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A SIS model with propagation of conducts

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Abstract In this work we study a Susceptible-Infected-Susceptible model coupled with a continuous opinion dynamics model. We assume that each individual can take measures to reduce the probability of contagion, and the level of effort each agent applies can change due to social interactions. We propose simple rules modeling the propagation of conducts that modify the level of effort, and analyze their impact on the dynamics of the disease.

We derive a finite dimensional set of ordinary differential equations describing the evolution of the epidemics and the mean value of the effort parameter, and analyze the equilibria of the system. Let us remark that the stability of the endemic phase and disease free equilibria depend only on the mean value of the levels of efforts, and not on the initial distribution of agents in the space of efforts.

Keywords epidemic models · social interactions · reproduction number · opinion dynamic

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1 Introduction

In the last few years emerged an increasing interest on epidemic models involving changes in socio-cultural norms and conducts, partly motivated by the emergence of anti-vaccine movements in different countries and the challenge they pose to the control of epidemic outbreaks.

Several authors divided the susceptible population in three (or more) different groups, the vaccinated, the non-vaccinated individuals, and the ones that decided to remain non-vaccinated, see for instance [15]. Also, related models considered two groups of susceptible agents, the ones who are aware and those who are not aware of the threat of an infection [5,11,22]. In those cases, each group has its own rate of contagion. Of course, a class of exposed agents, together with groups with different degree of susceptibility or age-dependent, were considered, although the difficulty to analyze the corresponding models increases since they lead to coupled differential equations systems, the size of each group being described through an ordinary differential equation, involving the many parameters that describe the transitions between the different groups.

Usually, agents can move from one group to another one due to the mechanisms of the disease (for instance, a susceptible agent was exposed and becomes infected, or an infected agent recovers after the infection), or due to a personal decision based on its own beliefs or the influence of other agents. So, the transitions involve a set of ordinary differential equations modelling the disease dynamic coupled with different social contact processes like the voter model, the cultural transmission model of Axelrod [2], or other discrete opinion dynamic models like the ones of Galam, Sznajd, or Ochrombel [10,12,17,19]. Now, the possibility of an epidemic outbreak, and its long-term impact will depend on the size of those groups, the mechanisms of transmission of the disease, the social contact process, and also on the clustering or other properties of the network modelling the social structure of the population, see for instance [7,16,18,21,23].

However, there are many infectious diseases that can not be analyzed with these kind of models because their prevention depends on a sustained effort over time through preventive measures, and there are no vaccines available. As a paradigmatic examples we can consider the actual Covid-19 pandemic, the 2009 flu pandemic caused by the H1N1 virus, or the outbreaks of Dengue, Zika, and Chikungunya [8]. In those cases it is possible to analyze the spread of conducts (and misconducts) related to infectious diseases using continuous opinion dynamic models, like the ones considered by Bellomo, Deffuant, Toscani, Weisbuch and their collaborators, see for instance [1,3,4,6,13,14,20].

So, we present here a variant of a classic Susceptible-Infected-Susceptible (SIS) model coupled with a continuous opinion dynamic model. Briefly, the state of any agent j in the society is characterized by the pair (a_j, p_j), where $a_j \in \{I, S\}$ means that the agent is infected or susceptible respectively, and $p_j \in (0, 1)$ is related to its level of effort to avoid the infection, the lower is p, the greater are the prevention measures taken by the agent to avoid contagion.

When two agents interact, they change their states as a consequence of both the epidemic and opinion dynamics, as detailed in Section §2. Although the rates of

contact β and recovery γ remain unchanged, a susceptible agent i is infected with probability $p_i\beta$ after a contact with an infected agent; then, both agents increase or decrease their protection levels depending on the existence or not of contagion after the interaction. If two susceptible or two infected agents interact, each one moves its own effort level toward the other agent value.

Let us mention a related model by Funk, Gilad, Watkins and Jansen [9], where the discrete set of positive integers $\{k\}_{k\geq 0}$ represent different levels of awareness or effort, the measures taken to reduce the susceptibility (being 0 the maximum level of awareness, which implies immunity against the disease). The spreading of awareness on the population depends on several mechanisms: information transmission due to interaction among agents, a decay term since individuals forget the acquired information, and a reset term since each infected agent becomes fully informed and goes to 0. A susceptible-infected-recovered (SIR) model was considered, which always stops in an equilibrium without infected people. When an infected agent interacts with a susceptible agent at level k, the probability of infection is given by $(1-\rho^k)\beta$, for some fixed $\rho \in [0,1]$. The authors studied this model using agent based simulations, and no mean field or kinetic equations were derived. Let us observe that long range jumps are accepted, since infected agents go directly to zero, introducing some kind of nonlocality on the equations.

We are mainly interested here in the extinction or not of the disease. To this end, in Section §3 we derive a system of ordinary equations describing the number of susceptible agents and the mean value of the level of efforts of the population, $\langle p\rangle$ in the mean field approximation.

Although the system depends on $\langle p_s\rangle$, the mean value of the level of efforts of the susceptible population, simulations show that $\langle p\rangle$ and $\langle pS\rangle$ are very similar, and coincide when the parameter in the social interaction goes to zero. So, assuming $\langle p\rangle = \langle p_s\rangle$, we study the system of ordinary differential equations satisfied by $\langle p\rangle$ and the proportion S of susceptible agents. We find the fixed points and classify them according to their stability in Section §4. We show in particular the existence of a critical parameter depending only on the contact rate β and on the recovery rate γ, and not in the initial value of $\langle p\rangle$, such that the disease does not become endemic if and only if

$$Rm := \frac{2\beta}{\gamma(1+\beta)} \leq 1.$$

(notice that 2β is the rate of contagion in this model). So this generalizes the classical result for the basic reproduction number $R0 = 2\beta/\gamma$ of the SIS model. and the factor $1+\beta$ helps to reduce the propagation of the disease.

Also, in the long-time limit, the value $\langle p\rangle$ converges to $(1+\beta)^{-1}$ independently of the initial distribution of values of p in the population. Agent based simulations of the dynamics strongly agree with the theoretical results.

We conclude in Section §5.

2 The Model

Let us assume that we have a population of n agents, each one characterized by a pair (a_i, p_i), where a_i represents the state of agent i, $a_i = S$ if it is susceptible and $a_i = I$ if it is infected, and $p_i \in [0,1]$ denotes its level of measures of prevention to avoid infection.

When two agents i, j interact, they will change their parameters from (a_i, p_i) to (a_i^*, p_i^*), and from (a_j, p_j) to (a_j^*, p_j^*) respectively. The dynamics of infection and recovery are similar to the classical SIS model: if a susceptible agent i interacts with an infected agent, it becomes infected with probability $p_i\beta$. On the other hand, an infected agent becomes susceptible in a unit of time with probability γ, without interactions.

The level of measures of prevention of two susceptible agents (or two infected agents) will change following a rule similar to the one introduced by Deffuant and Weibuch, see [6,14,20], namely both agents move their parameter p close to the other agent value. However, if a susceptible agent interacts with an infected agent, both will increase (respectively, decrease) p whenever the agent remains susceptible (resp., becomes infected).

More precisely, given a positive fixed parameter $h \leq 1/2$, both dynamics are defined by the following rules:

- *Contagion* and *Fear:* a susceptible agent i becomes infected with probability βp_i after an interaction with an infected agent j. In this case, i and j feel that their efforts are not enough, and change their p parameter to

$$p_i^* = p_i - hp_i,$$

$$p_j^* = p_j - hp_j.$$

- *Confidence:* a susceptible agent i remains susceptible with probability $1 - p_i\beta$ after an interaction with an infected agent j. In this case, i and j feel that their efforts are excessive and change it to

$$p_i^* = p_i + h(1 - p_i),$$

$$p_j^* = p_j + h(1 - p_j).$$

- *Persuasion:* a susceptible (respectively, infected) agent i remains susceptible if he interacts with another susceptible (resp., infected) agent j, and they change theirs levels of effort to

$$p_i^* = p_i + h(p_j - p_i),$$

$$p_j^* = p_j + h(p_i - p_j),$$

  that is, each one adopts an effort level intermediate between its own value and the one of the other agent.
- *Recovery:* a random agent i is selected and becomes susceptible with probability γ if it was infected, and no changes of p_i occur.

Let us observe that both dynamics are coupled, in the sense that the same contact which can produce a new infection implies a change in the levels of effort of both agents.

Of course, different rules can be imposed here. For instance, we are assuming that both agents know the state of the other one, in order to increase or decrease their levels of effort. Different rules can be studied in a similar way, deriving the corresponding equations. However, drastic changes in effort levels, such as those that occur in [9], need a study that includes long jumps in their values, instead of infinitesimal ones. We will consider this problem in a separate work.

3 Theoretical Analysis

In this section we derive an ordinary differential equation for $\langle p \rangle$, the mean value of agents levels of susceptibility, and another one for S, the proportion of susceptible agents.

It is possible to obtain a more detailed description through a Boltzmann-like equation for the distribution of agents on the levels of susceptibility, coupled with a SIS system describing the proportion of agents in the infected/susceptible states. We defer the technical details and the mathematical proofs of existence, uniqueness and stability of such equations to another work, since the relevant parameter for the epidemic analysis turns to be the mean value of p.

We introduce now the following notation which will be used below. We assume that there are finitely many agents, say n, and denote $k(t)$ the number of susceptible agents at time t. The proportions $S(t)$ and $I(t)$ of susceptible and infected agents are then given by

$$S(t) = \frac{k(t)}{n}, \qquad I(t) = 1 - S(t).$$

The means value $\langle p \rangle$ and $\langle p_s \rangle$ of the p parameter in the whole population and in the susceptible population are

$$\langle p \rangle = \frac{1}{n} \sum_i p_i, \qquad \langle p_s \rangle = \frac{P_S}{k(t)}, \qquad P_S = \sum_{i \in Sus} p_i,$$

where we denote by Sus and Inf the (time dependent) subsets of susceptible and infected agents respectively.

3.1 Derivation of the mean-field equations.

We assume that in a unit of time, a pair of agents is selected at random, uniformly and interact following the rules described in section §2. We aim at obtaingin differential equations describing the evolution of the expected values of $S(t)$ and $\langle p \rangle$.

Let us fix an agent, say i, and study the expected change $p_i(t + \Delta t) - p_i(t)$ in a small time window $[t, t + \Delta t]$. Notice this change depends strongly on the state of agent i. Supposing first that i is susceptible, we have

$$p_i(t+\Delta t) - p_i(t) = \frac{2h\Delta t}{n(n-1)} \left[-(n-k)\beta p_i^2 + (1-p_i)(n-k)(1-\beta p_i) - \sum_{j \in Sus} (p_i - p_j) \right], \tag{1}$$

where the three terms in the right-hand side of equation (1) comes from the possible interactions. Indeed $p_i(t + \Delta t) - p_i$ will vary as follows:

- $-hp_i$, when becoming infected after interacting with one of the $n - k$ infected agents, which occurs with probability $\frac{2}{n}\frac{n-k}{n-1}\beta p_i$;
- $h(1 - p_i)$ when remaining susceptible after interacting with one of the $n - k$ infected agents, which occurs with probability $\frac{2}{n}\frac{n-k}{n-1}(1 - \beta p_i)$;
- $-h(p_i - p_j)$ when interacting with another susceptible agent j.

Notice eventually that the factor 2 comes from the fact that agent i can be selected as the first or second agent in the interaction.

When agent i is infected we have

$$p_i(t+\Delta t) - p_i(t) = h\Delta t \left[-p_i P^* + (1-p_i)P^{**} - \frac{2}{n(n-1)} \sum_{j \in Inf} (p_i - p_j) \right], \quad (2)$$

where P^* and P^{**} are the probabilities that a contagion occurs or not during the interaction. Let us compute first P^*, the probability that i infects some susceptible agent. Notice that the probability agent i interacts with agent j and infects it is

$$P(i \text{ infects } j) = \frac{2}{n(n-1)} \beta p_j,$$

i.e., the probability that i was selected, j was selected, and the contagion occurs. Summing over all susceptible agents j we obtain

$$P^* = \frac{2\beta}{n(n-1)} \sum_{j \in Sus} p_j = \frac{2\beta P_S}{n(n-1)}.$$

Likewise, the probability of i interacting with a susceptible agent j but not infecting it is

$$P(i \text{ not infecting } j) = \frac{2}{n(n-1)} (1 - \beta p_j).$$

Hence, the probability of i interacting but not infecting a susceptible agent is

$$P^{**} = \frac{2}{n(n-1)} \sum_{j \in Sus} (1 - \beta p_j) = \frac{2(k - \beta P_S)}{n(n-1)}.$$

Inserting these expressions of P^* and P^{**} in (2) we obtain

$$p_i(t+\Delta t) - p_i(t) = \frac{2h\Delta t}{n(n-1)} \left[-\beta p_i P_S + (1-p_i)(k - \beta P_S) - \sum_{j \in Inf} (p_i - p_j) \right]. \quad (3)$$

We can now derive now the equation for $\langle p \rangle = \frac{1}{n} \sum_{i=1}^n p_i$, the mean value of p. Splitting the sum as $\sum_{i=1}^n = \sum_{i \in Sus} + \sum_{i \in Inf}$, and using (1) and (3), we obtain

$$\begin{aligned} &\frac{n^2(n-1)}{2h} \frac{\langle p(t+\Delta t) \rangle - \langle p(t) \rangle}{\Delta t} \\ &= \sum_{i \in Sus} \Big[-(n-k)\beta p_i^2 + (1-p_i)(n-k)(1-\beta p_i) - \sum_{j \in Sus} (p_i - p_j) \Big] \\ &\quad + \sum_{i \in Inf} \Big[-\beta p_i P_S + (1-p_i)(k - \beta P_S) - \sum_{j \in Inf} (p_i - p_j) \Big]. \end{aligned}$$

Noticing that

$$\sum_{i \in Sus} \sum_{j \in Sus} (p_i - p_j) = \sum_{i \in Inf} \sum_{j \in Inf} (p_i - p_j) = 0,$$

this simplifies to

$$\frac{n^2(n-1)}{2h}\frac{\langle p(t+\Delta t)\rangle - \langle p(t)\rangle}{\Delta t} = (n-k)\sum_{i\in Sus}(1-(1+\beta)p_i) + \sum_{i\in Inf}(k-\beta P_S - p_i k)$$
$$= (n-k)[2k-(1+2\beta)P_S] - k\sum_{i\in Inf} p_i.$$

Writing

$$\sum_{i\in Inf} p_i = \sum_{i=1}^{n} p_i - \sum_{i\in Sus} p_i = n\langle p\rangle - k\langle p_s\rangle = n(\langle p\rangle - \langle p_s\rangle) + (n-k)\langle p_s\rangle$$

we obtain

$$\frac{n^2(n-1)}{2h}\frac{\langle p(t+\Delta t)\rangle - \langle p(t)\rangle}{\Delta t} = 2(n-k)k[1-(1+\beta)\langle p_s\rangle] - kn(\langle p\rangle - \langle p_s\rangle).$$

i.e. dividing by n^2,

$$\frac{(n-1)}{2h}\frac{\langle p(t+\Delta t)\rangle - \langle p(t)\rangle}{\Delta t} = 2S(1-S)[1-(1+\beta)\langle p_s(t)\rangle] - S(\langle p\rangle - \langle p_s\rangle).$$

We send $\Delta t \to 0$ to deduce the differential equation

$$\frac{(n-1)}{h}\frac{d}{dt}\langle p(t)\rangle = 4S(1-S)[1-(1+\beta)\langle p_s(t)\rangle] - 2S(\langle p\rangle - \langle p_s\rangle),$$

and then rescale time to get rid off the factor $h/(n-1)$, resulting in the final equation

$$\frac{d}{dt}\langle p(t)\rangle = 4S(1-S)[1-(1+\beta)\langle p_s(t)\rangle] - 2S(\langle p\rangle - \langle p_s\rangle) \tag{4}$$

Remark 31 *In much the same way, we get*

$$\frac{d}{dt}S = -2\langle p_s\rangle\beta S(1-S) + \gamma(1-S). \tag{5}$$

We omit the full derivation since it is similar to usual derivations for SIS models. It can be obtained following the lines of the previous computation, the main difference with classical models are the mean susceptibility $\langle p_s\rangle\beta$, and the factor 2, which appears since both the first and the second selected agent can become infected.

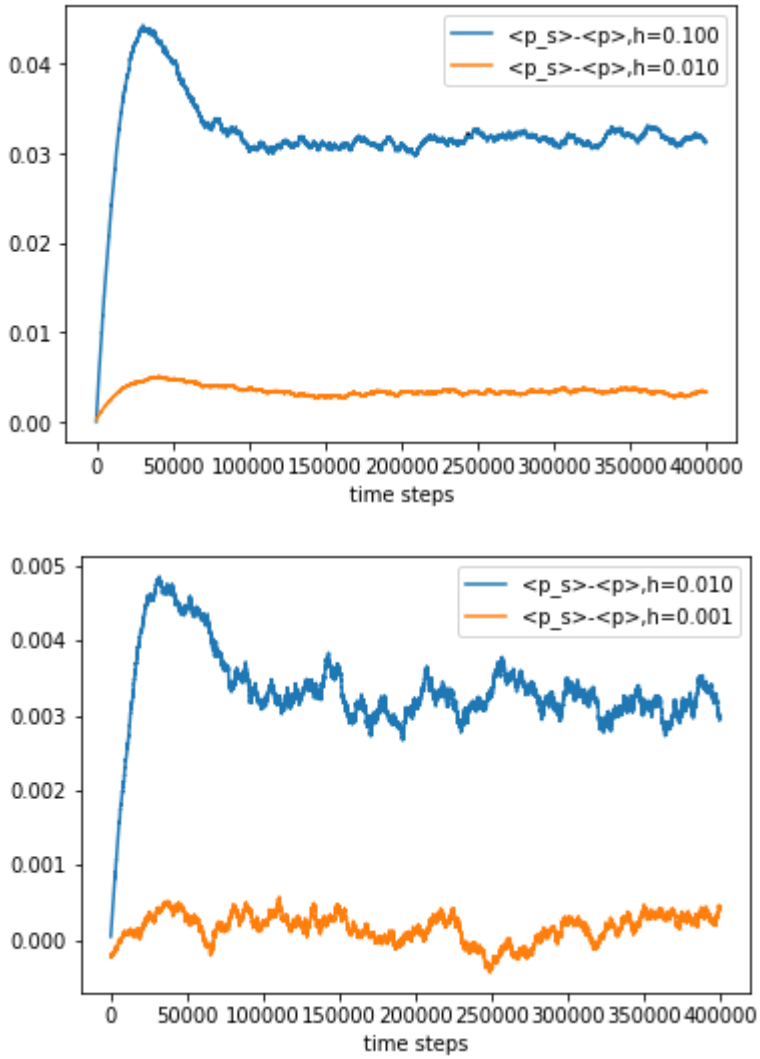

Fig. 1 Plot of $\langle p_s \rangle - \langle p \rangle$ for $h = 0.1$, 0.01, and 0.01 during a simulation with $n = 20000$ agents, $\beta = 0.7$, and $\gamma = 0.3$.

4 Qualitative study of the dynamic

Let us note that the definition of the microscopic interaction rules given in section §2 concerning the level of effort implies that both susceptible and infected agents react in the same way. This suggests that their mean level of effort would be the same. On the other hand, any time an agent is infected, its level of effort increases slightly, so we can expect a small bias toward lower levels for infected agents. The agent simulations in Figure 1 confirm this intuition, showing that the difference between $< p_s >$ and $< p >$ is positive of order h (however, this difference could

be negative for h small). Thus taking $h \ll 1$, we will assume from now on that $\langle p_s \rangle = \langle p \rangle$.

With this assumption, equations (4) and (5) for $\langle p \rangle$ and S become

$$\frac{d}{dt}\langle p \rangle = 4S(1-S)\Big(1-(1+\beta)\langle p \rangle\Big), \tag{6}$$

$$\frac{d}{dt}S = (1-S)\Big(\gamma - 2\langle p \rangle \beta S\Big). \tag{7}$$

Notice that the square $[0,1]\times[0,1]$ is clearly invariant for the system, and that its fixed points in $[0,1]\times[0,1]$ are the line $\{S=1\}$ and the point $(\langle p \rangle, S) = \Big(\frac{1}{1+\beta}, \frac{\gamma(1+\beta)}{2\beta}\Big)$ with $\frac{\gamma(1+\beta)}{2\beta} \leq 1$.

Let us study the asymptotic behaviour of a solution starting from a point $(\langle p \rangle(0), S(0)) \in [0,1]\times[0,1)$. Since $S(t) < 1$ for any t and noticing that $\frac{d}{dt}\langle p \rangle$ has same sign as $1-(1+\beta)\langle p \rangle$, we see that

$$\lim_{t\to+\infty} \langle p \rangle = \frac{1}{1+\beta}.$$

We can then rewrite (7) as

$$\begin{aligned}\frac{1}{2\beta}\frac{d}{dt}S &= \langle p \rangle (1-S)\Big(\frac{\gamma}{2\beta\langle p \rangle} - S\Big)\\ &= \langle p \rangle (1-S)\Big(Rm^{-1} + \varepsilon(t) - S\Big),\end{aligned}$$

where $Rm^{-1} = \frac{\gamma(1+\beta)}{2\beta}$ and $\varepsilon(t) \overset{t\to+\infty}{\longrightarrow} 0$. We now distinguish three cases.

If $Rm^{-1} > 1$ then there exists $T > 0$ such that $Rm^{-1} + \varepsilon(t) \geq 1$ for any $t \geq T$. It follows that for $t \geq T$ we have $\frac{d}{dt}S \geq 0$ with equality only when $S = 1$. We conclude that $\lim_{t\to+\infty} S(t) = 1$.

If $Rm^{-1} < 1$ then for any $a, b \in [0,1)$, $a < Rm^{-1} < b$, there exists $T' > 0$ such that for $t \geq T'$ we have

$$\begin{aligned}&\langle p \rangle (1-s)\Big(Rm^{-1} + \varepsilon(t) - s\Big) > 0 \qquad \forall\, s \in [0,a],\\ &\langle p \rangle (1-s)\Big(Rm^{-1} + \varepsilon(t) - s\Big) < 0 \qquad \forall\, s \in [b,1)\end{aligned}$$

Thus S enters the interval $[a,b]$ at some time $T \geq T'$ and stays there forever. Since this holds for any $a, b \in [0,1)$, $a < Rm^{-1} < b$, we deduce that $\lim_{t\to+\infty} S(t) = Rm^{-1}$.

If $Rm = 1$ then for any $\delta \in (0,1)$, there exists $T' > 0$ such that for $t \geq T'$, $|\varepsilon(t)| < \delta/2$ so that

$$\langle p \rangle (1-s)\Big(Rm^{-1} + \varepsilon(t) - s\Big) > 0 \qquad \forall\, s \in [0, 1-\delta].$$

Thus if $S(0) < 1-\delta$, then $S(t)$ must enter the interval $[1-\delta, 1]$ at some time $T \geq T'$ and then stays there forever. If $S(0) \geq 1-\delta$ then $S(t) \geq 1-\delta$ for any $t \geq 0$. Since this holds for any $\delta > 0$ we deduce that $\lim_{t\to+\infty} S(t) = 1$.

We can summarize the previous discussion in the following theorem.

Theorem 41 *For any initial condition* $(\langle p\rangle(0), S(0)) \in [0,1]\times[0,1]$*, the solution* $(\langle p\rangle, S)$ *of* (6)-(7) *satisfies*

$$\lim_{t\to+\infty}\langle p\rangle = \frac{1}{1+\beta}.$$

If $S(0)=1$ *then* $S(t)=1$ *for any* $t\geq 0$*, and if* $S(0)<1$ *then*

$$\lim_{t\to+\infty} S(t) = \begin{cases} 1 & \text{if } Rm\leq 1,\\ Rm^{-1} & \text{if } Rm>1,\end{cases}$$

where $Rm = \frac{2\beta}{\gamma(1+\beta)}$.

It follows from this result that Rm is the basic reproduction number of our model. Indeed if there is an epidemic outbreak i.e. $S(0)<1$, then the disease tends to disappear if $Rm\leq 1$ whereas it becomes endemic if $Rm>1$. Comparing with the basic reproduction number $R0=2\beta/\gamma$ of the classical SIS model, we observe the essential role played by the social interactions in our model which result in $Rm = R0/(1+\beta)$ thus effectively lowering the basic reproduction number.

We end this section by presenting some agent simulations to confirm that the ODE system (6)-(7) accurately models the agents dynamic and to illustrate the conclusions of Theorem 41. We show in figure 2 the time evolution of S and $\langle p\rangle$ averaged over 10 agent-based simulation of the dynamics with $n=20000$ agents, and parameters $\beta=0.4$, $\gamma=0.7$, starting all the agents with $p=1$. Observe that $S\overset{t\to+\infty}{\longrightarrow}1$ i.e., the disease goes to extinction, which agrees with the Theorem 41 since $Rm=0.8<1$. Notice also that $\langle p\rangle t\to+\infty\longrightarrow 0.7991$ while the expected equilibrium is $\frac{1}{1+\beta}=0.8026$, thus yielding a relative error of 0.4%.

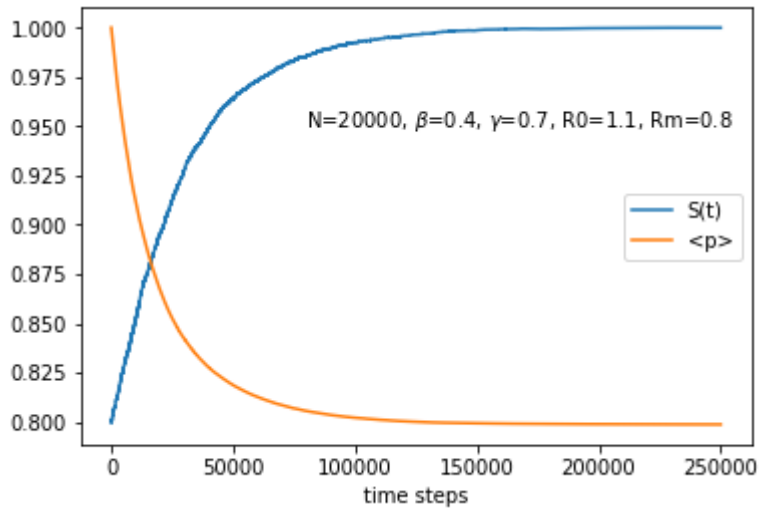

Fig. 2 Plot of $S(t)$ and $<p>$ averaged on 10 simulations with 20000 agents, $\beta=0.7$, $\gamma=0.3$, and $h=0.1$

Figure 3 displays a single run of an agent-based simulation of the dynamics with $n = 20000$ agents, and parameters $\beta = 0.7$, $\gamma = 0.3$, and $h = 0.1$, starting all the agents with $p = 1$ and $S(0) = 0.80$. Notice that $Rm \simeq 2.8 > 1$ so that disease should persists according to Theorem 41. In fact we can see in Figure 3 that $S \overset{t\to+\infty}{\longrightarrow} 0.3553$ thus yielding a relative error of 2.5% with respect to the theoretical value $Rm^{-1} \simeq 0.3643$. Notice that this is much greater than the asymptotic value of S in the classical SIS model. Eventually $\langle p \rangle \overset{t\to+\infty}{\longrightarrow} 0.5724$ which has a relative error of 2.7% with respect to the theoretical value $\frac{1}{1+\beta} \simeq 0.5882$.

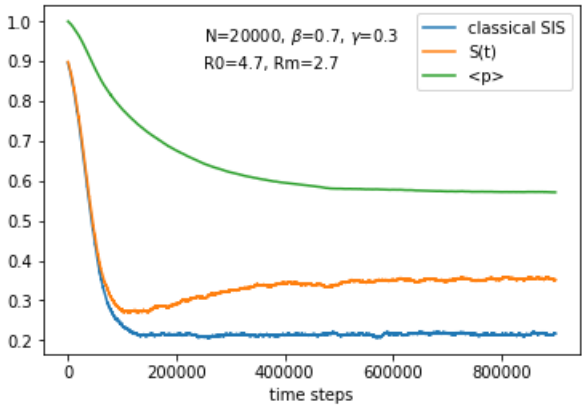

Fig. 3 Plot of $S(t)$ and $< p >$ in a single run with 20000 agents, $\beta = 0.7$, $\gamma = 0.3$, and $h = 0.1$

5 Conclusions and Final Remarks

We derived an epidemic model coupled with a continuous opinion dynamics model. We assumed that each individual can take measures to reduce the probability of contagion, and the level of effort that each agent applies change due to social interactions. We model few mechanisms, fear to contagion, confidence after a contact without contagion, and persuasion, as the main reasons for behavioral change, and we studied their impact on the dynamics of the disease.

We obtained a system of two ordinary differential equations, one for the proportion of infected people, and the second one for the mean value of the effort parameter. We study the asymptotic behaviour of the solutions, and we proposed a generalization of the basic reproduction number $R0$ denoted Rm and given by

$$Rm = \frac{2\beta}{\gamma(1+\beta)}$$

where β and γ are the contact and recovery rate of the disease. Let us remark that the factor 2 in this condition appears since both the first and second agents can become infected after an interaction. We prefer to keep this for simplicity and symmetry in the formulation of the problem, and a comparison with $R0$ for classical models must include the factor 2, that is, if the rate of contagion of a

disease studied with our model is β, this is equivalent to a rate 2β when only the first agent in some encounter can be infected.

There are several questions of practical interest, particularly in this moment with the Covid-19 pandemic active. To be clear, we are not claiming the direct applicability of our specific rules of social interaction, although trends in the population can be detected (like the degree of use of masks, sanitizer products, social distance), and this gives a personal probability of contagion β_i, which can be studied as the product $p_i\beta$ in our model. Of course, different questions are relevant if we consider Covid-19, since a SEIR model (including exposed and removed agents) seems to be a better one to analyze its evolution.

We have chosen the same scale of time for both dynamics. It is easy to change the frequency of interactions, by separating the ones related to the disease transmission, to the social ones ones which change the level of effort. However, this is an important point to consider when a SIR or SEIR model is involved.

Also, fundamental agents -governments, media, health organizations- were not considered here, and their role as social agents interacting with all the population cannot be neglected. On the negative side of social interactions, there are groups of people proposing innocuous and even harmful measures, like anti-vaccines movements, and we can see them today violating the social distance, or without masks, and trying to convince other people to imitate them. In a forthcoming paper we study their role and their influence on the stability of equilibria.

Declarations

Conflict of interest. The authors declare that they have no conflict of interest.
Ethics approval - Consent to participate - Availability of data and material. Not applicable.
Code availability. Python code could be available if required by the reviewer, and it will be uploaded to a repository after acceptance.
Authors' contributions. All the authors contributed equally.

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