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An Agent-Based Model for Integrated Contagion and Regulation of Negative Mood

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Abstract. Through social interaction, the mood of a person can affect the mood of others. The speed and intensity of such mood contagion can differ, depending on the persons and the type and intensity of their interactions. Especially in close relationships the negative mood of a depressed person can have a serious impact on the moods of the ones close to him or her. For short time durations, contagion may be the main factor determining the mood of a person; however, for longer time durations individuals also apply regulation mechanisms to compensate for too strong deviations of their mood. Computational contagion models usually do not take into account such regulation. This paper introduces an agent-based model that simulates the spread of negative mood amongst a group of agents in a social network, but at the same time integrates elements from Gross' emotion regulation theory, as the individuals' efforts to avoid a negative mood. Simulation experiments under different group settings pointed out that the model is able to produce realistic results, that explain negative mood contagion and emotion regulation behaviours posed in the literature.

Keywords: emotion contagion and regulation, agent-based model.

1 Introduction

There is a wide consensus in sociological literature that human mood spreads through social networks [9, 11]. This social phenomenon is known as contagion. Especially negative moods are strongly influenced by social contacts (e.g., family, friends, colleagues, and neighbours), for example, when the social interaction involves conflict issues or stressful events [4, 15]. Agent-based computational models for contagion of different types of mental states can be found, for example, in [1, 10]. However, in addition to contagion at the social level, also emotion regulation within individuals plays an important role [3]. Emotion regulation is a process through which individuals balance their emotions by exerting forms of control on how they feel [8]. For instance, by avoiding situations or persons who trigger negative emotions, or suppressing anger when receiving bad comments from interviewers. By such emotion regulation mechanisms, persons have the ability to suppress negative influences from interaction with others and maintain a form of emotional homeostasis [7, 8]. For example, if a partner of a depressed person has regulation mechanisms that are strong

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enough, he or she does not need to become depressed, but if the mechanisms are less strong, there is a serious risk that the partner also becomes depressed.

In recent years researchers have focused on understanding the mechanisms of emotion regulation, and social contagion separately [2, 13, 15]. However, little information is available to explain how these processes work in an integrated manner by means of computational models. In this paper, an agent-based model is proposed that formalizes and simulates the integrated contagion and regulation of negative mood. In order to exemplify the proposed model, simulation experiments have been performed with a variety of scenarios that include varying personal characteristics and group or network compositions. Attributes were configured, to represent the personality and social characteristics of different individuals. Simulation traces were generated, to show behaviour of these individuals over time, under multiple conditions.

2 Mood Contagion and Regulation

In this section, important ideas and concepts in negative mood contagion and emotion regulation research are addressed. These ideas form the basis of the current computational model that will be formally described in the next section. As described in [5], the degree of mood contagion in groups is influenced by the valence and energy of the mood. One of the fundamental components in mood contagion is the *contagion strength* between individuals within a group [6]. It involves the type of interaction between individuals (*channel strength* from sender to receiver) and personality characteristics of the sender (*expressiveness*) and receiver (*openness*). For negative mood contagion, channel strength can be defined as the intensity of the social interaction, either via *physical contact* (i.e, face-to-face), or *virtual interaction* (i.e, text message, social networking) [16]. Neighbourhood and personality characteristics, affect the openness for mood contagion of a person [11, 12]. For example, a neurotic individual tends to aggravate negative perception towards incoming mood [14]. In addition to this, a bad neighbourhood (physical or social) also creates a negative influence towards individual's perception in social interaction [12]. Expressiveness is related to the ability of an individual to induce contagion, where an extravert individual can induce a stronger contagion of a negative mood than an introvert individual, because an extravert person expresses his or her internal feelings stronger than an introvert person [1].

Besides mood contagion, emotion regulation plays a role in the experience and transfer of moods. It is important to understand the emotion regulation process, by knowing which different strategies individuals use to exert control over their moods [2]. To serve this purpose, Gross' emotion regulation theory provides a number of strategies to affect individuals' level of emotion [7]. This theory differentiates these strategies into *antecedent-focused strategies* and *response-focused strategies*. The former type of strategies refer to the process preparing for response tendencies before they are (fully) activated, and the latter deal with the actual activation or suppression of the expression of emotional responses [13]. Antecedent-focused strategies can involve the external situation of the person (e.g., avoiding certain places or persons), or the internal processes (e.g., redirecting attention or cognitive interpretation). Gross [7, 8] mentions four examples of antecedent-focused strategies: *situation selection, situation modification, attentional deployment,* and *cognitive change*. In a

response-focused strategy, response modulation is used (e.g., suppressing expressing of negative emotions, or amplifying expression of positive emotions).

Situation selection involves selecting a situation that supports the individual's emotional well-being. This may involve physical and/or social aspects. For example, if a person has a bad response on low light intensity, a form of regulation is to increase this intensity. Especially relevant to the integration with social contagion processes, is the regulation of the social situation. For example, if a person feels bad in a certain social environment, he/she can decrease his/her openness for and intensity of social interaction. Situation modification is similar to selection, but addresses only some aspects of a situation. Attentional deployment includes redirection of attention, for example, on more neutral or positive elements [7]. Cognitive change refers to change in how an individual interprets the situation. Response modulation refers to physical or behavioural actions that decrease the expression of negative emotions [8].

3 The Agent-Based Model

The agent-based model introduced in this section combines knowledge on mechanisms for mood contagion and emotion regulation, as briefly introduced above. In this computational model these mechanisms are encapsulated, allowing the simulation of how fragile individuals in their social environment are, towards negative mood contagion. The model describes a process to maintain homeostasis for mood. Through social interaction, there is a habitual tendency of an individual to perceive the negative mood of others and to regulate his or her own moods. Both processes are governed by individual's socio-culture, default (norm) personality, and his or her negative mood. In the formalized model, all nodes are designed to have values ranging from *0* (low) to *1* (high). The interaction will determine the new value for each node, either by a series of accumulations or an instantaneous interaction. To represent these relationships in agent terms, each variable will be coupled with an agent's name (*A* or *B*) and a time variable *t*. The description of these formalizations is described below. For a global overview, see Fig. 1.

3.1 Norm Values

Norm values indicate which level each individual is inclined to approximate during the process: an individual tries too keep itself within safe boundaries around these values. These norm values can be seen as a basis for 'default behavioural patterns'; e.g., the openness a person tends to have, based on neighbourhood characteristics and level of neuroticism, or a default level of expressiveness, based on personality characteristics. These norm values are also the natural initial settings of the persons in scenarios. The norm value C_{normAB} at some point in time t for the channel of agent A to agent B, can be related to the amount of physical (PI_{AB}) and virtual (VI_{AB}) interactions that take place**,** where *0* means no physical or virtual interaction with others, and *1* means a lot of physical interaction [12]. This interaction is regulated by the proportional parameter α. If $\alpha = 0.5$, both types of interactions have the same effect, otherwise, one of these types of interactions has more effect on the channel norm value.

$$C_{normAB}(t) = \alpha.\ PI_{AB}(t) + (1-\alpha).VI_{AB}(t) \quad (1)$$

Note that the interaction can be bidirectional, so that $C_{normAB}(t) = C_{normBA}(t)$, but this is not assumed to be always the case; the model also covers asymmetric cases, for example, where frequently text messages are sent from *A* to *B* but not conversely, or *B* follows *A* on Twitter but not the other way around.

Next, the openness norm value O_{normA} of agent *A*, first relates to the (bad) neighbourhood circumstances of *A* expressed in a concept NH_A, where a value of *1* means a very 'bad' neighbourhood, which makes a person vulnerable to negative mood, and the value *0* means the neighbourhood does not make a person more susceptible to negative mood of others. NH_A is modelled as the product of the social (SNH_A) and physical (PNH_A) neighbourhood and of the person. If $PNH_A = 1$, then the physical neighbourhood is very 'bad', and it will have a negative effect on the person's susceptibility. By multiplication of the social and physical neighbourhood in (2), a more 'positive' social neighbourhood (with a low value), will make the impact of the 'bad' physical neighbourhood smaller [12].

$$NH_A(t) = SNH_A(t).PNH_A(t) \tag{2}$$

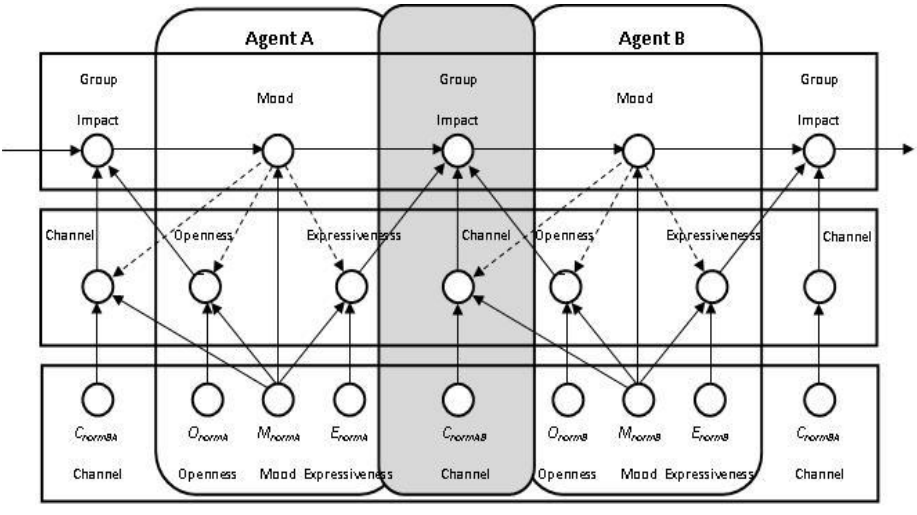

Fig. 1. Overview of the Agent-Based Model Integrating Mood Contagion and Regulation

The openness norm value O_{normA} of agent *A,* combines the concepts of a bad neighbourhood NH_A, with the concepts friends ratio NF_A and neuroticism N_A. In [12] it is described that the more friends you have, the less prone you are to negative mood contagion. The quantity NF_A is defined as a number between *0* and *1* (a 'friend ratio'): the number of friends is divided by a fixed number (serving as an upper bound) to normalise it. For example, if the upper bound taken is *10* (as in the simulations discussed in Section 4) then one friend will give $NF_A = 0.1$, whereas *7* friends will give $NF_A = 0.7$. Parameter φ regulates the equation; so that it can be modelled which concept can have more effect on the openness norm value than the other. In addition to this, [11] put forward that the more neurotic you are, the more susceptible you are

to negative mood of others. Therefore, the level of neuroticism N_A can amplify or reduce the positive effects of having such as a high number of friends and/or a not bad neighbourhood.

$$O_{normA}(t) = [\ \varphi.(1-NF_A(t)) + (1-\varphi).NH_A(t)].N_A(t) \quad (3)$$

Finally, in the current model, the expressiveness norm value E_{normA} of agent A is initialised by a number between *0* and *1*, not a formula. The number represents the level of expressiveness a person tends to approximate in daily life, where *0* means low expressiveness and *1*, high expressiveness.

3.2 The Dynamics of Mood Contagion and Emotional Regulation

In this section the dynamical model for mood contagion and regulation is introduced. A summary of the parameters and state variables of the model is shown in Table 1.

For the mechanisms behind mood contagion, elements from the model presented in [1] have been adopted. The main building block of mood contagion in this model is the contagion strength CS_{AB} from agent A to agent B, where it represents the type and intensity of the contact between agent A and agent B. The higher the value of CS_{AB}, the more contagion will take place.

$$CS_{AB}(t) = E_A(t).C_{AB}(t).O_B(t) \qquad \text{where } A \neq B \quad (4)$$

Here, E_A is the personal characteristic expressiveness (the degree in which a person can express his/her mood), C_{AB} the channel strength (intensity of contact, depending on the social relation) from A to B, and O_B the openness (the degree of susceptibility) of the receiver B. Using this equation, the group contagion strength is computed. The group contagion strength $CS_A^*(t)$ towards A is the overall strength by which the negative mood of all other group members is received by A:

$$CS_A^*(t) = \sum_{B \neq A} CS_{BA}(t) \quad (5)$$

Note that for the sake of simplicity here a linear (sum) combination is used. Alternatively, also a logarithmic or logistic combination function might be used. Given the mood levels $M_B(t)$ of the agents $B \neq A$ at time t, the weighted group impact $M_A^*(t)$ of all other agents in the group towards agent A is modelled as:

$$M_A^*(t) = \sum_{B \neq A} CS_{BA}(t).\ M_B(t) / CS_A^*(t) \quad (6)$$

More details of this model for contagion can be found in [1]. Next the dynamics of the mechanisms for integrated emotion regulation and negative mood contagion are modelled in (7), (8), (9), and (10). The general pattern underlying these dynamical relationships is

$$Y_A(t+\Delta t) = Y_A(t) + \tau.\ \langle change\_expression \rangle.\ \Delta t$$

Here the change of Y is specified for a time interval between t and $t + \Delta t$; the τ are personal flexibility parameters that represent the speed of the cognitive adjustment processes. Within <*change_expression*> two cases are considered: upward (positive) change <*upward_change*>, and downward (negative) change <*downward_change*>.

$$\langle change\_expression \rangle = (1-Y_A(t)).\ \langle upward\_change \rangle + Y_A(t).\ \langle downward\_change \rangle$$

The upward and downward change expressions are determined using the operator Pos(x) defined as Pos(x) = x when $x \geq 0$, else 0.

<*upward change*> = Pos(<*basic change*>)
<*downward change*> = - Pos(- <*basic change*>)

Within the basic change expression for (7), (8), and (9), two parts are considered. The first part incorporates the emotion regulation, and the second part the maintenance of homeostasis.

<*basic_change*> = <*regulation_change* > + <*maintenance_change* >

The latter change expressions were taken linear in the deviation:

<*regulation_ change* > = ζ. [M_{normA}-$M_A(t)$]
<*maintenance_ change* > = υ. [Y_{normA}-$Y_A(t)$]

Here ζ and υ are more specific flexibility parameters, for regulation and maintenance. Next it is shown how this general pattern was applied for channel strength (7), openness (8), and expressiveness (9). Firstly, the concepts of emotion regulation are represented in the dynamic adjustment of the strength of the channel from agent A to B. In (7) this occurs by comparing the current mood level to the mood norm value and comparing the current channel level with the channel norm value. These possible deviations influence the adjustment in the strength of the channel that the agent makes. This covers situations in which a person is infected by negative mood from other persons and directs his/her attention away, or physically moves to another place.

$$C_{BA}(t+\Delta t) = C_{BA}(t) + \tau_{CA}.[\,(1- C_{BA}(t)).\ \mathrm{Pos}(\zeta_{CA}\,[M_{normA}-M_A(t)] + \upsilon_{CA.}\,[C_{normBA}-C_{BA}(t)]) - C_{BA}(t).\ \mathrm{Pos}(-\zeta_{CA}\,[M_{normA}-M_A(t)] - \upsilon_{CA.}\,[C_{normBA}- C_{BA}(t)])]\ .\Delta t \quad (7)$$

The dynamic relation for the openness O_A of agent A models another antecedent-focused emotion regulation mechanism [7].

$$O_A(t+\Delta t) = O_A(t) + \tau_{OA}.[(1-O_A(t)).\mathrm{Pos}(\zeta_{OA}\,[M_{normA} - M_A(t)] + \upsilon_{OA}.\,[O_{normA}- O_A(t)]) - O_A(t).\ \mathrm{Pos}(-\zeta_{OA}\,.[M_{normA}-M_A(t)] - \upsilon_{OA}\,.[O_{normA}-O_A(t)])].\Delta t \quad (8)$$

The expressiveness E_A of agent A involves a response-based emotion regulation mechanism [7, 8]. In (9), expressiveness is adjusted towards the norm value, but also adjusted to decrease expression of negative mood.

$$E_A(t+\Delta t) = E_A(t) + \tau_{EA}.[(1-E_A(t)).\ \mathrm{Pos}(\zeta_{EA.}\,[M_{normA} - M_A(t)] + \upsilon_{EA.}\,[E_{normA} - E_A(t)]) - E_A(t).\ \mathrm{Pos}(-\zeta_{EA.}\,[M_{normA}-M_A(t)] - \upsilon_{EA.}\,[E_{normA} - E_A(t)])].\Delta t \quad (9)$$

Finally in (10), an internal antecedent-focused emotion regulation mechanism called re-appraisal [8] is modelled. Here within the generic pattern discussed above the expression <*basic_change*> is instantiated as follows.

<*basic_change*> = <*contagion_change*> + <*reappraisal_ change*>

Table 1. Parameters and state variables of the model

Concepts	***Formalization***
negative mood of agent *A*	M_A
norm value for the negative mood of agent *A*	M_{normA}
weighted group impact	$M_A{}^*$
expressiveness of agent *A* (sending side)	E_A
norm value for expressiveness of agent *A*	E_{normA}
channel strength from agent *A* to agent *B*	C_{AB}
norm value for channel from agent *A* to agent *B*	C_{normAB}
contagion strength from agent *A* to agent *B*	CS_{AB}
overall group contagion strength towards agent *A*	$CS_A{}^*$
openness of agent *A* (receiving side)	O_A
norm value for openness of agent *A*	O_{normA}
physical interaction from *A* to *B* (face-to-face)	PI_{AB}
virtual interaction from *A* to *B*	VI_{AB}
number of friends 'friend ratio' of agent *A*	NF_A
bad neighbourhood of agent *A*	NH_A
level of neuroticism of agent *A*	N_A
bad social neighbourhood of *A*	SNH_A
bad physical neighbourhood of *A*	PNH_A
proportional parameter for C_{normA}	α
proportional parameter for O_{normA}	φ
flexibility parameter for *Y (*regulation_change);	ζ_{YA}
flexibility parameter for *Y* (maintenance_change);	υ_{YA}
flexibility parameter of agent *A* for the re-appraisal emotion regulation in (10)	λ_A
bias of agent *A*	β_A
flexibility parameter of *Y* (in a change expression); see (7), (8), (9), (10)	τ_{YA}

where

$$<reappraisal\_change> = \lambda_A.\ [M_{normA}\text{-}M_A(t)]$$
$$<contagion\_change> = CS_A{}^*(t).\ [\ \beta_A\ .(1\text{-}(1\text{-}M_A(t))\ (1\text{-}M_A{}^*(t))) + (1\text{-}\beta_A)\ .M_A(t).\ M_A{}^*(t)\ \text{-}\ M_A(t)]$$

The latter expression was adopted from [1]. This provides the following mood dynamics relation:

$$M_A(t+\Delta t)= M_A(t) + \tau_{MA}\ .[(1\text{-}M_A(t)).\ \text{Pos}(CS_A{}^*(t)\ [\ \beta_A.(1\text{-}(1\text{-}M_A(t)).(1\text{-}\ M_A{}^*(t))) + (1\text{-}\beta_A).\ M_A(t).M_A{}^*(t)\ \text{-}\ M_A(t)] -\ \lambda_A.[M_{normA}\text{-}M_A(t)])] - M_A(t).\ \text{Pos}(-\ CS_A{}^*(t).[\ \beta_A\ .(1\text{-}(1\text{-}M_A(t)).(1\text{-}M_A{}^*(t))) + (1\text{-}\beta_A)M_A(t)M_A{}^*(t) -\ M_A(t)] + \lambda_A[M_{normA}\text{-}M_A(t)])\ ]\ .\Delta t \quad (10)$$

4 Simulation Results

The model was implemented in different numerical software environments, one of which was Matlab. Multiple compositions of groups and networks were simulated, but for the sake of brevity, in this section the simulation scenario with only three agents are considered: namely; (**A**) a 'depressed' person with a very negative mood, (**B**) his/her life partner, and (**C**) his/her friend. Through this scenario, it is explored how the negative mood of a person can spread through his/her social network and can be controlled by emotion regulation mechanisms in the receiving persons. For all scenarios, the current simulations used the following parameters settings; t_{max}=*1000*, Δt = *0.1*, flexibility parameters τ_{YA} = *0.5* for *openness*, *channel strength*, *expressiveness,* and *0.1* for negative mood. These settings were obtained from previous systematic experiments to determine to the most suitable parameters values in the model. It means, several experiments were conducted to determine how a reasonable time scale and grain size of the simulation could be obtained. In this way, an appropriate setting for the parameters for speed of change, and of the time step Δt was chosen. The other parameters in principle can be chosen in any form as they reflect characteristics of the situation modelled. Table 2 summarizes the (initial) settings for the different agents.

Table 2. Individual Profiles for Each Agent

	Scenario #1			**Scenario # 2**			**Scenario # 3**			**Scenario # 4**		
	A	**B**	**C**	**A**	**B**	**C**	**A**	**B**	**C**	**A**	**B**	**C**
Initial M	0.9	0.4	0.2	0.9	0.4	0.2	0.9	0.4	0.2	0.9	0.4	0.2
M_{norm}	0	0	0	0	0	0	0	0	0	0	0	0
O_{norm}	1	1	1	1	1	1	1	1	1	1	1	1
E_{norm}	1	1	1	1	1	1	1	1	1	1	1	1
C_{norm}	1	1	1	1	1	1	1	1	1	1	1	1
λ	0	0	0	0	1	1	0	0	0	0.5	0.5	0
B	1	0.5	0	1	1	1	0.5	0.5	0.5	1	0.5	0
υ (for all opennes O, channels C and expressiveness E)	0	0	0	0	0.1	0.1	0	0	0	0.1	0.1	0
ζ (for all opennes O, channels C and expressiveness E)	0	0	0	0.1	0.1	0.1	0	0	0	0.1	0.1	0.1

Scenario # 1

The results of this scenario are shown in Fig. 2. During the simulation, the agent *A* stays on his negative initial mood. He is not capable of regulating his mood (since he is too depressed; his emotion regulation mechanisms do not work) and transmits his negative mood to his partner and friend.

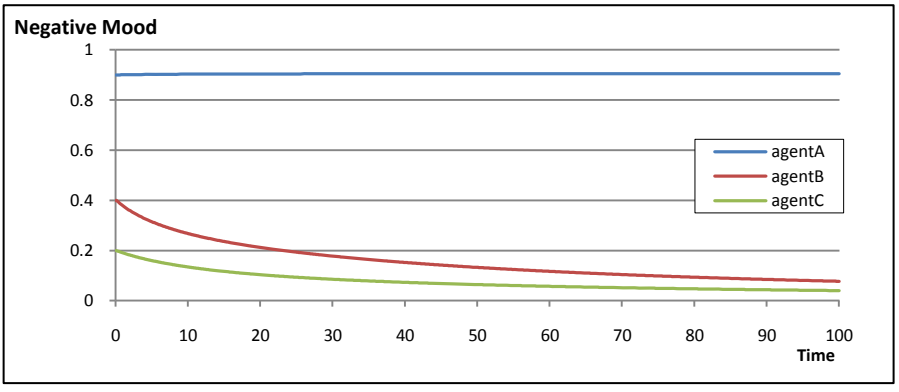

Fig. 2. Simulation results scenario 1

Because the partner and friend do have intact emotion regulation mechanisms, they are not infected to the level of the 'depressed' person's negative mood. The stronger their emotion regulation mechanisms are, the less the 'depressed' person can infect them with his negative mood. Furthermore, agent *B* has a higher negative mood bias ($\beta = 0.5$), than agent A ($\beta = 0$), therefore, agent *B*'s negative mood decreases less fast than for agent *C*.

Scenario # 2

Here all agents have a maximum negative mood bias ($\beta = 1$), by which they all approximate the highest initial negative mood (in this case that of the 'depressed' person, agent *A*). If no agent would have working emotion regulation capacities, all agents would increase to a negative mood level of 0.9. Now agent *B* and *C* have small emotion regulation capacities and therefore, they do not fully increase to the initial mood level of agent *A*. Fig. 3 depicts the results for this scenario.

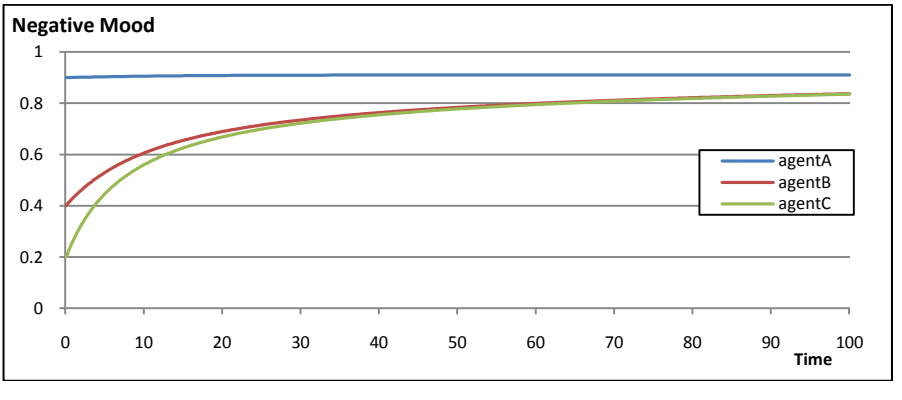

Fig. 3. Simulation results scenario 2

Scenario #3
This scenario represents the baseline where no emotion regulation mechanisms exist in the three agents. In this case, all agents have a negative mood bias ($\beta = 0.5$), which has the effect that all the agent's mood levels approximate the average initial mood setting (see Fig. 4).

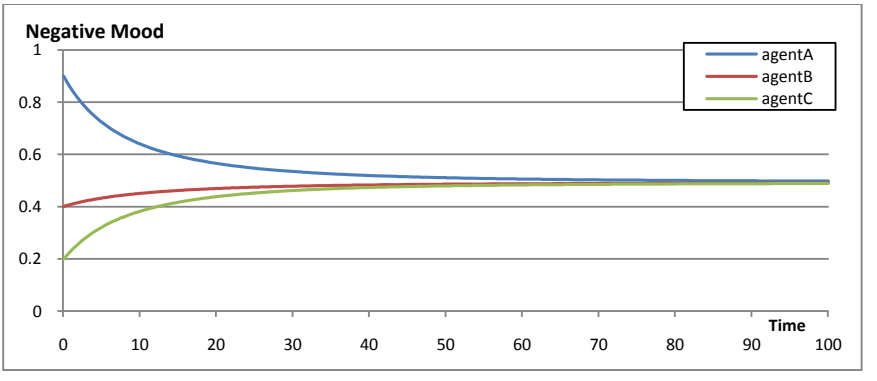

Fig. 4. Simulation results for scenario 3

The emotion regulation mechanisms in agent *A* and *B*, let the negative mood levels of agent *A* and *B* increase to a lesser extent. As can be seen from Fig. 4, this scenario shows how the negative bias β and emotion regulation mechanism have opposite effects.

Scenario #4
In this scenario, agent *C* does not have working emotion regulation mechanisms, but agent *A* and *B* do. In Fig. 5 it is shown that the emotion regulation mechanisms in agent *A* and *B*, let the negative mood levels of agent *A* and *B* decrease to a lesser extent, than that of Agent *C*, compared with scenario 3 (Fig. 5), where no agent had emotion regulation mechanisms that work. This shows how the negative bias β and emotion regulation mechanism have opposite effects: A high negative bias (β>0.5) can increase the negative mood of the agent, intact emotion regulation mechanisms (λ_A or υ of openness *O*, channel strength *C* or expressiveness *E* nonzero) will reduce this effect.

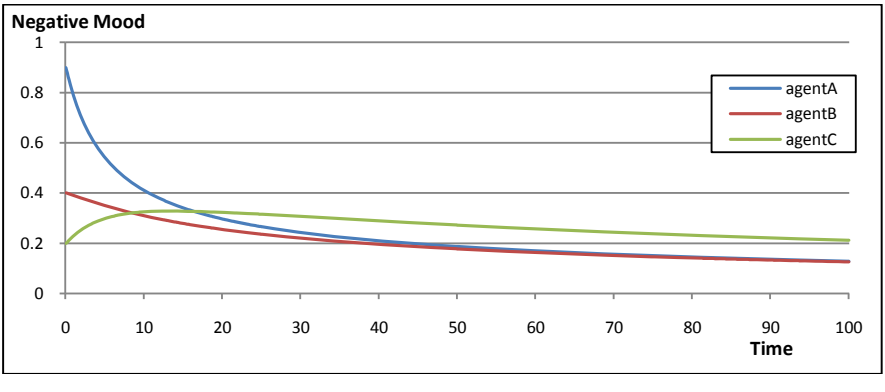

Fig. 5. Simulation results for scenario 4

5 Mathematical Analysis

In this section, an analysis is made of possible equilibria of the model. These are values for the variables of the model for which no change occurs. Taking as a point of departure the generic pattern,

$$Y_A(t+\Delta t) = Y_A(t) + \tau.<change\_expression>.\Delta t$$

and assuming τ nonzero, this is equivalent to $<change\ expression> = 0$ for all variables Y_A. Moreover, as

$$<change\_expression> = (1-Y_A(t)).\text{Pos}(<basic\_change>) - Y_A(t).\text{Pos}(-<basic\_change>)$$

the criterion for an equilibrium is:

$$(1-Y_A(t)).\text{Pos}(<basic\_change>) - Y_A(t).\text{Pos}(-<basic\_change>) = 0$$

Note that always $Pos(x) = 0$ or $Pos(-x) = 0$; this implies the following lemma:

Lemma 1
For any nonzero η_1 and η_2 it holds
$\eta_1 Pos(x) + \eta_2 Pos(-x) = 0$ iff $x = 0$.

By Lemma 1 it follows that for cases that $Y_A(t)$ is nonzero and < 1, the equilibrium criterion is

$$<basic\_change> = 0$$

If this is applied to dynamic relations (7) to (10) the following four equilibrium equations are obtained:

$$\zeta_{CA}.[M_{normA}-M_A] + \upsilon_{CA}.[C_{normBA}-C_{BA}] = 0 \quad (11)$$
$$\zeta_{OA}.[M_{normA}-M_A] + \upsilon_{OA}.[O_{normA}-O_A] = 0 \quad (12)$$
$$\zeta_{EA}.[M_{normA}-M_A] + \upsilon_{EA}.[E_{normA}-E_A] = 0 \quad (13)$$
$$\beta_A.(1-(1-M_A).(1-M_A*)) + (1-\beta_A).M_A.M_A*-M_A + \lambda_A.[M_{normA}-M_A] = 0 \quad (14)$$

The first three equations are equivalent to (here the following short notation is used: $devY = Y_{norm} - Y$ (deviation of Y from norm value)):

$$devC_{BA} = -(\zeta_{CA}/\upsilon_{CA}).devM_A \quad \text{from (11)}$$
$$devO_A = -(\zeta_{OA}/\upsilon_{OA}).devM_A \quad \text{from (12)}$$
$$devE_A = -(\zeta_{EA}/\upsilon_{EA}).devM_A \quad \text{from (13)}$$

In particular, it follows that either none of C_{BA}, O_A, E_A, M_A deviates from its norm, or all of them deviate from their norm (in a proportional manner). For the special case $M_{normA} = 0$ used in the experiments, it holds $devM_A = -M_A$, and therefore the equations are:

$devC_{BA} = (\zeta_{CA}/\upsilon_{CA}).\ M_A$
$devO_A = (\zeta_{OA}/\upsilon_{OA}).\ M_A$
$devE_A = (\zeta_{EA}/\upsilon_{EA}).M_A$

Having exploited the first three equations, what remains is the fourth one. To analyse this one, the following lemma is useful.

Lemma 2
For any A it holds:
$M_A* = 0$ iff $M_B = 0$ for all $B \neq A$ with nonzero CS_{BA}
$M_A* = 1$ iff $M_B = 1$ for all $B \neq A$ with nonzero CS_{BA}

As the fourth equation is rather complex in its general form, it is analysed for a number of special cases. In particular, assume $\lambda_A = 0$ (no re-appraisal). Then the fourth equation can be rewritten as follows:

$\beta_A.M_A* - M_A.[1 - \beta_A - M_A* + 2\beta_A.M_A*] = 0$
$M_A = \beta_A.M_A*/[(1 - \beta_A).(1 - M_A*) + \beta_A.M_A*]$,
if $(1 - \beta_A).(1 - M_A*) + \beta_A.M_A* \neq 0$

For this case, equilibria can occur with values different from 0 and 1, which may depend on the initial values. In addition, three special cases for β_A are considered: $\beta_A = 0$, $\beta_A = 0.5$, $\beta_A = 1$.

Case I. $\lambda_A = 0$, $\beta_A = 0$
In this case the fourth equation can be rewritten into

$M_A.M_A* - M_A = 0,$

which is equivalent to

$M_A = 0$ or $M_A* = 1$

By Lemma 2 this is equivalent to

$M_A = 0$ or $M_B = 1$ for all $B \neq A$ with nonzero CS_{BA}

This implies that for this case no equilibria exist with values different from 0 and 1.

Case II. $\lambda_A = 0$, $\beta_A = 0.5$
In this case the fourth equation can be rewritten into

$0.5.(M_A + M_A* - M_A.M_A*) + 0.5.M_A.M_A* - M_A = 0,$

which is equivalent to $M_A = M_A*$
For this case equilibria can occur with values different from 0 and 1, which may depend on the initial values.

Case III. $\lambda_A = 0$, $\beta_A = 1$
In this case the fourth equation can be rewritten into

$$M_A - M_A.M_A^* = 0$$

which is equivalent to

$$M_A = 1 \text{ or } M_A^* = 0$$

By Lemma 2 this is equivalent to

$$M_A = 1 \text{ or } M_B = 0 \text{ for all } B \neq A \text{ with nonzero } CS_{BA}$$

As for Case I, this implies that for this case no equilibria exist with values different from *0* and *1*.

6 Discussion

Research into the mechanisms of emotion regulation and social contagion has mainly been conducted separately [2, 13, 15]. In the current work, it was investigated how these processes work in an integrated manner, by means of a computational model. An agent-based model is proposed, that formalizes and simulates the integrated contagion and regulation of negative mood. The current model was inspired by a number of theories, namely emotion contagion and Gross' emotion regulation theory [1, 2, 5, 7]. For short time durations, contagion may be the main factor determining the mood of a person; however, for longer time durations individuals also apply regulation mechanisms to compensate for too strong deviations of their mood. Computational contagion models usually do not take into account such regulation.
Simulation results show interesting patterns that illustrate the combined effect of negative mood contagion and emotion regulation. Together, these elements can be used to understand how a person is capable to maintain his or her mood, while maintaining social interactions with another person. For this model, a mathematical analysis shows how such equilibria are indeed possible for the model. Note that for the sake of simplicity mood affecting external events during a simulated process have been left out of consideration. However, it is not difficult to include them too.

In follow up research, more attention will be focused to implement this model in a large scale social networks and to see important emergent behaviours that possibly exist when more agents are involved. Furthermore, it would be interesting to study a situation at a societal level where agents can also change their behaviours (such as relapse, recovery, and susceptibility), by introducing additional attributes and parameters into the model. In addition, this model can be used as a foundation to design software agents that capable to understand and aware about humans and their interactions. By using this model, software agents will use this as knowledge to provide appropriate actions to support humans pertinent to their predicted states (e.g. the level of negative mood). Future work of this model can be extended to incorporate multiple types of emotion and their interaction. Moreover, this model has a potential

to be useful to provide a foundation to understand how negative mood can be propagated via social media (e.g., Facebook, MySpace, Twitter).

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