_A^2 \gamma_{BA}^2] / \alpha_A s^2 [1 + \alpha_A \gamma_{BA}]$$

$$\omega_{1iA} = 1 - (\zeta / \eta) [1 - \alpha_A \gamma_{BA}] / \alpha_A s^2$$

$$\omega_{1iA} = 1 - (\zeta / \eta) [(1/\alpha_A) - \gamma_{BA}] / s^2$$

$$\omega_{1iA} = 1 - (\zeta / \eta) [(1/\sigma_2) - \sigma_1 \omega_{2iA} [\omega_{3iA} + \lambda_i \varepsilon_A] - \gamma_{BA}] / s^2$$

When values for the parameters and for ω_{2iA} and ω_{3iA} are given, this can be used to find an estimation for the equilibrium value for ω_{1iA} . Note that in general this will be close to but just below 1, for example, between $1 - (\zeta / \eta)$ and 1. Furthermore, notice that this value is higher when the contagion strength γ_{BA} is higher. For example, this can be found by differentiating the above expression to γ_{BA} :

$$\frac{\partial \omega_{1iA}}{\partial \gamma_{BA}} = \zeta / \eta s^2$$

This shows that the equilibrium value proportionally increases with γ_{BA} . This can also be found in a more global manner: when $\gamma_{BA} = 0$, then

$$\omega_{1iA} = 1 - (\zeta / \eta) [(1/\sigma_2) - \sigma_1 \omega_{2iA} [\omega_{3iA} + \lambda_i \varepsilon_A]] / s^2$$

which is lower than the general expression above by

$$(\zeta / \eta) \gamma_{BA} / s^2$$

and this is proportional to γ_{BA} .

Estimation of equilibria: ω_{2iA}

For this case from the differential equation

$$\frac{d\omega_{2iA}(t)}{dt} = \eta f_{iA}(t) p_{iA}(t) (1 - \omega_{2iA}(t)) - \zeta \omega_{2iA}(t)$$

it follows that an equilibrium for ω_{2iA} has to satisfy

$$\eta f_{iA} p_{iA} (1 - \omega_{2iA}) = \zeta \omega_{2iA}$$

Using the estimations (1) and (2) for the states obtained above, this can be rewritten as:

$$\eta \sigma_1 [\omega_{3iA} + \lambda_i \varepsilon_A] p_{iA} p_{iA} (1 - \omega_{2iA}) = \zeta \omega_{2iA}$$

$$\eta \sigma_1 [\omega_{3iA} + \lambda_i \varepsilon_A] \mu_{AB}^2 s^2 [\omega_{1iA} + \alpha_B \gamma_{BA} \omega_{1iB}]^2 (1 - \omega_{2iA}) = \zeta \omega_{2iA}$$

For the case that $\gamma_{BA} = 0$, this expression is:

$$\eta \sigma_1 [\omega_{3iA} + \lambda_i \varepsilon_A] \alpha_A^2 s^2 \omega_{1iA}^2 (1 - \omega_{2iA}) = \zeta \omega_{2iA}$$

Again, for the symmetric case where the two agents have exactly the same parameter values, this can be simplified further, assuming $\omega_{1iB} = \omega_{1iA} \neq 0$, and $\mu_{AB} = \mu_{BA}$ $\alpha_B = \alpha_A$, $\gamma_{AB} = \gamma_{BA}$:

$$\eta \sigma_I [\omega_{3iA} + \lambda_i \varepsilon_A] [\mu_{AB}^2 s^2 [\omega_{1iA} + \alpha_B \gamma_{BA} \omega_{1iA}]^2 (1 - \omega_{2iA}) = \zeta \omega_{2iA}$$

Also this expression can be differentiated to γ_{BA} to find out how the equilibrium value for ω_{2iA} depends on γ_{BA} , but this is a bit more complex than in the previous case, as also α_B and μ_{AB} depend on γ_{BA} .

The learning speed ratio for ω_{1iA}

This case focuses on the learning speed of the connection ω_{1i} from the sensory representation state $srs(w)$ to the preparation state $prep_state(b)$. The point of departure is the differential equation for the Hebbian learning, which expresses how the learning speed depends on activation levels and connection strength:

$$\frac{d\omega_{1iA}(t)}{dt} = \eta s p_A(t) (1 - \omega_{1iA}(t)) - \zeta \omega_{1iA}(t)$$

Here $s = srs(w)(t)$ which is assumed constant s (e.g., 1), and $p_A(t)$ can be estimated by the expression (2) found above; this can be filled in the differential equation, thus obtaining

$$\frac{d\omega_{1iA}(t)}{dt} = \eta \mu_{AB} s^2 [\omega_{1iA}(t) + \alpha_B \gamma_{BA} \omega_{1iB}(t)] (1 - \omega_{1iA}(t)) - \zeta \omega_{1iA}(t)$$

Note that the single agent case (without contagion) is obtained from this by putting $\gamma_{BA} = 0$, in which case $\mu_{AB} = \alpha_A$:

$$\frac{d\omega_{1iA}(t)}{dt} = \eta \alpha_A s^2 \omega_{1iA}(t) (1 - \omega_{1iA}(t)) - \zeta \omega_{1iA}(t)$$

Now the estimated comparison of the learning speed between multi-agent and single agent case can be made by considering the ratio of the two expressions:

$$\text{learning speed ratio for } \omega_{1iA}(t) = \frac{\eta \mu_{AB} s^2 [\omega_{1iA}(t) + \alpha_B \gamma_{BA} \omega_{1iB}(t)] (1 - \omega_{1iA}(t)) - \zeta \omega_{1iA}(t)}{\eta \alpha_A s^2 \omega_{1iA}(t) (1 - \omega_{1iA}(t)) - \zeta \omega_{1iA}(t)}$$

In a slightly different form (dividing by $\omega_{1iA}(t)$) this can be written as:

$$\text{learning speed ratio for } \omega_{1iA}(t) = \frac{\eta \mu_{AB} s^2 [1 + \alpha_B \gamma_{BA} \frac{\omega_{1iB}(t)}{\omega_{1iA}(t)}] (1 - \omega_{1iA}(t)) - \zeta}{\eta \alpha_A s^2 (1 - \omega_{1iA}(t)) - \zeta}$$

Note that from this it can be seen that the higher the ratio $\omega_{1iB}(t)/\omega_{1iA}(t)$, the higher the learning speed ratio for agent A. Some values for the learning speeds for given values of parameters and of $\omega_{1iA}(t)$ and the ratio for agent A are listed in Table 1 (for $s = 1$). Note that for the ratio the learning speeds for the single agent and multi-agent (contagion) case are considered assuming the same values for the connection strengths. In an ongoing process, this can occur in an initial phase as illustrated in Section 6, but as one of them changes with a higher speed than the other one, after this initial phase different values for the connection strengths will occur.

Table 1. Estimated learning speeds for ω_{1iA} for a number of parameter settings and values for ω_{1iA}

parameter values															speed	speed	ratio
η	σ_1	σ_2	λ_i	ε_i	ω_{2i}	ω_{3i}	α_A	α_B	γ_{BA}	γ_{AB}	μ_{AB}	ζ	ω_{1iA}	ω_{1iB}	multi	single	
0.04	0.6	0.4	0.8	1	1	1	0.704	0.704	1	1	1.40	0.0015	0.1	0.1	0.0084	0.0024	3.53
0.04	0.6	0.4	0.8	1	1	1	0.704	0.704	1	1	1.40	0.0015	0.5	0.5	0.0231	0.0063	3.66
0.04	0.6	0.4	0.8	1	1	1	0.704	0.704	1	1	1.40	0.0015	0.9	0.9	0.0072	0.0012	6.09
0.04	0.6	0.4	0.8	1	1	1	0.704	0.704	1	1	1.40	0.0015	0.1	0.5	0.0226	0.0024	9.47
0.04	0.6	0.4	0.8	1	1	1	0.704	0.704	1	1	1.40	0.0015	0.1	0.9	0.0368	0.0024	15.41
0.04	0.6	0.4	0.8	1	1	1	0.704	0.704	1	1	1.40	0.0015	0.5	0.9	0.0309	0.0063	4.92
0.04	0.6	0.4	0.8	1	1	1	0.704	0.704	1	1	1.40	0.0015	0.9	0.1	0.0041	0.0012	3.44
0.04	0.6	0.4	0.8	1	1	1	0.704	0.704	0.5	0.5	0.80	0.0015	0.5	0.5	0.0101	0.0063	1.61

It turns out that when the learned connection strengths $\omega_{1iA}(t)$ and $\omega_{1iB}(t)$ are equal in both agents, then under full contagion ($\gamma_{AB} = \gamma_{BA} = 1$) the learning with contagion is estimated to be about 3 to 6 times faster than without contagion. Moreover, the ratio can be still higher (up to a factor 15 or more) for agent A when the learned connection strength $\omega_{1iB}(t)$ for agent B is higher than the $\omega_{1iA}(t)$ for agent A. When no full contagion takes place (e.g., $\gamma_{AB} = \gamma_{BA} = 0.5$), then the learning speed ratio is estimated proportionally lower.

Neglecting extinction (assuming small ζ), by putting $\zeta = 0$ the expression for learning speed can be simplified to:

$$\frac{d\omega_{1iA}(t)}{dt} = \eta \mu_{AB} s^2 [\omega_{1iA}(t) + \alpha_B \gamma_{BA} \omega_{1iB}(t)] (1 - \omega_{1iA}(t))$$

In this case the learning speed ratio can be simplified:

$$\text{learning speed ratio for } \omega_{1iA}(t) \text{ without extinction} =$$

$$\begin{aligned} & \frac{\eta \mu_{AB} s^2 [1 + \alpha_B \gamma_{BA} (\frac{\omega_{1iB}(t)}{\omega_{1iA}(t)})] (1 - \omega_{1iA}(t))}{\eta \alpha_A s^2 (1 - \omega_{1iA}(t))} \\ &= \frac{(\mu_{AB})}{\alpha_A} [1 + \alpha_B \gamma_{BA} (\frac{\omega_{1iB}(t)}{\omega_{1iA}(t)})] \\ &= \frac{1}{1 - \alpha_A \alpha_B \gamma_{AB} \gamma_{BA}} [1 + \alpha_B \gamma_{BA} (\frac{\omega_{1iB}(t)}{\omega_{1iA}(t)})] \end{aligned}$$

From this it can easily be seen that the estimated speed ratio is higher when γ_{BA} is higher, or when $\omega_{1iB}(t)/\omega_{1iA}(t)$ is higher, and is 1 when $\gamma_{BA} = 0$. For relatively small γ_{BA} a first order approximation can be made which shows how the speed ratio linearly depends on the incoming contagion factor γ_{BA} with coefficient $\alpha_B [\alpha_A \gamma_{AB} + \frac{\omega_{1iB}(t)}{\omega_{1iA}(t)}]$ as follows:

$$\begin{aligned} & \text{learning speed ratio for } \omega_{1iA}(t) \text{ without extinction} = \\ &= [1 + \alpha_B \gamma_{BA} (\frac{\omega_{1iB}(t)}{\omega_{1iA}(t)})] [1 + \alpha_A \alpha_B \gamma_{AB} \gamma_{BA}] \\ &= 1 + \alpha_B [\alpha_A \gamma_{AB} + \frac{\omega_{1iB}(t)}{\omega_{1iA}(t)}] \gamma_{BA} \end{aligned}$$

The learning speed ratio for ω_{2iA}

This case focuses on the connection ω_{2iA} from the feeling state f_{iA} to the preparation state p_{iA} . From the differential equation

$$\frac{d\omega_{2iA}(t)}{dt} = \eta f_{iA}(t) p_{iA}(t) (1 - \omega_{2iA}(t)) - \zeta \omega_{2iA}(t)$$

an estimation of the learning speed can be obtained by filling the earlier found expressions (1) and (2) for f_{iA} and p_{iA} :

$$\begin{aligned} \frac{d\omega_{2iA}(t)}{dt} &= \eta f_{iA}(t) p_{iA}(t) (1 - \omega_{2iA}(t)) - \zeta \omega_{2iA}(t) \\ &= \eta \sigma_I [\omega_{3iA} + \lambda_i \varepsilon_A] [\mu_{AB} s [\omega_{1iA} + \alpha_B \gamma_{BA} \omega_{1iB}]]^2 (1 - \omega_{2iA}(t)) - \zeta \omega_{2iA}(t) \end{aligned}$$

Note that the single agent case (without contagion) is obtained from this by putting $\gamma_{BA} = 0$, in which case $\mu_{AB} = \alpha_A$:

$$\frac{d\omega_{1iA}(t)}{dt} = \eta \sigma_I [\omega_{3iA} + \lambda_i \varepsilon_A] [\alpha_A s \omega_{1iA}]^2 (1 - \omega_{2iA}(t)) - \zeta \omega_{2iA}(t)$$

Now the estimated comparison of the learning speed between the multi-agent and single agent case can be made by considering the ratio of the two expressions:

$$\begin{aligned} & \text{learning speed ratio for } \omega_{2iA}(t) = \\ & \frac{\eta \sigma_I [\omega_{3iA} + \lambda_i \varepsilon_A] [\mu_{AB} s [\omega_{1iA} + \alpha_B \gamma_{BA} \omega_{1iB}]]^2 (1 - \omega_{2iA}(t)) - \zeta \omega_{2iA}(t)}{\eta \sigma_I [\omega_{3iA} + \lambda_i \varepsilon_A] [\alpha_A s \omega_{1iA}]^2 (1 - \omega_{2iA}(t)) - \zeta \omega_{2iA}(t)} \end{aligned}$$

For a number of parameter settings the values of this ratio are shown in Table 2 (for $s = 1$).

Table 2. Estimated learning speeds for ω_{2iA} for a number of parameter settings

parameter values															speed	speed	ratio
η	σ_1	σ_2	λ_i	ε_i	ω_{1i}	ω_{3i}	α_A	α_B	γ_{BA}	γ_{AB}	μ_{AB}	ζ	ω_{2iA}	ω_{2iB}	multi	single	
0.04	0.6	0.4	0.8	1	1	1	0.418	0.418	1	1	0.51	0.0015	0.1	0.1	0.0199	0.0066	3.00
0.04	0.6	0.4	0.8	1	1	1	0.510	0.510	1	1	0.69	0.0015	0.5	0.5	0.0227	0.0049	4.66
0.04	0.6	0.4	0.8	1	1	1	0.654	0.654	1	1	1.14	0.0015	0.9	0.9	0.0141	0.0005	28.28
0.04	0.6	0.4	0.8	1	1	1	0.418	0.510	1	1	0.53	0.0015	0.1	0.5	0.0219	0.0066	3.30
0.04	0.6	0.4	0.8	1	1	1	0.418	0.654	1	1	0.58	0.0015	0.1	0.9	0.0257	0.0066	3.87
0.04	0.6	0.4	0.8	1	1	1	0.510	0.654	1	1	0.77	0.0015	0.5	0.9	0.0282	0.0049	5.78
0.04	0.6	0.4	0.8	1	1	1	0.654	0.418	1	1	0.90	0.0015	0.9	0.1	0.0082	0.0005	16.49
0.04	0.6	0.4	0.8	1	1	1	0.510	0.510	0.5	0.5	0.55	0.0015	0.5	0.5	0.0094	0.0049	1.93

For this case it turns out that when the learned connection strengths $\omega_{2iA}(t)$ and $\omega_{2iB}(t)$ are equal in both agents, then under full contagion ($\gamma_{AB} = \gamma_{BA} = 1$) the learning with contagion is estimated to be more than 3 times faster than without contagion. When no full contagion takes place (e.g., $\gamma_{AB} = \gamma_{BA} = 0.5$), then the learning speed ratio is estimated proportionally lower.

Neglecting extinction (assuming small ζ), the expression for learning speed can be simplified; in this case the learning speed ratio can be simplified by putting $\zeta=0$:

$$\begin{aligned} & \text{learning speed ratio for } \omega_{2iA}(t) \text{ without extinction} = \\ & \frac{[\mu_{AB} [\omega_{1iA} + \alpha_B \gamma_{BA} \omega_{1iB}]]^2}{[\alpha_A \omega_{1iA}]^2} \\ &= (\mu_{AB} / \alpha_A)^2 [1 + \alpha_B \gamma_{BA} (\frac{\omega_{1iB}}{\omega_{1iA}})]^2 \end{aligned}$$

$$= \left(\frac{1}{1 - \alpha_A \gamma_{AB} \gamma_{BA}} \right)^2 \left[1 + \alpha_B \gamma_{BA} \left(\frac{\omega_{1B}}{\omega_{1A}} \right) \right]^2$$

Note that this expression directly relates (as a square) to the expression for the learning speed ratio without extinction for $\omega_{1A}(t)$ above.