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# Intransigent vs. volatile opinions in a kinetic epidemic model with imitation game dynamics

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**Abstract**

In the mathematical epidemiology community, there is an increasing interest in shaping the complex interplay between human behaviour and disease spreading. We give a contribution in this direction by illustrating a method to derive behavioural change epidemic models from a stochastic particle description by the means of kinetic equations. We consider an Susceptible–Infected–Removed–like model where contact rates depend on the behavioural patterns adopted across the population. The selection of the social behaviour happens during the interactions between individuals adopting alternative strategies and it is driven by an imitation game dynamics. Agents have a double microscopic state: a discrete label, that denotes the epidemiological compartment to which they belong, and the degree of flexibility of opinion, that is a measure of the personal attitude to change opinion and, hence, to switch between the alternative social contact patterns. We derive kinetic evolution equations for the distribution functions of the degree of flexibility of opinion of the individuals for each compartment, whence we obtain macroscopic equations for the densities and average flexibilities of opinion. After providing the basic properties of the macroscopic model, we numerically investigate it by focusing on the impact of the flexibility of opinion on the epidemic course and on the consequent behavioural responses.

**Keywords:** Boltzmann–type equations, Markov–type jump processes, SIR model, behavioural epidemiology, game theory

**Mathematics Subject Classification:** 35Q20, 37N25, 35Q70

## 1 Introduction

Among the many factors known to influence the spread of infectious diseases in modern societies, a central role is played by the *human behaviour*. A large body of evidence shows that spontaneous – as well as enforced – behavioural changes deeply influence the course of an epidemics. An

emblematic contemporary example is constituted by the ongoing COVID–19 pandemic, whose curbing has been threatened by the individual non–compliance to hygiene–related measures [39] and the hesitance about vaccines [23]. The analysis of Spanish flu data have shown that even one century ago the control of that devastating pandemic was strongly affected by behavioural changes, both in Europe [10, 43] and in the USA [4].

In the mathematical epidemiology community, the increasing awareness of the strict relation between humans and infectious diseases has led, during the last twenty years, to the development of a new scientific discipline: the behavioural epidemiology of infectious diseases (see [34, 49] and references therein). Behavioural change models have contributed to the understanding of the epidemic dynamics of, inter alia, vaccine–preventable paediatric diseases [2, 7, 12, 49], H1N1 influenza [27, 41] and also the COVID–19 disease [6, 8, 26]. In particular, the complex dynamics of human decision making has been modelled as an *imitation game* [2, 42] or, equivalently, as an infection of ideas process [49]. The imitation game, which is a mechanism typical of evolutionary game theory [47], formalizes the idea that human decisions are conducted by imitating others who appear to have adopted successful strategies. Specifically, different individuals' behaviours are modelled as different strategies whose convenience is defined by a cost–benefit balance. In epidemic modelling, this results in explicitly considering the interplay between the disease transmission and the spontaneous response of the population, where changes in human behaviour (and in particular in the cost–benefit assessment) are triggered by the epidemic dynamics and vice versa.

The imitation game dynamics has been investigated in many specific epidemic models described by different mathematical tools ranging from deterministic ODE models [2, 7, 12, 35, 42], to reaction–diffusion PDE models [33], up to fully stochastic models, such as network and individual–based models [20, 38], and a conspicuous body of results is now available (see also the review papers [46, 48]). On one hand, ODE and PDE models – being implemented at population level – are relatively simple and amenable to analytical and numerical investigations, but they disregard the individuals' characteristics. On the other hand, network and individual–based models allow to introduce (more) heterogeneity in the population structure, but they have to face the right balance between model complexity and computational boundaries.

A promising compromise between the macroscopic deterministic approach and the stochastic particle description is provided by methods based on mesoscopic theories. The mesoscopic scale acts as a *bridge* between the two antipodal scales by responding to the challenge of extracting all the macroscopically relevant information from the microscopic dynamics. There are mesoscopic methods – stemming from *statistical mechanics* – that propose a statistical description of the microscopic system under investigation and allow to derive macroscopic models. One of the most succesful ones is kinetic theory and the Boltzmann equation, that have proved to be a very effective tool to describe complex phenomena in many disparate fields [40], ranging from socio–economic dynamics to biological applications [28, 29]. Kinetic theory allows to start from the description of the evolution of a microscopic quantity associated to each individual through *interaction rules* and to derive a macroscopic model for the average quantities related to the population.

In epidemic modelling, the adoption of a kinetic theory approach has recently allowed to enhance the description of infectious diseases dynamics. In particular, the heterogeneity of disease transmission and progression linked to the viral load of each infected individual has been investigated [13, 31, 32]. Also, Boltzmann–like equations have been used to incorporate in the model the role of: social structure and wealth distribution within the host population [14, 15, 52], contact heterogeneity [37], and spatial propagation of the infection [3, 32].

However, to the best of our knowledge, no attempts have been made to derive behavioural change models and, in particular, game theoretical epidemic models, through a mesoscopic (kinetic) approach. For this class of models a specific microscopic trait is the personal attitude to change of opinion. As a matter of fact, each individual affected by the epidemics may be more or less persuadable on the convenience of a given strategy and, hence, more or less inclined to adopt

it. This leads, in the imitation game dynamics, to 'breaking' the homogeneity of the cost–benefit assessment among members of the same group and the symmetry between alternative strategies. The mechanisms of opinion variation are a hot topic in socio–physics and in computational social sciences. Roughly speaking, the heterogeneity of opinion formation and dynamics within a population may explain critical and complex social phenomena such as group polarization, conformity and extremisms (see, just to mention a few of papers, [11, 21, 22, 24]). Opinion dynamics has been also studied by the means of classical tools of kinetic theory starting from the pioneering work by Toscani [44], that gave rise to a wide subsequent literature, see e.g. [1, 5, 16, 17, 18, 19, 30, 45]. In particular, in the paper [30], the authors proposed a Boltzmann–type kinetic description of opinion formation on social networks, which takes into account a general connectivity distribution of the individuals described by a fixed distribution.

Motivated by the aforementioned reasons, in this paper we present a method relying on tools of kinetic theory to derive macroscopic epidemic models with imitation game dynamics from a stochastic particle model. We consider the host population as a *multi–agent* system, where the individuals (namely, the *agents*) are characterized by means of suitable microscopic variables, and the microscopic dynamics is modelled through interaction rules [13, 31, 32]. By introducing a probability density function of the microscopic states, we recover a statistical description of the system, whose evolution is described through kinetic equations. Then, from the kinetic evolution equations of the probability density function, we derive macroscopic equations for the moments of the probability density function, i.e. a macroscopic model, that naturally inherits a large number of features of the original microscopic dynamics.

Specifically, we associate to the agents a double microscopic state: a discrete label, that denotes the epidemiological compartment to which they belong, and a microscopic trait, that is related to the degree of *flexibility of opinion*. Since the novelty of our approach in this field of application, we consider a rather simple game theoretical framework – firstly introduced by Poletti *et al.* [42] – where an epidemic outbreak develops according to an Susceptible–Infected–Removed (SIR) model, but contact rates depend on the behavioural patterns adopted across the population. Precisely, susceptible individuals can conform to either the 'normal' or the 'altered' pattern of social contacts. The first group is composed of individuals that do not modify their social contacts with respect to the pre–epidemic situation and, as a consequence, are subject to the baseline risk of contracting the disease. The second group is composed of individuals that react to the epidemics by spontaneously reducing their contacts, and are hence subject to a lower risk of contracting the disease with respect to the first group. However, the latter perceive some extra cost deriving from social isolation. The switching between the two groups follows an imitation game dynamics, by depending on the individual cost–benefit balance in the mutual interactions [42]. Anyway, our approach is quite general and can be easily adapted to other epidemic frameworks (involving, for example, the crucial issue of vaccination behaviour).

The microscopic trait associated to each agent is the degree of flexibility of opinion, that is a measure of the individual propensity to change the currently adopted strategy during an interaction with one adopting the alternative strategy. The flexibility of opinion is intended here as an unchanging trait of the individual personality, not *a priori* related to the epidemic events. We assume that a high [resp. low] degree of opinion flexibility is characteristic of individuals that tend to overestimate [resp. underestimate] the cost they perceive with respect to that perceived by the others and, as a consequence, are more likely to switch [resp. preserve] their strategy. Specifically, we consider that each susceptible individual may give a different weight to the net cost associated to the contact pattern he/she is adopting (the normal one or the altered one) with respect to the net cost associated to the alternative contact pattern. From the ensuing heterogeneity in the individuals' cost–benefit assessment it follows that members of the same group have a different probability of switching to the other group depending on their degree of flexibility of opinion.

The rest of the manuscript is organized as follows. Section 2 deals with the development of

the behavioural change epidemic model: we illustrate the chosen compartmental structure, the imitation game dynamics as well as the role of the flexibility of opinion (Section 2.1). Then, we implement the modelling assumptions in a discrete in time stochastic process (Section 2.2), from which we derive a kinetic equation and, eventually, a macroscopic system of ODEs for the densities and the average flexibilities of opinion of the epidemiological compartments (Section 2.3). In Section 3, we provide the basic properties of the macroscopic model for some specific shapes of the functions related to the selection of the social contact pattern. In Section 4, we numerically investigate the transient and asymptotic behaviour of the ensuing model with emphasis on the impact of the individual intransigence/volatility of opinion; we also consider two relevant parameter settings differing from the magnitude of the risk of infection perceived by the two susceptible groups (Sections 4.1 and 4.2). Finally, concluding remarks and future perspectives are discussed in Section 5. The manuscript is complemented by the Appendix A.

# 2 The model

Let us consider a system of interacting individuals in the presence of an infectious disease that spreads through social contacts. We assume that a spontaneous reduction in the number of contacts could develop, as a defensive response, during the epidemics and affect the course of infection events [42]. This leads the emergence of two different behavioural patterns within the population: the 'normal' one and the 'altered' one. Individuals adopting the normal behaviour do not modify their contacts with respect to the pre–epidemic situation. Individuals adopting the altered behaviour spontaneously reduce their contacts in response to the epidemics.

We characterize individuals – that we also call *agents* – by a label $x \in \mathcal{X}$, which denotes the epidemiological compartment to which they belong, and by a microscopic quantity $c \in \mathcal{C}$, which measures the degree of flexibility of opinion, namely the individual attitude to change opinion and, hence, to switch between the alternative social contact patterns. We want to describe the microscopic mechanisms modelling the interactions between individuals and the switches between compartments that follow from either the disease progression or the change of social behaviour.

In order to give a statistical description of the multi–agent system, whose total mass is conserved in time, we introduce a distribution function for describing the probability density distribution of the agents characterized by the pair $(x, c) \in \mathcal{X} \times \mathcal{C}$, as

$$f(x, c, t) = \sum_{i \in \mathcal{X}} \delta(x - i) f_i(c, t), \tag{1}$$

where $\delta(x - i)$ is the Dirac delta distribution centred at $x = i$, and we assume that $f(x, c, t)$ is a probability density function, namely

$$\int_{\mathcal{X}} \int_{\mathcal{C}} f(x, c, t) dc dx = \sum_{i \in \mathcal{X}} \int_{\mathcal{C}} f_i(c, t) dc = 1, \quad \forall\, t \geq 0. \tag{2}$$

In (1)–(2), $f_i = f_i(c, t) \geq 0$ is the distribution function of the microscopic state $c$ of the agents that are in the $i$th compartment at time $t$. Hence, $f_i(c, t)dc$ is the proportion of agents in the compartment $i$, whose microscopic state lies between $c$ and $c + dc$ at time $t$. In general, the $f_i$'s, $i \in \mathcal{X}$, are not probability density functions because their $c$–integral varies in time due to the fact that agents move from one compartment to another.

Then, we can define macroscopic quantities by considering the moments of the distributions $f_i$'s. Precisely, we denote by

$$\rho_i(t) = \int_{\mathcal{C}} f_i(c, t) dc \tag{3}$$

the *density* of agents in the compartment $i$, thus $0 \leq \rho_i(t) \leq 1$ and

$$\sum_{i \in \mathcal{X}} \rho_i(t) = 1, \quad \forall\, t \geq 0.$$

If $\rho_i(t) > 0$, then we can also define the *average flexibility of opinion* as

$$m_i(t) = \frac{1}{\rho_i(t)} \int_{\mathcal{C}} c\, f_i(c,t) dc. \tag{4}$$

We observe that $\rho_i(t) = 0$ implies instead necessarily $f_i(c,t) = 0$ and, in such a case, the average flexibility of opinion is not defined because the corresponding compartment is empty. We also remark that, if the compartment is *almost* empty, then the average flexibility of opinion $m_i$, $i \in \mathcal{X}$, might not be fully consistent with the empirical average flexibility resulting from the particle description because the law of large numbers does not apply.

## 2.1 The underlying mechanisms

Our model is based on an SIR scheme, but in which contact rates depend on the behavioural patterns adopted across the population. Specifically, we assume that the total population (say, $N_{tot}$) is divided into the following disjoint compartments [42]:

- susceptible adopting the normal behaviour ($x = S_n$): healthy individuals subject to the baseline risk of contracting the disease. Members of this class do not manifest a spontaneous behavioural response to the epidemics;
- susceptible adopting the altered behaviour ($x = S_a$): healthy individuals subject to a reduced risk of contracting the disease. Members of this class spontaneously reduce their social contacts as a defensive response to the epidemics;
- infectious ($x = I$): individuals infected by the disease who can transmit the virus to others;
- recovered ($x = R$): individuals who recovered after the infectious period.

Since we model single epidemic outbreaks, the vital dynamics of the population is not taken into account. Hence, the set of possible labels targeting the agents is given by

$$\mathcal{X} = \{S_n, S_a, I, R\}.$$

Individuals from any of the two susceptible classes can become infected by interaction with an infectious individual, $I$, with the difference that the $S_a$'s are exposed to a lower risk with respect to the $S_n$'s. A susceptible individual may be persuaded to change his/her contact pattern by a member of the alternative group, depending on the personal cost–benefit assessment in the mutual interaction. Once infected, the individual experiences an infectious period and, eventually, recovers by moving to the $R$ class. For the sake of simplicity, infectious individuals all conform to a given pattern of social contacts; this can be interpreted as an effect of their status, independently of the state of the epidemics [42].

It follows that the non–vanishing *transitions* between compartments are: $S_n \to I$, $S_a \to I$, $S_n \to S_a$, $S_a \to S_n$, $I \to R$, where the notation $(j \to i)$ indicates the transfer from compartment $j$ to compartment $i$.

The transitions related to the selection of the social behaviour ($S_n \to S_a$, $S_a \to S_n$) are driven by an imitation game dynamics [42, 49]. Let us denote by $\pi_n$, $\pi_a$ the *net* costs at population level induced by the normal and by the altered pattern of social contacts, respectively (the costs associated to, e.g., disease safety, social and psychological well–being, ...). We assume that these

costs depend on the disease status in the community and, hence, they evolve in time according to the epidemic dynamics. Note that, if considered with opposite sign, then $\pi_n$, $\pi_a$ can be seen in the economic jargon as the *payoffs* for the corresponding strategies, i.e. $-\pi_n$, $-\pi_a$, respectively [42].

**Remark 1.** *The imitation game dynamics can be equivalently inferred by employing a statistical physics–oriented approach [7, 12, 49]. In such a case, the basic concept is that the switching between strategies is ruled by a 'double contagion' of ideas between members of the alternative groups. Here, we deliberately choose to follow the classical economics–oriented approach since we consider it more suitable to the role and the interpretation attributed to the microscopic quantity $c \in \mathcal{C}$.*

As done in the model [42], the $S_n$ and $S_a$ individuals are assumed to decide whether to change or preserve their social behaviour on the basis of the composition of the pool of susceptible individuals with whom they interact by exchanging ideas. In other words, in the process of decision making, susceptible individuals take into account only the ideas of those who have not yet contracted the disease. During an interaction between agents coming from alternative groups, the probability for the $S_n$ individual of switching to the $S_a$ compartment is non–null only if

$$\text{sign}\left(c_h^{\text{own}}\pi_n - c_h^{\text{opp}}\pi_a\right) = \text{sign}\left(\frac{c_h^{\text{own}}}{c_h^{\text{opp}}}\pi_n - \pi_a\right)$$

is positive, where $c_h^{\text{own}}$ [resp. $c_h^{\text{opp}}$] is a non–negative constant representing the weight that the agent under consideration attaches to the net cost of the own strategy, $\pi_n$ [resp. of the opposite strategy, $\pi_a$]. The degree of flexibility of opinion of the agent under consideration is represented by the ratio

$$c_h = \frac{c_h^{\text{own}}}{c_h^{\text{opp}}} \geq 0,$$

that is the relative weight of the net cost $\pi_n$ over the weight of the net cost $\pi_a$. More precisely, if $c_h > 1$ [resp. $c_h < 1$], then the agent tends to overestimate [resp. underestimate] the cost of adopting the own behavioural pattern with respect to that of adopting the alternative behavioural pattern and, as a consequence, is more inclined [resp. less inclined] to switch to the $S_a$ compartment. Analogously, the probability for the $S_a$ individual involved in the interaction of switching to the $S_n$ compartment is non–null only if the difference $c_k\pi_a - \pi_n$ is positive, where $c_k = c_k^{\text{own}}/c_k^{\text{opp}} \in \mathcal{C}$ is his/her corresponding degree of flexibility of opinion. Note that in such a case $c_k^{\text{own}}$ [resp. $c_k^{\text{opp}}$] is the weight that the agent under consideration attaches to $\pi_a$ [resp. to $\pi_n$].

Generally speaking, $c_h$, $c_k$ are the specific values assumed by the microscopic quantity $c \in \mathcal{C}$ when targeting the two considered agents. Note that, besides the trivial case that $c$ is the same for all the agents, the introduction of an individual–based weight in the cost–benefit balance 'breaks' two classical assumptions of the imitation game dynamics: i) the indistinguishability among members of the same group in terms of the perception of the costs; ii) the 'symmetry' between the probabilities of switching from a strategy to the alternative one during an interaction. Indeed, in the classical framework, the probabilities of switching for the two involved agents are simply proportional to the positive and to the negative part of the difference of the net costs of the strategies, so that when one if positive the other one is null and vice versa. In our new framework, there is no relationship between the two probabilities, so introducing the possibility that they are both null or both positive during the interaction, namely that neither or both of the involved agents may change compartment.

For the sake of simplicity, we consider here

$$\mathcal{C} = [0, 2],$$

so that agents with fully balanced opinions are targeted by $c = 1$, more intransigent agents are targeted by a $c \in [0, 1)$ and agents with more volatile opinions are targeted by a $c \in (1, 2]$. We

highlight that the derivation of the model can be straightforwardly adapted to different choices of $\mathcal{C}$.

We assume that $c \in \mathcal{C}$ is continuous and distributed with a fixed probability density function $k_i$, $i \in \mathcal{X}$ (similarly to what done in the paper [30], where an opinion exchange is described and $c$ is the number of social connections of the individual). A possible simple choice for $k_i(c)$, the initial fixed probability density function of the degree of flexibility of opinion for the agents in the $i$th compartment, $i \in \mathcal{X}$, is

$$k_i(c) = \chi_{\{0 \le c \le 1\}} q_i + \chi_{\{1 < c \le 2\}}(1 - q_i),$$

where $\chi$ is the Heaviside function, $q_i \in [0, 1]$ is the probability for an agent of having a degree of flexibility smaller than or equal to 1, so that, conversely, $1 - q_i$ is the probability of having a degree of flexibility larger than 1. As a consequence, the initial distribution function of the degree of flexibility of opinion for the agents in the $i$th compartment, $i \in \mathcal{X}$, is

$$f_i(c, 0) = \rho_i(0) k_i(c).$$

This determines the initial average flexibilities of opinion:

$$m_i(0) = \int_{\mathcal{C}} c\, k_i(c)\, dc, \quad i \in \mathcal{X}.$$

**Remark 2.** *The concept of* volatility *of opinion is intended here as a trait of the individual personality. It is different from the concept of* speed *of the imitation game, that refers to the frequency at which the information are exchanged through interactions. This last aspect, increasingly important in the era of social media, has been addressed in some game theoretical epidemic models (see, e.g., [12, 42]) by introducing a faster time scale for the imitation game dynamics with respect to the disease transmission.*

## 2.2 A microscopic stochastic model

In this section, we propose a microscopic stochastic process implementing the modelling assumptions defined so far.

Let us consider a representative agent of the system characterized at time $t$ by the pair of random variables $(X_t, C_t) \in \mathcal{X} \times \mathcal{C}$, where $X_t$ is the label denoting the compartment to which the agent belongs and $C_t$ is his/her degree of flexibility of opinion at time $t$. The joint probability density distribution of such a pair is $f(x, c, t)$, as given in (1).

We need to express the transitions as a stochastic process for $X_t$, from which we derive a kinetic equation for $f$. As already mentioned, the non–vanishing transitions between compartments are: $S_n \to I$, $S_a \to I$, $S_n \to S_a$, $S_a \to S_n$, $I \to R$. The first four transitions happen as a consequence of interactions among individuals. As we embrace the spirit of kinetic theory (already adopted in the epidemic model [13]), we consider *binary interactions*, i.e. interactions between two individuals. Specifically, the transitions $S_n \to I$, $S_a \to I$ happen as a consequence of physical interactions between susceptible individuals and infectious individuals. The transitions $S_n \to S_a$, $S_a \to S_n$ happen as a consequence of binary interactions between susceptible individuals adopting alternative behavioural strategies who exchange their ideas; since the decision whether to change or preserve the social behaviour is based only on the composition of the susceptible population, we assume that the information exchanged through interactions is 'normalized' to the actual size of the susceptible compartment. The transition $I \to R$ follows from the physiological progression of the illness and, in the jargon of kinetic theory, it is commonly named an *autonomous process*, since it is not due to binary interactions. In the current formalism, the transitions between compartments are generally named *label switch processes* and they are modelled as Markov–jump processes, as the new label of the agent only depends on the previous one. Then, the processes of interest are:

**P1** the binary interaction process between susceptible individuals and infectious individuals leading to the spread of the contagion in the population ($S_n \to I$, $S_a \to I$);

**P2** the binary interaction process between susceptible individuals with normal and with altered behaviour leading to a switch in the choice of the adopted strategy ($S_n \to S_a$, $S_a \to S_n$);

**P3** the autonomous process related to the physiological recovery from the disease for infected individuals ($I \to R$).

Hence, during a sufficiently small time interval $\Delta t > 0$, the label $X_t$ of the agent may change or not according to whether a binary interaction or an autonomous process takes place. The label switch process may be written as a discrete in time stochastic process in the general form

$$X_{t+\Delta t} = (1 - \Theta)X_t + \Theta J_t, \tag{5}$$

where $\Theta$ is a Bernoulli random variable with parameter $\mu\Delta t$, being $\mu$ the frequency of the binary interaction or of the autonomous process that causes the label switch, and $J_t$ is the random variable that takes into account the new label, i.e. the new compartment of the agent. As regards the degree of flexibility of opinion, it does not vary in time:

$$C_{t+\Delta t} = C_t. \tag{6}$$

As a consequence, we can introduce a probability density function for the random variable $J_t$ alone

$$P(j; c) = \text{Prob}(J_t = j; C_t), \tag{7}$$

that may depend on the (constant) value of the degree of flexibility of opinion, $C_t = c$. Since the label switch process is described as a Markov–type jump process [32], for each switch $X_t \to J_t$ we introduce appropriate transition probabilities.

**Remark 3.** *If one assumed that the flexibility of opinion of the agents evolves in time, then, in place of (6)–(7), a change of the microscopic state $C_t$ would be described by a proper discrete in time stochastic process and a joint probability density function for the pair of random variables $J_t$ and $C_t$, namely $P(J_t = j, C_t = c)$, would be considered. In the present case, this corresponds to setting $P(J_t = j, C_t = c) = P(J_t = j; C_t)k_{J_t}(C_t = c)$, where $k_{J_t}$ is the probability density function of the initial distribution of the degree of flexibility of opinion in the compartment $J_t$.*

In the following, we characterize the microscopic discrete in time stochastic process (5) in each of the cases P1, P2, P3.

### 2.2.1 Transitions $S_n \to I$, $S_a \to I$

The transition from any of the two susceptible classes to the infectious one is due to a binary interaction between the agent $(X_t, C_t)$ and another agent $(X_t^*, C_t^*)$, where $X_t = S_n$ or $X_t = S_a$ and $X_t^* = I$. This implies that $X_t = S_n$ or $X_t = S_a$ and $J_t = I$ in (5). We denote by $\mu = \lambda_{X_t, X_t^*}$ the frequency of binary interactions. Generally speaking, we can express the probability density function of the random variable $J_t$ as

$$P(j; c) = \int_{\mathcal{X}^2} \int_{\mathcal{C}} P(j|x, x_*; c) f_2(x, x_*, c, c_*, t) dc_* dx dx_*,$$

where $f_2(x, x_*, c, c_*, t)$ is the joint probability density function of the interacting agents $(x, c)$, $(x_*, c_*)$, so that $f_2(x, x_*, c, c_*, t)dxdx_*dcdc_*$ is the number of pairs of agents having label $x$, $x_*$ and degree of flexibility of opinion in $[c, c + dc]$, $[c_*, c_* + dc_*]$, respectively. As usually done in kinetic theory, we assume that the *propagation of chaos* holds, i.e. the pairs of random variables

$(X_t, C_t)$ and $(X_t^*, C_t^*)$ are stochastic independent since the agents retain no memory of previous interactions. This allows the simplification

$$f_2(x, x_*, c, c_*, t) = f(x, c, t) f(x_*, c_*, t).$$

The conditional probability $P(j|x, x_*; c)$ describes the probability that an individual characterized by the pair $(X_t = x, c)$ moves to the compartment $J_t = j$ when interacting with $X_t^* = x_*$. This process may, in general, depend on both the quantities $c$, $c_*$, associated to the two agents. In the present work, we assume that it may depend solely on $c$, namely the microscopic state of the agent whose label is switching (such a dependence is accounted for in the process P2).

Since we are considering the case that $X_t = S_a$ or $X_t = S_n$, $X_t^* = I$ and $J_t = I$, it follows that the probability density functions to take into account are

$$P(I|S_i, I; c) = \beta_{d,i} \in [0, 1], \quad i \in \{n, a\}.$$

### 2.2.2 Transitions $S_n \to S_a$, $S_a \to S_n$

The switching between the normal and the altered pattern of social contacts happens as a consequence of binary interactions between susceptible individuals adopting alternative strategies. Therefore, we consider $X_t = S_a$, $X_t^* = J_t = S_n$ or vice versa in (5). Again, we denote by $\mu = \lambda_{X_t, X_t^*}$ the frequency of binary interactions. For symmetry considerations, we set

$$\lambda_{S_n, S_a} = \lambda_{S_a, S_n} = \lambda.$$

Generally speaking, for this kind of process, the probability density function may be expressed as

$$P(j; c) = \int_{\mathcal{X}^2} \int_{\mathcal{C}} P(j|x, x_*; c) \frac{f_2(x, x_*, c, c_*, t)}{\rho_{S_n}(t) + \rho_{S_a}(t)} dc_* dx dx_*.$$

The presence of the ratio $f_2(x, x_*, c, c_*, t)/(\rho_{S_n}(t) + \rho_{S_a}(t))$ is related to the assumption that the process of exchange of ideas exclusively involve susceptible individuals who, then, take their decision on the basis of the composition of the susceptible population (that has density $\rho_{S_n} + \rho_{S_a}$), i.e. by looking at the *fraction* of individuals adopting the alternative strategy with respect to the total susceptible sample [42]. Note that the quantity in the denominator, $\rho_{S_n} + \rho_{S_a}$, is non–vanishing since it is proved that in SIR–like epidemic models the susceptible compartment cannot completely empty [25].

The conditional probability $P(j|x, x_*; c)$ describes again the probability that an individual characterized by the pair $(x, c)$ moves to the compartment $J_t = j$ given a binary interaction with an individual characterized by the pair $(x_*, c_*)$. Specifically, during the interaction between an agent $(S_i, c)$ and an agent $(S_j, c_*)$, with $i, j \in \{n, a\}$, $i \neq j$, the former may enter the compartment of the latter depending on the individual cost–benefit balance. The agent $(S_i, c)$ evaluates the quantity

$$c\pi_i(t) - \pi_j(t), \tag{8}$$

that is the difference between the net cost associated to the own behavioural strategy at time $t$, $\pi_i$, and the net cost associated to the alternative behavioural strategy at time $t$, $\pi_j$, by assigning to the first one the relative weight $c$ with respect to the second one. If the difference (8) is positive, then there is a chance that the agent changes compartment (the alternative behavioural strategy is perceived as more convenient than the own one); otherwise, if the difference (8) is negative, then the agent remains in place (the alternative behavioural strategy is perceived as less convenient than the own one). Therefore, the transition probabilities to take into account are

$$P(S_j|S_i, S_j; c) = g(c\pi_i(t) - \pi_j(t)), \quad i, j \in \{n, a\},\ i \neq j, \tag{9}$$

where $g : \mathbb{R} \to [0,1]$ is a continuous, differentiable and non–decreasing function such that

$$g(y) = 0,\ \forall y \leq 0,\ \text{ and }\ g(y) \in (0,1],\ \forall y > 0. \tag{10}$$

If $c = 1$, then the agent is fully balanced; if $c \in [0,1)$, then the agent is likely to be intransigent; if $c \in (1,2]$, then the agent is likely to have volatile opinions.

As far as the net costs $\pi_n$, $\pi_a$ are concerned, they represent the costs at population level (in terms of both health and social well–being) associated to the adoption of the normal and the altered behavioural strategy, respectively. They evolve in time according to the status of the disease in the community. Without loss of generality, we assume that they are continuous and differentiable functions of the densities of the compartments:

$$\pi_i(t) = \pi_i(\rho_{S_n}(t), \rho_{S_a}(t), \rho_I(t), \rho_R(t)), \quad i \in \{n, a\}. \tag{11}$$

### 2.2.3 Transition $I \to R$

The transfer of an infectious individual to the recovered compartment is due to a physiological process, that is independent of interactions with other individuals. Then, we consider $X_t = I$, $J_t = R$ in (5), and denote by $\mu = \lambda_I^R$ the frequency of the recovery process. In this case, the probability density function of $J_t$ may be simply written as

$$P(j;c) = \int_{\mathcal{X}} P(j|x) f(x,c,t) dx,$$

where $P(j|x)$ is the conditional probability density function of the random variable $J_t$ given $X_t$. Note that, in general, $P(j|x)$ may also depend on the microscopic quantity $c$, but this dependence is here disregarded since the flexibility of opinion of the agent only affects the switch between the classes $S_n$ and $S_a$ (namely, the process P2). Then, the only transition probability to take into account in this case is

$$P(R|I) = \gamma_r \in [0,1].$$

Altogether, we express the discrete–in–time random process as

$$\begin{aligned} X_{t+\Delta t} &= \Xi\left[(1-\Theta_d)X_t + \Theta_d J_t\right] + \Psi\left[(1-\Theta_b)X_t + \Theta_b J_t\right] + \Sigma\left[(1-\Theta_r)X_t + \Theta_r J_t\right], \\ C_{t+\Delta t} &= C_t, \end{aligned} \tag{12}$$

where $\Xi$, $\Psi$ and $\Sigma$ are indicator functions. Specifically, the processes P1, P2, P3 are represented, respectively, by the mutually exclusive cases:

1 $\Xi(x, x_*) = 1$, $\Psi(x, x_*) = 0$, $\Sigma(x, x_*) = 0$, if $(x, x_*) \in \{(S_n, I), (S_a, I)\}$;

2 $\Xi(x, x_*) = 0$, $\Psi(x, x_*) = 1$, $\Sigma(x, x_*) = 0$, if $(x, x_*) \in \{(S_n, S_a), (S_a, S_n)\}$;

3 $\Xi(x, x_*) = 0$, $\Psi(x, x_*) = 0$, $\Sigma(x, x_*) = 1$, if $(x, x_*) \in \{(I, x_*), x_* \in \mathcal{X}\}$.

In (12), the quantities $\Theta_d$, $\Theta_b$, $\Theta_r \in \{0,1\}$ are Bernoulli random variables (of parameter $\mu\Delta t$ with $\mu \in \{\lambda_{S_n,I}, \lambda_{S_a,I}\}$, $\mu = \lambda$, $\mu = \lambda_I^R$, respectively) discriminating whether a label switch takes place (namely, $\Theta_d$, $\Theta_b$, $\Theta_r = 1$) or not (namely, $\Theta_d$, $\Theta_b$, $\Theta_r = 0$) during the time interval $\Delta t$.

## 2.3 Aggregate description: from kinetic to macroscopic equations

Being our final aim the proposal of a macroscopic model, we derive as an intermediate stage a statistical description at the mesoscopic level of our multi–agent system through kinetic equations.

Let $\phi = \phi(x,c)$ be an observable quantity defined on $\mathcal{X}\times\mathcal{C}$. From (12), together with the assumed independence of $\Theta_d$, $\Theta_b$, $\Theta_r$, we obtain that the average variation rate of $\phi$ in the time interval $\Delta t$ satisfies

$$\begin{aligned}\frac{\langle\phi(X_{t+\Delta t},C_{t+\Delta t})\rangle - \langle\phi(X_t,C_t)\rangle}{\Delta t} = \\ \frac{\langle\Xi(1-\mu\Delta t)\phi(X_t,C_t)\rangle + \langle\Xi\mu\Delta t\phi(J_t,C_t)\rangle - \langle\Xi\phi(X_t,C_t)\rangle}{\Delta t} \\ + \frac{\langle\Psi(1-\mu\Delta t)\phi(X_t,C_t)\rangle + \langle\Psi\mu\Delta t\phi(J_t,C_t)\rangle - \langle\Psi\phi(X_t,C_t)\rangle}{\Delta t} \\ + \frac{\langle\Sigma(1-\mu\Delta t)\phi(X_t,C_t)\rangle + \langle\Sigma\mu\Delta t\phi(J_t,C_t)\rangle - \langle\Sigma\phi(X_t,C_t)\rangle}{\Delta t}\end{aligned}$$

where $\langle\cdot\rangle$ indicates the average with respect to the random variables. Whence we deduce the instantaneous time variation of the average of $\phi$ in the limit $\Delta t\to 0^+$ as

$$\begin{aligned}\frac{d}{dt}\langle\phi(X_t,C_t)\rangle = &\langle\Xi\mu\phi(J_t,C_t)\rangle + \langle\Psi\mu\phi(J_t,C_t)\rangle + \langle\Sigma\mu\phi(J_t,C_t)\rangle \\ &- \langle\Xi\mu\phi(X_t,C_t)\rangle - \langle\Psi\mu\phi(X_t,C_t)\rangle - \langle\Sigma\mu\phi(X_t,C_t)\rangle.\end{aligned} \tag{13}$$

The first [resp. last] three terms on the right–hand side of (13) are the gain [resp. loss] terms related, respectively, to the label switches P1, P2, P3. Generally speaking, the gain terms may be written as follows

$$\langle\Xi\mu\phi(J_t,C_t)\rangle = \int_{\mathcal{X}^3}\int_{\mathcal{C}^2}\Xi\lambda_{x,x_*}\phi(j,c)P(j|x,x_*;c)f(x,c,t)f(x_*,c_*,t)dcdc_*dxdx_*dj,$$

$$\langle\Psi\mu\phi(J_t,C_t)\rangle = \int_{\mathcal{X}^3}\int_{\mathcal{C}^2}\Psi\lambda_{x,x_*}\phi(j,c)P(j|x,x_*;c)f(x,c,t)\frac{f(x_*,c_*,t)}{\rho_{S_n}(t)+\rho_{S_a}(t)}dcdc_*dxdx_*dj,$$

$$\langle\Sigma\mu\phi(J_t,C_t)\rangle = \int_{\mathcal{X}^2}\int_{\mathcal{C}}\Sigma\lambda_x^j\phi(j,c)P(j|x)f(x,c,t)dcdxdj,$$

while the loss terms as follows

$$\langle\Xi\mu\phi(X_t,C_t)\rangle = \int_{\mathcal{X}^3}\int_{\mathcal{C}^2}\Xi\lambda_{x,x_*}\phi(x,c)P(j|x,x_*;c)f(x,c,t)f(x_*,c_*,t)dcdc_*dxdx_*dj,$$

$$\langle\Psi\mu\phi(X_t,C_t)\rangle = \int_{\mathcal{X}^3}\int_{\mathcal{C}^2}\Psi\lambda_{x,x_*}\phi(x,c)P(j|x,x_*;c)f(x,c,t)\frac{f(x_*,c_*,t)}{\rho_{S_n}(t)+\rho_{S_a}(t)}dcdc_*dxdx_*dj,$$

$$\langle\Sigma\mu\phi(X_t,C_t)\rangle = \int_{\mathcal{X}^2}\int_{\mathcal{C}}\Sigma\lambda_x^j\phi(x,c)P(j|x)f(x,c,t)dcdxdj.$$

Then, the kinetic equation for the probability density distribution of the agents, $f(x,c,t)$, specialises as

$$\begin{aligned}&\frac{d}{dt}\int_{\mathcal{X}}\int_{\mathcal{C}}\phi(x,c)f(x,c,t)dcdx = \\ &+\int_{\mathcal{X}^3}\int_{\mathcal{C}^2}\Xi\lambda_{x,x_*}\Big(\phi(j,c)-\phi(x,c)\Big)P(j|x,x_*;c)f(x,c,t)f(x_*,c_*,t)dcdc_*dxdx_*dj \\ &+\int_{\mathcal{X}^3}\int_{\mathcal{C}^2}\Psi\lambda_{x,x_*}\Big(\phi(j,c)-\phi(x,c)\Big)P(j|x,x_*;c)f(x,c,t)\frac{f(x_*,c_*,t)}{\rho_{S_n}(t)+\rho_{S_a}(t)}dcdc_*dxdx_*dj \\ &+\int_{\mathcal{X}^2}\int_{\mathcal{C}}\Sigma\lambda_x^j\Big(\phi(j,c)-\phi(x,c)\Big)P(j|x)f(x,c,t)dcdxdj,\end{aligned}$$

which has to hold for every $\phi : \mathcal{X} \times \mathcal{C} \to \mathbb{R}$. Choosing $\phi(x, c) = \psi(x)\varphi(c)$ with $\psi$ such that $\psi(i) = 1$ for a certain $i \in \mathcal{X}$ and $\psi(x) = 0$ for all $x \in \mathcal{X} \setminus \{i\}$ and considering (1), we can obtain the system of weak equations for the distribution functions of the agents that are in the $i$th compartment, $f_i$'s, $i \in \mathcal{X}$:

- susceptible individuals with normal behaviour ($i = S_n$)

$$\begin{aligned}\frac{d}{dt}\int_{\mathcal{C}} \varphi(c) f_{S_n}(c,t)dc = &-\lambda_{S_n,I}\int_{\mathcal{C}^2} \varphi(c)\beta_{d,n} f_{S_n}(c,t) f_I(c_*,t)dcdc_* \\ &+\lambda\int_{\mathcal{C}^2} \varphi(c) g(c\pi_a(t) - \pi_n(t)) f_{S_a}(c,t)\frac{f_{S_n}(c_*,t)}{\rho_{S_n}(t)+\rho_{S_a}(t)}dcdc_* \\ &-\lambda\int_{\mathcal{C}^2} \varphi(c) g(c\pi_n(t) - \pi_a(t)) f_{S_n}(c,t)\frac{f_{S_a}(c_*,t)}{\rho_{S_n}(t)+\rho_{S_a}(t)}dcdc_*,\end{aligned} \tag{14}$$

- susceptible individuals with altered behaviour ($i = S_a$)

$$\begin{aligned}\frac{d}{dt}\int_{\mathcal{C}} \varphi(c) f_{S_a}(c,t)dc = &-\lambda_{S_a,I}\int_{\mathcal{C}^2} \varphi(c)\beta_{d,a} f_{S_a}(c,t) f_I(c_*,t)dcdc_* \\ &+\lambda\int_{\mathcal{C}^2} \varphi(c) g(c\pi_n(t) - \pi_a(t)) f_{S_n}(c,t)\frac{f_{S_a}(c_*,t)}{\rho_{S_n}(t)+\rho_{S_a}(t)}dcdc_* \\ &-\lambda\int_{\mathcal{C}^2} \varphi(c) g(c\pi_a(t) - \pi_n(t)) f_{S_a}(c,t)\frac{f_{S_n}(c_*,t)}{\rho_{S_n}(t)+\rho_{S_a}(t)}dcdc_*,\end{aligned} \tag{15}$$

- infectious individuals ($i = I$)

$$\begin{aligned}\frac{d}{dt}\int_{\mathcal{C}} \varphi(c) f_I(c,t)dc = &\lambda_{S_n,I}\int_{\mathcal{C}^2} \varphi(c)\beta_{d,n} f_I(c,t) f_{S_n}(c_*,t)dcdc_* \\ &+\lambda_{S_a,I}\int_{\mathcal{C}^2} \varphi(c)\beta_{d,a} f_I(c,t) f_{S_a}(c_*,t)dcdc_* \\ &-\lambda_I^R\int_{\mathcal{C}} \varphi(c)\gamma_r f_I(c,t)dc,\end{aligned} \tag{16}$$

- recovered individuals ($i = R$)

$$\frac{d}{dt}\int_{\mathcal{C}} \varphi(c) f_R(c,t)dc = \lambda_I^R\int_{\mathcal{C}} \varphi(c)\gamma_r f_I(c,t)dc. \tag{17}$$

Equations (14)–(17) have to hold for every $\varphi : \mathcal{C} \to \mathbb{R}$.

In order to obtain the evolution equations for the macroscopic densities (3) [resp. the average flexibilities of opinion (4)] of each compartment, we set

$$\varphi(c) = c^\alpha,$$

with $\alpha = 0$ [resp. $\alpha = 1$], in (14)–(17). After some rearrangement, we obtain an exact closed system without the need of other assumptions.

The macroscopic model is given by the following non–linear ODEs:

$$
\begin{aligned}
\frac{d}{dt}\rho_{S_n}(t) &= \lambda\left[g(m_{S_a}(t)\pi_a(t)-\pi_n(t))-g(m_{S_n}(t)\pi_n(t)-\pi_a(t))\right]\frac{\rho_{S_n}(t)}{\rho_{S_n}(t)+\rho_{S_a}(t)}\rho_{S_a}(t)\\
&\quad-\lambda_{S_n,I}\beta_{d,n}\rho_{S_n}(t)\rho_I(t),\\
\frac{d}{dt}\rho_{S_a}(t) &= \lambda\left[g(m_{S_n}(t)\pi_n(t)-\pi_a(t))-g(m_{S_a}(t)\pi_a(t)-\pi_n(t))\right]\frac{\rho_{S_n}(t)}{\rho_{S_n}(t)+\rho_{S_a}(t)}\rho_{S_a}(t)\\
&\quad-\lambda_{S_a,I}\beta_{d,a}\rho_{S_a}(t)\rho_I(t),\\
\frac{d}{dt}\rho_I(t) &= \left(\lambda_{S_n,I}\beta_{d,n}\rho_{S_n}(t)+\lambda_{S_a,I}\beta_{d,a}\rho_{S_a}(t)\right)\rho_I(t)-\lambda_R^I\gamma_r\rho_I(t),\\
\frac{d}{dt}\rho_R(t) &= \lambda_R^I\gamma_r\rho_I(t),\\
\frac{d}{dt}m_{S_n}(t) &= \lambda g(m_{S_a}(t)\pi_a(t)-\pi_n(t))(m_{S_a}(t)-m_{S_n}(t))\frac{\rho_{S_a}(t)}{\rho_{S_n}(t)+\rho_{S_a}(t)},\\
\frac{d}{dt}m_{S_a}(t) &= \lambda g(m_{S_n}(t)\pi_n(t)-\pi_a(t))(m_{S_n}(t)-m_{S_a}(t))\frac{\rho_{S_n}(t)}{\rho_{S_n}(t)+\rho_{S_a}(t)},\\
\frac{d}{dt}m_I(t) &= \left(\lambda_{S_n,I}\beta_{d,n}\rho_{S_n}(t)+\lambda_{S_a,I}\beta_{d,a}\rho_{S_a}(t)\right)m_I(t)-\lambda_R^I\gamma_r m_I(t),\\
\frac{d}{dt}m_R(t) &= \lambda_R^I\gamma_r m_I(t).
\end{aligned}
\tag{18}
$$

In the following sections, we investigate the basic properties and the numerical solutions of the model (18) for some specific functional shapes of the net costs of the behavioural strategies $\pi_n$, $\pi_a$, and of the conditional probability $g$.

# 3 Functional shapes and basic properties

In this section, we provide some basic properties of model (18) given a specific choice of the functions $\pi_n$, $\pi_a$ and $g$. Precisely:

- *Net costs of the behavioural strategies.*
  Analogously to what done by Poletti *et al.* [42], we assume that the net costs at population level associated to the two behavioural strategies (see (11)) are linear–affine functions of the density of infectious individuals, $\rho_I$ (the disease *prevalence*). We set

$$
\pi_n(t)=\nu_n\rho_I(t),\ \ \pi_a(t)=\nu_a\rho_I(t)+k,
\tag{19}
$$

  where: $\nu_n>\nu_a>0$ represent the perceived risks of infection for $S_n$ individuals and for $S_a$ individuals, respectively (they may be related to, e.g., the risk of developing symptoms); $k>0$ represents the extra cost induced by the altered behaviour. Indeed, in terms of disease–safety, the cost induced by the normal behavioural pattern is higher than that induced by the altered behavioural pattern, since $S_n$ individuals are exposed to a higher risk of infection ($\nu_n>\nu_a$). However, from the social and psychological point of view, $S_a$ individuals suffer the reduction of contacts with people (less travelling, less visiting friends and relatives, ...). This disadvantage can be thought as an extra fixed cost $k$.

- *Switching rates from a social behaviour to the alternative one.*
  The function $g$ represents the conditional probability that a susceptible individual changes behavioural strategy during an interaction with a member of the alternative group (see (9)). Given that $\lambda$ is the frequency of these interactions and $g$ must satisfy the properties (10), the

most natural and simple choice appears us to set $\lambda g(y) = y^+$, where the apices $^+$ indicates the positive part of $y$. This yields

$$\begin{aligned} g_{na}(t) &= \lambda g(m_{S_n}(t)\pi_n(t) - \pi_a(t)) = (m_{S_n}(t)\pi_n(t) - \pi_a(t))^+, \\ g_{an}(t) &= \lambda g(m_{S_a}(t)\pi_a(t) - \pi_n(t)) = (m_{S_a}(t)\pi_a(t) - \pi_n(t))^+, \end{aligned} \tag{20}$$

where

$$\begin{aligned} (m_{S_n}(t)\pi_n(t) - \pi_a(t))^+ &= \max\{0, m_{S_n}(t)\pi_n(t) - \pi_a(t)\}, \\ (m_{S_a}(t)\pi_a(t) - \pi_n(t))^+ &= \max\{0, m_{S_a}(t)\pi_a(t) - \pi_n(t)\}, \end{aligned}$$

being $g_{na}$ [resp. $g_{an}$] the switching rate of susceptible individuals from the normal [resp. altered] to the altered [resp. normal] social behaviour. By substituting the explicit expressions of $\pi_n$, $\pi_a$, as given in (19), in the formula (20), one finally obtains

$$\begin{aligned} g_{na}(t) &= \left((\nu_n m_{S_n}(t) - \nu_a)\rho_I(t) - k\right)^+, \\ g_{an}(t) &= \left(k m_{S_a}(t) - (\nu_n - \nu_a m_{S_a}(t))\rho_I(t)\right)^+. \end{aligned} \tag{21}$$

Also, we note that in model (18) the disease transmission rates for $S_n$ individuals and for $S_a$ individuals are represented by

$$\beta_i = \lambda_{S_i,I}\beta_{d,i}, \quad i \in \{n, a\},$$

respectively, and the rate of recovery from the disease is represented by

$$\gamma = \lambda_R^I \gamma_r.$$

As a result of the above choices and by observing that the differential equations for $\rho_{S_n}$, $\rho_{S_a}$, $\rho_I$, $m_{S_n}$, $m_{S_a}$, do not depend on $\rho_R$, $m_I$, $m_R$, the model (18) reduces to

$$\frac{d}{dt}\rho_{S_n}(t) = (g_{an}(t) - g_{na}(t))\,\frac{\rho_{S_n}(t)}{\rho_{S_n}(t) + \rho_{S_a}(t)}\rho_{S_a}(t) - \beta_n \rho_{S_n}(t)\rho_I(t), \tag{22a}$$

$$\frac{d}{dt}\rho_{S_a}(t) = (g_{na}(t) - g_{an}(t))\,\frac{\rho_{S_n}(t)}{\rho_{S_n}(t) + \rho_{S_a}(t)}\rho_{S_a}(t) - \beta_a \rho_{S_a}(t)\rho_I(t), \tag{22b}$$

$$\frac{d}{dt}\rho_I(t) = (\beta_n \rho_{S_n}(t) + \beta_a \rho_{S_a}(t))\,\rho_I(t) - \gamma\rho_I(t), \tag{22c}$$

$$\frac{d}{dt}m_{S_n}(t) = g_{an}(t)(m_{S_a}(t) - m_{S_n}(t))\frac{\rho_{S_a}(t)}{\rho_{S_n}(t) + \rho_{S_a}(t)}, \tag{22d}$$

$$\frac{d}{dt}m_{S_a}(t) = g_{na}(t)(m_{S_n}(t) - m_{S_a}(t))\frac{\rho_{S_n}(t)}{\rho_{S_n}(t) + \rho_{S_a}(t)}, \tag{22e}$$

with $g_{na}$, $g_{an}$ given in (21), to which we associate the following initial conditions

$$\rho_{S_i}(0) = \rho_{S_i,0} > 0, \;\; \rho_I(0) = \rho_{I,0} > 0, \;\; m_{S_i}(0) = m_{S_i,0} \geq 0, \;\; i \in \{n, a\}. \tag{23}$$

Given the aspect of the equations in (22), it is useful to introduce the quantity

$$p(t) = \frac{\rho_{S_n}(t)}{\rho_{S_n}(t) + \rho_{S_a}(t)}, \tag{24}$$

that is the fraction of susceptible individuals with normal behaviour with respect to the total susceptible population. The differential equation for $p$ can be simply obtained from (22): it is given by

$$\frac{d}{dt}p(t) = (g_{an}(t) - g_{na}(t))\,p(t)(1 - p(t)) - (\beta_n - \beta_a)\rho_I(t)p(t)(1 - p(t)). \tag{25}$$

The dynamics of $p$ will be object of some numerical investigations performed in Section 5.

It is straightforward to verify that the region

$$\mathcal{D} = \left\{(\rho_{S_n}(t), \rho_{S_a}(t), \rho_I(t), m_{S_n}(t), m_{S_a}(t)) \in [0,1]^3 \times [0,2]^2 \ | 0 < \rho_{S_n}(t) + \rho_{S_a}(t) + \rho_I(t) \leq 1 \right\}$$

with initial conditions in (23) is positively invariant for model (22), namely any solution of (22) starting in $\mathcal{D}$ remains in $\mathcal{D}$ for all $t \geq 0$. This ensures that the model is mathematically and epidemiologically well–posed.

Model (22) is an SIR–like epidemic model where the demographic dynamics of the population is not taken into account. As very well–known (starting from the seminal paper by Kermack and McKendrick [25]), this class of models admits only *disease–free* equilibria, i.e. stationary states characterized by the absence of the infection. Let us denote by

$$E = (\rho_{S_n}^\infty, \rho_{S_a}^\infty, \rho_I^\infty, m_{S_n}^\infty, m_{S_a}^\infty), \tag{26}$$

the generic equilibrium of model (22), namely the solutions of the algebraic system obtained by setting the right–hand side of (22) equal to zero. As expected, from the equation (22c) it is necessarily $\rho_I^\infty = 0$. Also, at the equilibrium the switching rates $g_{na}$ and $g_{an}$ reduce to $g_{na} = (-k)^+ = 0$ and $g_{an} = (k m_{S_a}^\infty)^+ = k m_{S_a}^\infty$. Then, from the equations (22a)–(22b)–(22d) it follows that at least one of the conditions

$$\rho_{S_a}^\infty = 0, \ m_{S_a}^\infty = 0$$

holds.

**Remark 4.** *In the hypothetical case that all the agents have the same degree of flexibility of opinion, $c$, the averages $m_{S_n} = m_{S_a}$ are equal to their initial value $m_{S_n,0} = m_{S_a,0}$ for all the time, so playing the role of a constant in the individual cost–benefit balances encoded in the equations (22a)–(22b). In particular, if $c \equiv 1$, namely all the agents have fully balanced opinions, then we retrieve the model in the paper [42] as a special case of (22).*

# 4 Numerical investigations

In this section, we numerically investigate the transient and the asymptotic behaviour of the macroscopic model (22). We previously verified the accordance between the numerical solutions of the particle model (12) and those of the macroscopic model (22) that was formally derived by the former. An exemplary comparison is reported in the Appendix A.

Our aim is to qualitatively assess the impact of the flexibility of opinion of susceptible individuals on the switching between the normal and the altered patterns of social contacts. Namely, we want to stress the role of the relative weight $c$ (assimilated to the averages $m_{S_n}$ and $m_{S_a}$ in the macroscopic equations (22)) in the individual cost–benefit balance between the two social behaviours, and the ensuing consequences on the epidemic dynamics. To this end, we have implemented the modelling assumptions by Poletti *et al.* [42] for what concerns the net costs associated with the two behavioural strategies, $\pi_n$, $\pi_a$, that are given in (19). This allows us to derive the model in the paper [42] as a special case of the macroscopic model (22) (see Remark 4), so making direct comparisons between the corresponding outcomes possible.

Since our investigations are purely qualitative, the values assigned to the epidemiological and social parameters do not address a specific disease and/or spatial area. They refer to generic epidemic outbreaks for which containment strategies rely on the social distancing among individuals, as typically happens for new emerging infectious diseases (e.g., 2003–2004 SARS outbreak [50], 2014–2016 Western African Ebola virus epidemics [9], the first phase of the ongoing COVID–19 pandemic [51]), and for constantly evolving viruses (e.g., the seasonal flu).

All the parameters of the model as well as their baseline values are reported in Table 1.

| Notation | Description | Baseline value |
|---|---|---|
| $\beta_n$ | Transmission rate for susceptible individuals with normal behaviour | 0.28 days$^{-1}$ |
| $\beta_a$ | Transmission rate for susceptible individuals with altered behaviour | 0.08 days$^{-1}$ |
| $\gamma$ | Recovery rate | 1/7 days$^{-1}$ |
| $\nu_n$ | Perceived risk of infection for $S_n$ | 10 – 40$\beta_n$ |
| $\nu_a$ | Perceived risk of infection for $S_a$ | 10 – 40$\beta_a$ |
| $k$ | Extra cost induced by the altered behaviour | 2.5 $\cdot 10^{-2}$ days$^{-1}$ |
| $t_f$ | Time horizon | 15 – 60 months |
| $N_{tot}$ | Total population | $10^6$ |
| $\rho_{S_n,0}$ | Initial density of susceptible individuals with normal behaviour | $0.99(1-\rho_{I,0})$ |
| $\rho_{S_a,0}$ | Initial density of susceptible individuals with altered behaviour | $0.01(1-\rho_{I,0})$ |
| $\rho_{I,0}$ | Initial density of infectious individuals | $1/N_{tot}$ |
| $m_{S_n,0}$ | Initial average flexibility of opinion for $S_n$ | [0,2] |
| $m_{S_a,0}$ | Initial average flexibility of opinion for $S_a$ | [0,2] |

Table 1: List of model parameters and initial conditions, with corresponding description and baseline value.

We assume a population of $N_{tot} = 10^6$ individuals, representing, for example, the inhabitants of a European metropolis. The baseline transmission rate of the disease is set to $\beta_n = 0.28$ days$^{-1}$ and reduces to $\beta_a = 0.08$ days$^{-1}$ for susceptible individuals that adopt the altered pattern of social contacts ($S_a$). We assume a recovery rate of $\gamma = 1/7$ days$^{-1}$, which means that 7 days is the average time for infectious individuals to recover.

The net costs of the social behavioural strategies are linear–affine functions of the density of infectious individuals $\rho_I$, see (19). Specifically, we consider that the perceived risk of infection for susceptible individuals adopting the normal [resp. altered] behaviour is proportional to the corresponding transmission rate: $\nu_n \propto \beta_n$ [resp. $\nu_a \propto \beta_a$]. We distinguish between the following two case studies according to the magnitude of this proportionality:

**C1** the case of relatively high perceived risk of infection: $\nu_i = 40\beta_i$, $i \in \{n, a\}$;

**C2** the case of relatively low perceived risk of infection: $\nu_i = 10\beta_i$, $i \in \{n, a\}$.

In addition, members of $S_a$ compartment pay an extra fixed cost induced by the altered behaviour: $k = 2.5 \cdot 10^{-2}$ days$^{-1}$.

Initial data are set to the beginning of the epidemics, namely we consider a single infectious individual in a totally susceptible population: $\rho_{I,0} = 1/N_{tot}$, $\rho_{S_n,0} + \rho_{S_a,0} = 1 - \rho_{I,0}$. As a consequence, it seems us reasonable to assume that the most part of the susceptible population initially adopts the normal behaviour: $\rho_{S_n,0} = 0.99(\rho_{S_n,0}+\rho_{S_a,0})$. As regards the initial values of the average flexibilities of opinion for $S_n$ and $S_a$ compartments ($m_{S_n,0}$ and $m_{S_a,0}$, respectively), we vary them in the range $\mathcal{C} = [0, 2]$ in order to deeply stress their role on the model outcomes.

Numerical simulations are performed in MATLAB® [36]. We implement the 4th order Runge–Kutta method with constant step size for integrating the system (22). Platform–integrated functions are used for getting the plots.

## 4.1 The case of relatively high perceived risk of infection

We start by investigating the case study C1, where the perceived risk of infection for both $S_n$ and $S_a$ individuals is assumed to be 40 times larger than the corresponding transmission rate.

Fig. 1 displays the numerical solutions of model (22) in the baseline scenario of fully balanced opinions, namely $m_{S_n,0} = m_{S_a,0} = 1$, implying that $m_{S_n} = m_{S_a} \equiv 1$ (see equations (22d)–(22e), and Remark 4). We also sketch the dynamics of the fraction of susceptible individuals with normal

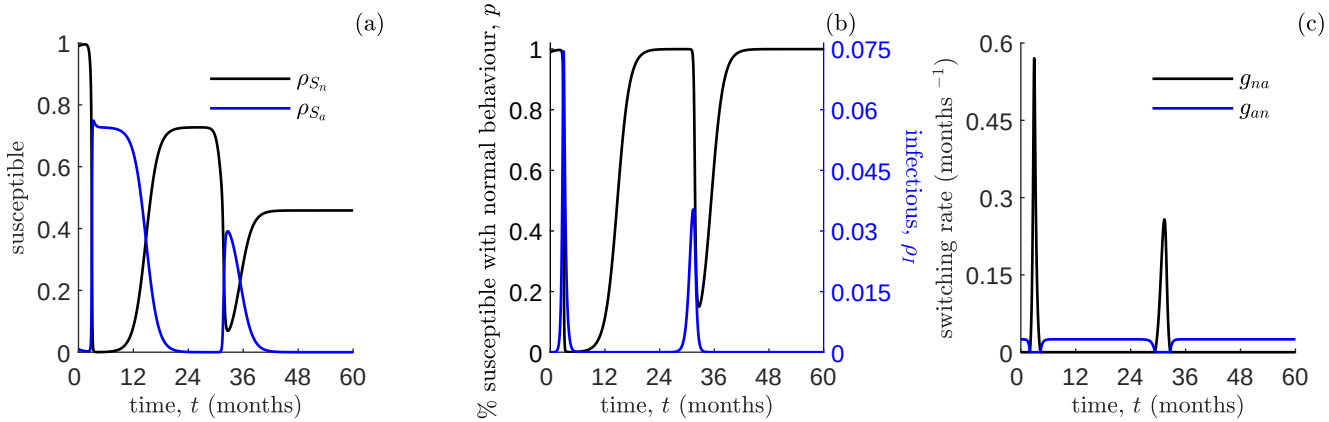


Figure 1: Case study C1. Numerical solutions as predicted by model (22) by assuming $m_{S_n,0} = m_{S_a,0} = 1$. Panel (a): density of susceptible individuals with normal ($\rho_{S_n}$, black line) and altered ($\rho_{S_a}$, blue line) behaviours. Panel (b), left y–axis: fraction of susceptible individuals with normal behaviour, $p$ (black line). Panel (b), right y–axis: density of infectious individuals, $\rho_I$ (blue line). Panel (c): rate of opinion switching from $S_n$ to $S_a$ ($g_{na}$, black line) and from $S_a$ to $S_n$ ($g_{an}$, blue line) compartments. Parameters values and other initial conditions are given in Table 1.

behaviour $p$, defined in (24), that is ruled by the differential equation (25). In such a scenario, the disease dynamics is characterized by two epidemic waves: the first peak occurs about 3 months after the initial time and reaches 7.4% of the total population, the second one follows 28 months after the preceding one and reaches 3.5% of the population (Fig. 1b, blue line). The mutual position of the rates of switching $g_{na}$ and $g_{an}$ reverses before and after that each peak occurs (Fig. 1c): the initial positive $g_{an}$ rate (switching from the $S_a$ to the $S_n$ compartment) vanishes about 1 month before the first [resp. 2 months before the second] peak and then returns positive 40 days [resp. 38 days] after it. Hence, susceptible individuals tend to switch from the altered to the normal behaviour and vice versa, alternately (Fig. 1a). Correspondingly, the fraction of the susceptible population adopting the normal behaviour, $p$, initially very high, alternates between phases of rapid decline with phases of relatively slower rising (Fig. 1b, black line).

In order to investigate the impact of the average relative weights $m_{S_n}$ and $m_{S_a}$ in the individual evaluation about the change of social contact patterns, we compare the results by Fig. 1 with the cases of more or less flexible opinions.

Specifically, we consider the cases of balanced opinions for individuals initially in $S_a$ ($m_{S_a,0} = 1$) and more or less flexible opinions for individuals in $S_n$ ($m_{S_n,0} = 1.5$ or $m_{S_n,0} = 0.5$, respectively). Comparative numerical simulations are given in Fig. 2. From Fig. 2c1–c3 and Fig. 2d, we note that the average flexibilities of opinion for individuals in $S_n$ and $S_a$ compartments tend to rapidly equalize. Indeed, most of the susceptible population initially adopts the normal behaviour ($p(0) = \rho_{S_n,0}/(\rho_{S_n,0} + \rho_{S_a,0}) = 0.99$); when individuals switch en masse from $S_n$ to $S_a$ due to the arrival of the first epidemic peak (namely, when the rate of switching $g_{na}$ passes from being null to positive and overcomes $g_{an}$), they also bring with them their degree of flexibility of opinion. Hence, the ensuing average $m_{S_a}$ is mostly due to individuals coming from the $S_n$ compartment. From Fig. 2a, we observe that, when individuals have on average more volatile opinions (red lines), the switching between different social behaviours is more rapid with respect to the baseline case of fully balanced opinions (black line). As a consequence, the epidemic peaks turn to be more frequent (three instead of two), but generally less high than in the baseline case (Fig. 2b). Conversely, when individuals are on average more intransigent, the switching between the alternative social behaviours occurs more slowly than in the baseline case of fully balanced opinions (Fig. 2a, blue vs. black lines). As a consequence, the epidemic peaks reduce to only one, that however is much higher: it reaches 13.4% of the total population vs. 7.4% by the first peak in the baseline case (Fig. 2b, blue vs.

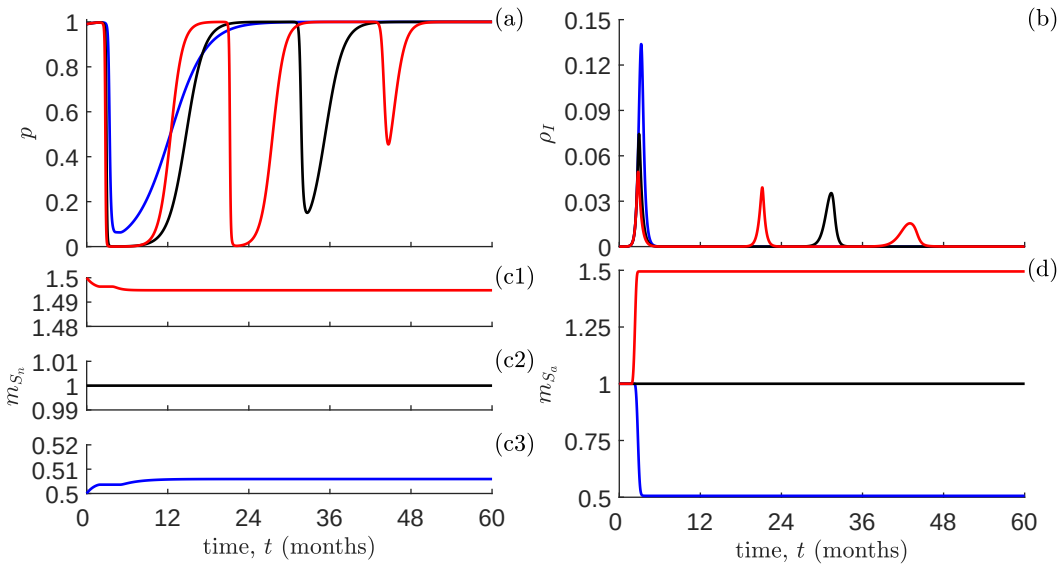


Figure 2: Case study C1. Numerical solutions as predicted by model (22) by assuming $m_{S_a,0} = 1$ and $m_{S_n,0} = 0.5$ (blue lines), $m_{S_n,0} = 1$ (black lines), $m_{S_n,0} = 1.5$ (red lines). Panel (a): fraction of susceptible individuals with normal behaviour, $p$. Panel (b): density of infectious individuals, $\rho_I$. Panels (c1)–(c3): average flexibility of opinion for the $S_n$ compartment, $m_{S_n}$. Panel (d): average flexibility of opinion for the $S_a$ compartment, $m_{S_a}$. Parameters values and other initial conditions are given in Table 1.

| | $m_{S_n,0} = 1$<br>$m_{S_a,0} = 1$ | $m_{S_n,0} = 1.5$<br>$m_{S_a,0} = 1$ | $m_{S_n,0} = 0.5$<br>$m_{S_a,0} = 1$ | $m_{S_n,0} = 1$<br>$m_{S_a,0} = 1.5$ | $m_{S_n,0} = 1$<br>$m_{S_a,0} = 0.5$ |
|---|---|---|---|---|---|
| $\rho_R(t_f)N_{tot}$ | $5.42 \cdot 10^5$ | $5.55 \cdot 10^5$ | $5.70 \cdot 10^5$ | $5.92 \cdot 10^5$ | $4.95 \cdot 10^5$ |

Table 2: Case study C1. Number of recovered individuals at the end of the time horizon, $t_f = 60$ months, as predicted by model (22) for five combinations of the initial average flexibilities of opinion $m_{S_n,0}$ and $m_{S_a,0}$. Parameters values and other initial conditions are given in Table 1.

black lines). As theoretically predicted, we also observe that, at variance with the case of fully balanced opinions where if $g_{na}$ is positive, then $g_{an}$ is null and vice versa (Fig. 1c), in the case of more [resp. less] flexible opinions the switching rates $g_{na}$, $g_{an}$ can be both positive [resp. both null] at the same time (curves not reported here).

In Table 2, we report the values at the end of the time horizon ($t_f = 60$ months) of the number of recovered individuals ($\rho_R(t_f)N_{tot}$) in the different simulation scenarios. Being $\rho_I(t_f) \approx 0$, the quantity $\rho_R(t_f)N_{tot}$ matches the cumulative *incidence* of the disease (i.e., the total number of infections) in the interval $[0, t_f]$. From Table 2, we notice that both the cases of more or less flexible opinions for individuals initially in $S_n$ (second and third columns, respectively) produce globally little more infections than the baseline case (first column): 2.6% and 5.2% more, respectively.

We also investigate the cases of balanced opinions for individuals initially in $S_n$ ($m_{S_n,0} = 1$) and more or less flexible opinions for individuals in $S_a$ ($m_{S_a,0} = 1.5$ or $m_{S_a,0} = 0.5$, respectively). Numerical simulations are given in Fig. 3. Also in such cases, the initial average flexibility of opinion for the $S_n$ compartment remains that predominant in the course of time. Namely, the average $m_{S_a}$ for the compartment of susceptible individuals with altered behaviour gets close to 1 regardless of its initial value (Fig. 3d). With respect to the baseline case of fully balanced opinions (black lines), having $m_{S_a,0}$ different from 1 has the effect of slowing down/speeding up (red/blue lines, respectively) the switching of individuals between the alternative social behaviours (see Fig. 3a). From Fig. 3b we note that, when individuals initially in $S_a$ have more volatile opinions (red lines), the first epidemic peak turns to be 11% higher than in the baseline case, but the second peak is

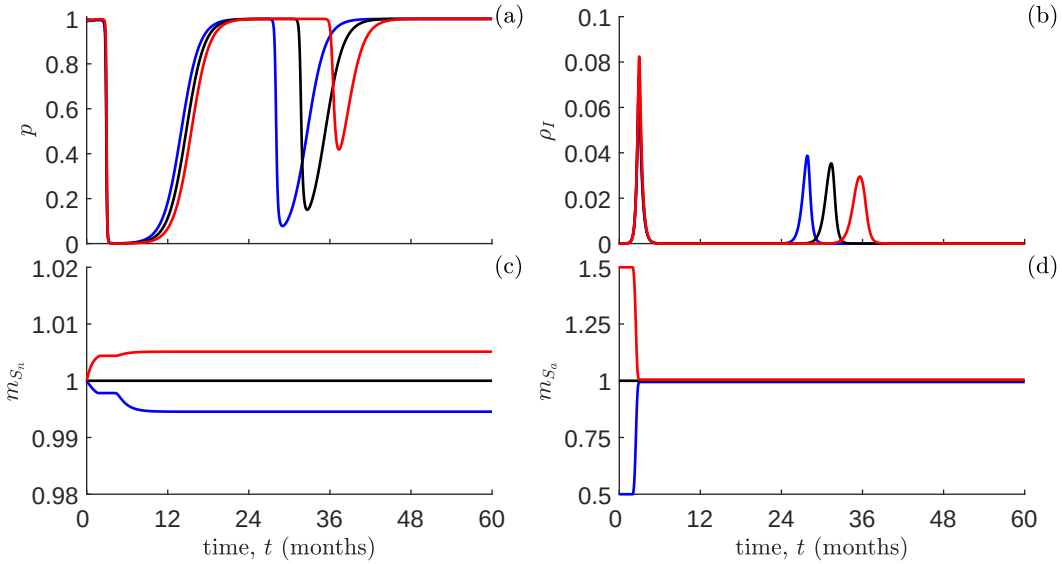


Figure 3: Case study C1. Numerical solutions as predicted by model (22) by assuming $m_{S_n,0} = 1$ and $m_{S_a,0} = 0.5$ (blue lines), $m_{S_a,0} = 1$ (black lines), $m_{S_a,0} = 1.5$ (red lines). Panel (a): fraction of susceptible individuals with normal behaviour, $p$. Panel (b): density of infectious individuals, $\rho_I$. Panels (c): average flexibility of opinion for the $S_n$ compartment, $m_{S_n}$. Panel (d): average flexibility of opinion for the $S_a$ compartment, $m_{S_a}$. Parameters values and other initial conditions are given in Table 1.

delayed by over 4 months and 16% lower than in the case of fully balanced opinions. Globally, the total number of infections (namely, $\rho_R(t_f)N_{tot}$) is about 9% larger than in the baseline case (see Table 2, fourth column). Conversely, when individuals initially in $S_a$ are more intransigent (Fig. 3b, blue lines), the first epidemic peak is 10% lower, but the second peak is anticipated by 3 and a half months and 10% higher than in the baseline case. In such a case, the final cumulative incidence $\rho_R(t_f)N_{tot}$ is 9% smaller than in the case of fully balanced opinions (Table 2, last column).

An overall view of the impact of the flexibility of opinion on the asymptotic behaviour of the model (22) is given in Fig. 4. Specifically, we report the asymptotic (stationary) densities of susceptible individuals with normal and with altered behaviour (namely, the components $\rho^\infty_{S_n}$ and $\rho^\infty_{S_a}$, respectively, of the generic equilibrium (26)), and the corresponding average flexibilities of opinion ($m^\infty_{S_n}$ and $m^\infty_{S_a}$, respectively) by varying the pair $(m_{S_n,0}, m_{S_a,0})$ in the parameter space $[0, 2]^2$. The values are numerically approximated to those at $t = 20$ years after the initial time. Note that from $\rho^\infty_{S_n}$ and $\rho^\infty_{S_a}$ one can also derive the stationary density of the individuals that have been infected and recovered, say

$$\rho^\infty_R = 1 - \rho^\infty_{S_n} - \rho^\infty_{S_a},$$

being the total population constant and the equilibria of model (22) at the disease–free status ($\rho^\infty_I = 0$). From Fig. 4c and Fig. 4d we note that in most of the parameter space $(m_{S_n,0}, m_{S_a,0}) \in [0, 2]^2$ the stationary average flexibility of opinion for both $S_n$ and $S_a$ compartments is close to the average flexibility of individuals initially in $S_n$, $m_{S_n,0}$. An exception is constituted by the cases of very intransigent individuals initially in $S_n$ ($m_{S_n,0} \lesssim 0.35$): in such cases $m_{S_a}$ is asymptotically close to its initial value ($m^\infty_{S_a} \approx m_{S_a,0}$) regardless of $m_{S_n,0}$ (Fig. 4d). The explanation of this outcome relies on the observation that when $m_{S_n,0}$ is very small, the switching rate from the normal to the altered behaviour, $g_{na}$, remains null all long, so that the $S_a$ compartment (whose size is initially very small) can only be further emptied. For the same reason, the stationary number of $S_n$ individuals is rather constant for $m_{S_n,0} \lesssim 0.35$ (Fig. 4a); but it greatly varies for larger $m_{S_n,0}$: the density $\rho^\infty_{S_n}$ ranges from 21.5% to 52.5% of the total population. Interestingly, $\rho^\infty_{S_n}$ does not monotonically change with neither $m_{S_n,0}$ nor $m_{S_a,0}$, but overall it is affected more by

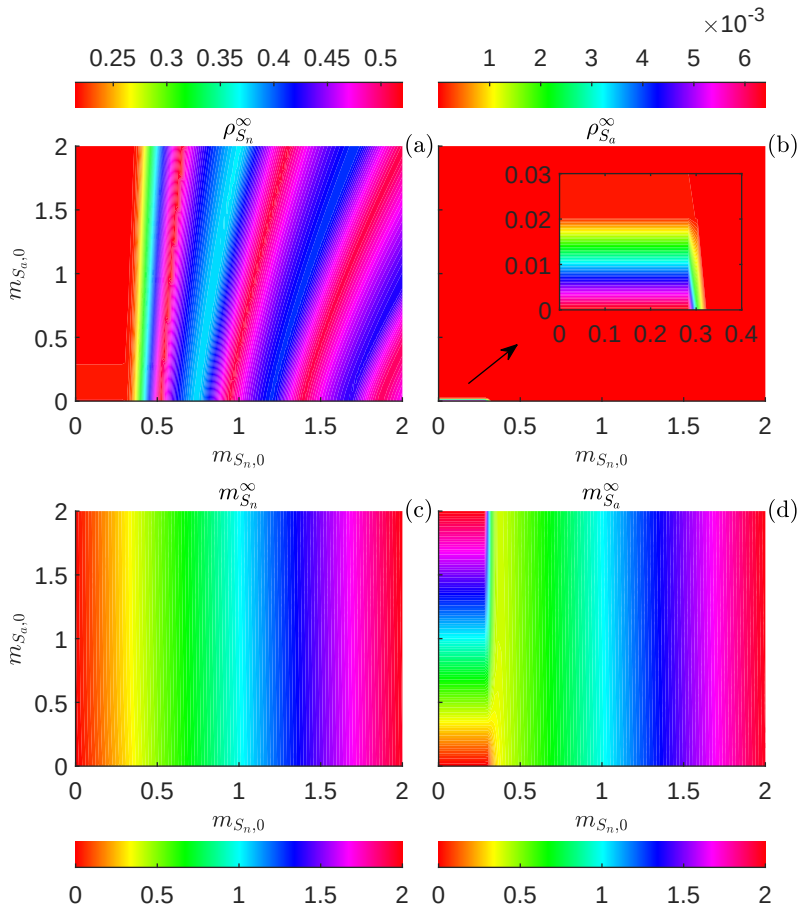


Figure 4: Case study C1. Contour plots of asymptotic quantities as predicted by model (22) versus the initial average flexibility of opinion for the $S_n$ compartment, $m_{S_n,0}$, and for the $S_a$ compartment, $m_{S_a,0}$. Panel (a): stationary density of susceptible with normal behaviour, $\rho^{\infty}_{S_n}$. Panel (b): stationary density of susceptible with altered behaviour, $\rho^{\infty}_{S_a}$; in–box: zoom in for the parameter subspace $(m_{S_n,0}, m_{S_a,0}) \in [0, 0.4] \times [0, 0.03]$. Panel (c): stationary average flexibility of opinion for the $S_n$ compartment, $m^{\infty}_{S_n}$. Panel (d): stationary average flexibility of opinion for the $S_a$ compartment, $m^{\infty}_{S_a}$. Parameters values and other initial conditions are given in Table 1.

variations of $m_{S_n,0}$ with respect to $m_{S_a,0}$. As far as the stationary density of $S_a$ individuals, $\rho^{\infty}_{S_a}$, is concerned, it is almost null in any case (Fig. 4b). This yields the asymptotic cumulative incidence of the disease well approximated by $\rho^{\infty}_R N_{tot} \approx (1 - \rho^{\infty}_{S_n}) N_{tot}$.

**Remark 5.** *As anticipated in Section 2, when a compartment is almost empty, the macroscopic model may not well reproduce the corresponding average flexibility of opinion as predicted by the stochastic particle model. This is due to the inconsistency of average quantities when the number of particles is very small. In that case, the deterministic macroscopic model cannot be justified by means of the law of large numbers and statistical fluctuations should be taken into account. In our case, this happens for example at the end of the time horizon when the size of the $S_a$ compartment is rather null (Fig. 4b). The goodness of the match between the solutions from the macroscopic and the microscopic approaches was deeply stressed in the epidemic model [30], where the time–varying viral load of each agent is tracked. Analogously to what happens in that case, we expect that the* empirical *average flexibility of opinion approaches zero when the compartment is nearly empty.*

## 4.2 The case of relatively low perceived risk of infection

Then, we investigate the case study C2, where the perceived risk of infection for both $S_n$ and $S_a$ individuals is just 10 times larger than the corresponding transmission rate.

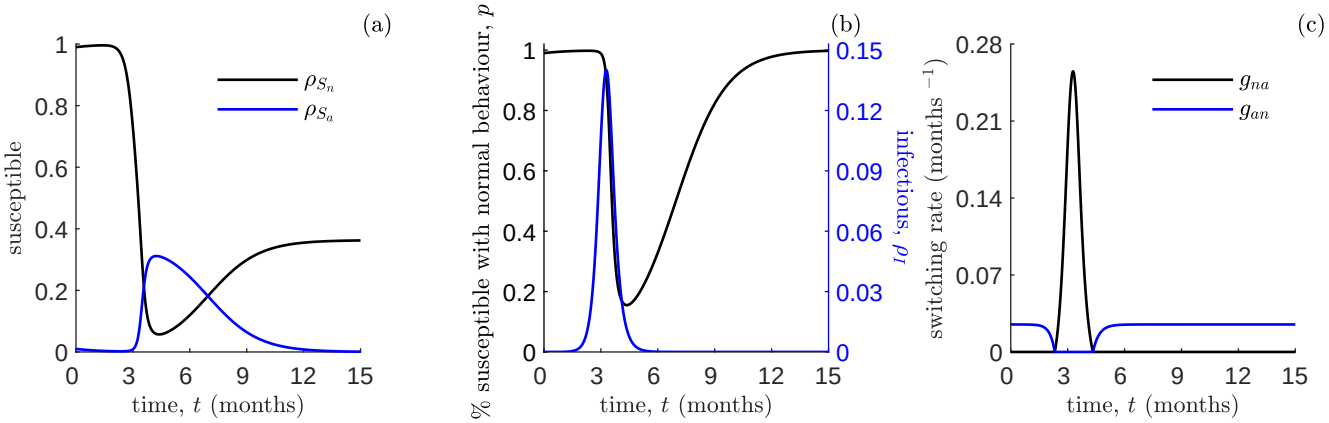


Figure 5: Case study C2. Numerical solutions as predicted by model (22) by assuming $m_{S_n,0} = m_{S_a,0} = 1$. Panel (a): density of susceptible individuals with normal ($\rho_{S_n}$, black line) and altered ($\rho_{S_a}$, blue line) behaviours. Panel (b), left y–axis: fraction of susceptible individuals with normal behaviour, $p$ (black line). Panel (b), right y–axis: density of infectious individuals, $\rho_I$ (blue line). Panel (c): rate of switching from $S_n$ to $S_a$ ($g_{na}$, black line) and from $S_a$ to $S_n$ ($g_{an}$, blue line) compartments. Parameters values and other initial conditions are given in Table 1.

Fig. 5 displays the numerical solutions of model (22) in the baseline scenario of fully balanced opinions, namely $m_{S_n} = m_{S_a} \equiv 1$. At variance with the case study C1 (see Fig. 1), in such a case the disease dynamics is characterized by just one epidemic wave, but much more severe: the peak occurs on the 99th day and reaches 14% of the total population (Fig. 5b, blue line). Indeed, the relatively low perceived risk of infection leads the individuals to be less reactive than in the case C1, and the switching from the $S_n$ to the $S_a$ compartment occurs more slowly (compare the rates of switching $g_{na}$ in Fig. 1c and Fig. 5c). The maximum fraction of susceptible individuals adopting the altered behaviour (namely, $1 - \min(p)$) reaches about the 84% versus almost the 100% in the case C1 (Fig. 5b, black line).

The scenarios of balanced opinions for individuals initially in $S_a$ ($m_{S_a,0} = 1$) and more or less flexible opinions for individuals in $S_n$ ($m_{S_n,0} = 1.5$ or $m_{S_n,0} = 0.5$, respectively) are depicted in Fig. 6. Corresponding relevant quantities concerning both the transient and the asymptotic system dynamics are reported in Table 3 (second and third columns) and compared with the baseline case of fully balanced opinions (first column). We observe that variations in the average flexibility of opinion have little impact on the height of the epidemic peak and do not induce subsequent waves, but modify the descent of the epidemic curve (Fig. 6b). This may be explained by the fact that the perceived risk of infection is so low that individuals react to the epidemic outbreak when the rising is already out of control. Even when the switch from the normal to the altered behaviour is massive, as in the case of averagely more volatile opinions (see Fig. 6a, red line), it occurs too late to significantly reduce the epidemic peak. Nonetheless, the impact on the descent epidemic phase can significantly affect the total number of infections: the asymptotic cumulative incidence of the disease, $\rho_R^\infty N_{tot}$ (that is well approximated by $(1 - \rho_{S_n}^\infty)N_{tot}$), varies from about the 49% of the population, when individuals have more volatile opinions ($m_{S_n,0} = 1.5$), to the 78%, when individuals are more intransigent ($m_{S_n,0} = 0.5$). In the latter scenario of less flexible opinions, note also that the stationary average flexibility of opinion for the $S_a$ compartment is greater than that for the $S_n$ compartment: $m_{S_a}^\infty = 0.6 > 0.51 = m_{S_n}^\infty$ (Table 3, third column), this is because the number of $S_n$ individuals entered in the $S_a$ compartment is not enough for their opinion flexibility to fully prevail in it (see Fig. 6a, blue line).

Finally, the scenarios of balanced opinions for individuals initially in $S_n$ ($m_{S_n,0} = 1$) and more or less flexible opinions for individuals in $S_a$ ($m_{S_a,0} = 1.5$ or $m_{S_a,0} = 0.5$, respectively) are depicted in Fig. 7. Corresponding relevant quantities concerning the system dynamics are reported in Table

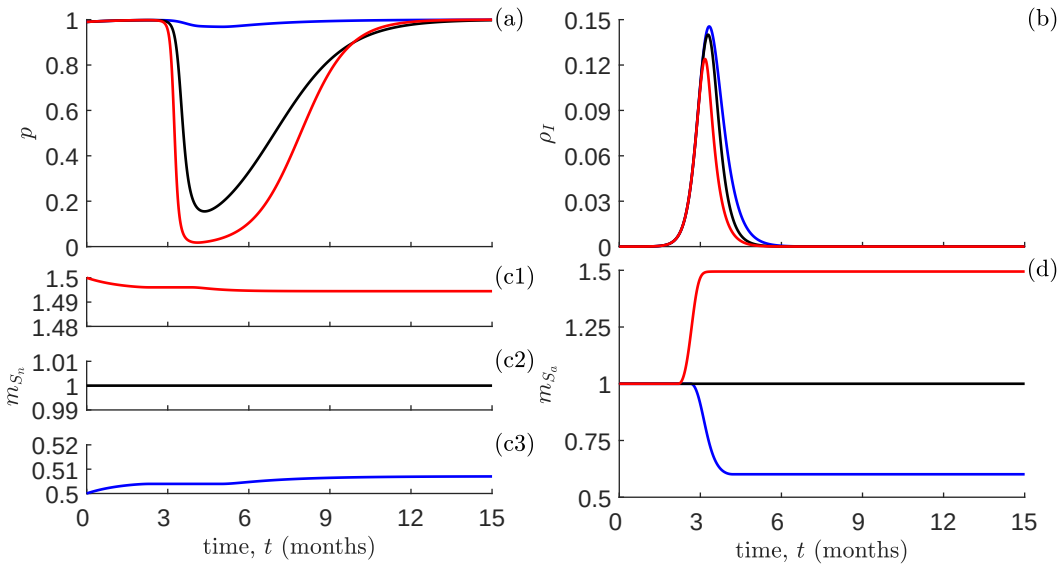


Figure 6: Case study C2. Numerical solutions as predicted by model (22) by assuming $m_{S_a,0} = 1$ and $m_{S_n,0} = 0.5$ (blue lines), $m_{S_n,0} = 1$ (black lines), $m_{S_n,0} = 1.5$ (red lines). Panel (a): fraction of susceptible individuals with normal behaviour, $p$. Panel (b): density of infectious individuals, $\rho_I$. Panels (c1)–(c3): average flexibility of opinion for the $S_n$ compartment, $m_{S_n}$. Panel (d): average flexibility of opinion for the $S_a$ compartment, $m_{S_a}$. Parameters values and other initial conditions are given in Table 1.

| | $m_{S_n,0} = 1$<br>$m_{S_a,0} = 1$ | $m_{S_n,0} = 1.5$<br>$m_{S_a,0} = 1$ | $m_{S_n,0} = 0.5$<br>$m_{S_a,0} = 1$ | $m_{S_n,0} = 1$<br>$m_{S_a,0} = 1.5$ | $m_{S_n,0} = 1$<br>$m_{S_a,0} = 0.5$ |
|---|---|---|---|---|---|
| $\max(\rho_I)$ | 0.14 | 0.12 | 0.15 | 0.14 | 0.13 |
| $\arg\max(\rho_I)$ (days) | 98.61 | 95.11 | 99.61 | 99.11 | 98.11 |
| $\min(p)$ | 0.16 | 0.02 | 0.97 | 0.21 | 0.12 |
| $\rho_{S_n}^{\infty}$ | 0.36 | 0.51 | 0.22 | 0.33 | 0.40 |
| $\rho_{S_a}^{\infty}$ | $1.61 \cdot 10^{-15}$ | $1.47 \cdot 10^{-15}$ | $1.61 \cdot 10^{-15}$ | $1.41 \cdot 10^{-15}$ | $1.79 \cdot 10^{-15}$ |
| $m_{S_n}^{\infty}$ | 1.00 | 1.49 | 0.51 | 1.01 | 0.99 |
| $m_{S_a}^{\infty}$ | 1.00 | 1.49 | 0.60 | 1.01 | 0.99 |

Table 3: Case study C2. Relevant quantities as predicted by model (22) for five combinations of the initial average flexibilities of opinion $m_{S_n,0}$ and $m_{S_a,0}$. Parameters values and other initial conditions are given in Table 1.

3 (fourth and fifth columns). As expected, due to the initial small size of the $S_a$ compartment, the impact of $m_{S_a,0}$ on both the transient and the asymptotic dynamics of model (22) is minor: when $m_{S_a,0}$ varies from 1.5 to 0.5, the total number of infections caused by the epidemic outbreak ($\rho_R^{\infty} N_{tot} \approx (1 - \rho_{S_n}^{\infty}) N_{tot}$) decreases from the 67% to about the 60% of the total population, and the maximum fraction of the susceptible population adopting the altered behaviour ($1 - \min(p)$) increases from the 79% to the 88%. Other relevant quantities undergo even more little variations.

# 5 Concluding remarks

Shaping the complex dynamics of spontaneous behavioural changes in response to an epidemics is crucial for public health authorities to plan control strategies. Mathematical models proved to be a valid tool to improve the understanding of the intricate interplay between human behaviour and disease spreading. Here, without referring to any specific disease, we give a contribution in this direction by using a kinetic theory approach.

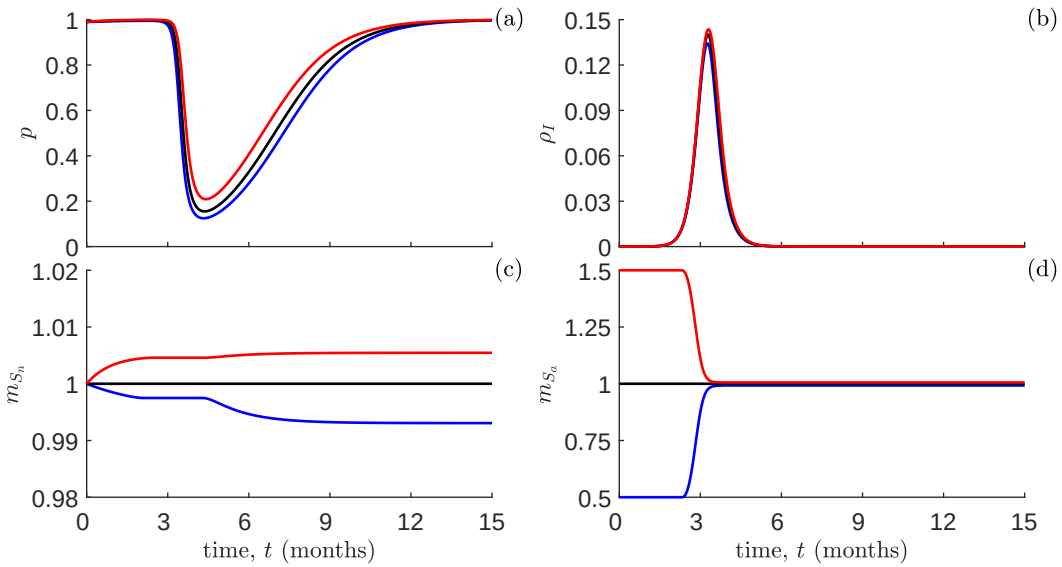


Figure 7: Case study C2. Numerical solutions as predicted by model (22) by assuming $m_{S_n,0} = 1$ and $m_{S_a,0} = 0.5$ (blue lines), $m_{S_a,0} = 1$ (black lines), $m_{S_a,0} = 1.5$ (red lines). Panel (a): fraction of susceptible individuals with normal behaviour, $p$. Panel (b): density of infectious individuals, $\rho_I$. Panels (c): average flexibility of opinion for the $S_n$ compartment, $m_{S_n}$. Panel (d): average flexibility of opinion for the $S_a$ compartment, $m_{S_a}$. Parameters values and other initial conditions are given in Table 1.

We develop a game theoretical epidemic model where the flexibility of opinion of the individuals about the change of social contact patterns is explicitly taken into account. Starting from a stochastic particle model, we derive a statistical description at the mesoscopic level of our multi–agent system and, eventually, a macroscopic model given by a system of non–linear ODEs. This latter model naturally inherits a large number of features of the original microscopic dynamics with the advantage that it is more amenable to analytical and numerical investigations.

Among the main results, we evidence that

- By attributing a degree of flexibility of opinion to each agent, we account for the personal attitude to change opinion. This leads the heterogeneity of the cost–benefit assessments of individuals who have to choose whether to change or preserve their social behaviour;
- During an interaction between individuals adopting alternative social behaviours, it may happen that both or neither of them perceive their own strategy as less convenient than the alternative one. It follows that at population level the switching rates between the two strategies may be both positive or both null at the same time;
- With respect to the hypothetical case that all the individuals have the same flexibility of opinion, having variegated personal attitudes produces both a qualitative and a quantitative impact on the disease dynamics and on the consequent behavioural responses;
- In the case of relatively high perceived risk of infection within the population (Section 4.1), more or less flexible opinions may: i) lead to an increase or a decrease of the number of epidemic waves; ii) modify the height of the peak and the time it occurs; with respect to the baseline scenario of fully balanced opinions. However, the corresponding variations in the cumulative disease incidence at the end of the time horizon are small (not more than 9%);
- In the case of relatively low perceived risk of infection (Section 4.2), more or less flexible opinions do not increment or decrement the number of epidemic waves with respect to the

scenario of balanced opinions, and have little impact on the height and time occurrence of the peak. However, the cumulative incidence may vary from the 49% of the population, when individuals have more volatile opinions, to the 78%, when they are more intransigent.

We stress that our approach is quite general and could be easily adapted to other epidemiological frameworks where individual opinions play a key role. For example, the voluntary adherence to immunization programs is a *hot topic* of behavioural epidemiology of infectious diseases [2, 7, 8, 12, 34, 49]. In such a context, a critical phenomenon is the 'pseudo–rational' exemption to vaccination [2, 7, 12, 49]: individuals are more or less inclined to overweight real and presumed vaccine side effects and underweight actual risks due to the disease. Also, our approach could be extended by including more heterogeneity in the behavioural strategies that individuals can adopt. For example, one can consider a finite number (greater than two) of alternative strategies or even a *continuous* dynamics between the two extremal ones.

Finally, we plan to incorporate the role of the flexibility of opinion in a behavioural change epidemic model where the disease dynamics explicitly depends on the individual viral load. Namely, we want to combine the approach introduced here with that considered in the papers [31, 32]. In such a case, each agent would be characterized by – besides the epidemiological compartment to which he/she belongs – two microscopic traits: the viral load, which influences the transmission and progression of the infection, and the flexibility of opinion, which influences the imitation game dynamics.

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# A Numerical comparison between the particle and the macroscopic models

In Fig. A1 we report an illustrative comparison between the numerical solutions of the particle model (12) (markers) and those of the macroscopic model (22) (solid lines). We consider the case study C2 in the baseline scenario of fully balanced opinions, namely $m_{S_n} = m_{S_a} \equiv 1$ (see Fig. 5). In order to simulate the stochastic particle model, we implement a Monte Carlo algorithm. We remark that this corresponds to integrate the kinetic model (14)—(17) in a Monte Carlo framework. The Monte Carlo simulations are performed by setting the time step $dt = 10^{-3}$ days and the parameters $\lambda_{S_i,I} = \lambda = \lambda_R^I = 1$, $\beta_{d,i} = \beta_i$, $\gamma_r = \gamma$, $i \in \{n, a\}$. As expected, from Fig. A1 we note a very good match between the two approaches in predicting the dynamics of compartment densities.

We verified that similar results hold when different sets of parameter values are considered (plots not shown here).

# References

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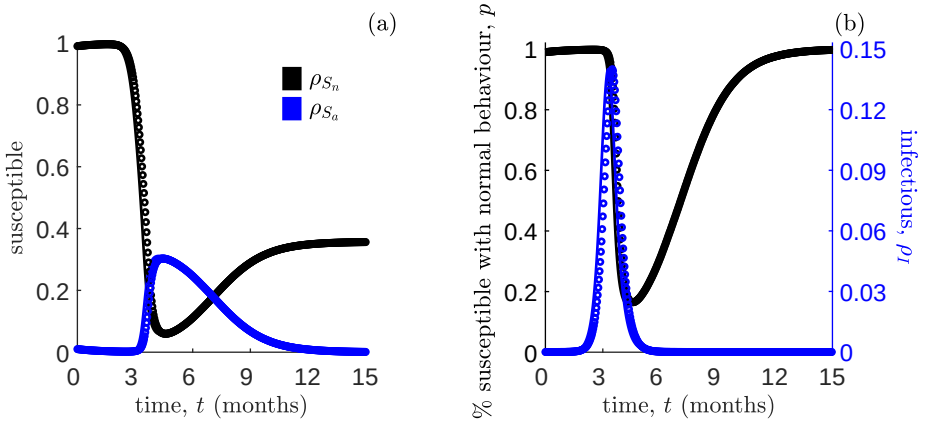


Figure A1: Case study C2. Numerical solutions as predicted by the particle model (12) (markers) and by the macroscopic model (22) (solid lines) by assuming $m_{S_n,0} = m_{S_a,0} = 1$. Panel (a): density of susceptible individuals with normal ($\rho_{S_n}$, black colour) and altered ($\rho_{S_a}$, blue colour) behaviours. Panel (b), left y–axis: fraction of susceptible individuals with normal behaviour, $p$ (black colour). Panel (b), right y–axis: density of infectious individuals, $\rho_I$ (blue colour). Parameters values and other initial conditions are given in Table 1 and in the Appendix A.


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