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Wearing face masks to protect oneself and/or others: counter-intuitive results from a simple epidemic model accounting for selfish and altruistic human behaviour

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Abstract

We study a simple SIS epidemic model accounting for human behaviour. Individuals can decide at each instant of time whether or not they wear a face mask. Mask wearing decreases susceptibility to and/or transmission of the disease. We consider a situation in which individuals are unaware of their health status (infected or not), but can perceive disease prevalence at the population level. This assumption fits situations in which asymptomatic people can be numerous and tests are not widely available. Individual decision dynamics depend both on disease prevalence, as a proxy for the risk of being infected or infecting others, and/or the fraction of the population complying to mask-wearing, which people can observe in their everyday life. Specifically, human behaviour is assumed to be driven by imitation dynamics. When the disease does not naturally die out, the model has three types of endemic equilibria: no-compliance, partial-compliance, and full-compliance. Only one of these equilibria can be stable at a time. We assume that the effectiveness of mask-wearing is positively correlated to its cost at the individual level. Increasing mask effectiveness and therefore its individual cost can make the system switch from full-compliance to partial-compliance. This way, increasing mask effectiveness may increase disease prevalence at equilibrium. In other words, disease prevalence is minimized for intermediate mask effectiveness and cost. This is because, when mask-wearing is too effective and therefore costly, part of the population free-rides on the effort of others and drops mask, resulting in increased prevalence. Altogether, our results show that the interplay between epidemiology and human behaviour may lead to counter-intuitive but nevertheless intelligible outcomes, which should be anticipated when designing public health policies.

1 Introduction

In the contemporary landscape of infectious disease research, the integration of human behavior in mathematical epidemic models has emerged as a continued trend [34, 35, 78, 75, 15, 8, 38, 32, 67], ironically called in its ‘epidemic phase’ some years ago in [53]. Patient 0 of models in behavioral epidemiology can be traced back to [12], in which the dynamics of the disease and that of human behavior are mingled in an unspecified way, in the sense that the force of infection depends of the prevalence in a non-monotonous manner. Behavioral epidemiology modelling has then evolved in various directions, as is very well exposed in [32] specifically on modelling for Covid-19.

As vaccines have been a major breakthrough in the never-ending struggle between mankind and infectious diseases, and paradoxically the subject of public scare [2, 44, 30], they are logically subject to many models, *e.g.* [7, 9, 10, 79]. Among the tools borrowed from game theory, imitation dynamics or related concepts proved particularly fruitful, both in an ODE setting [6, 22, 20, 83, 21, 70, 11, 4, 17, 18, 47, 24, 24] or agent-based models [68, 54, 80] to study the compliance to vaccination as a powerful mean to fight an epidemic.

However in the early stages of the last Covid-19 pandemic, vaccines are not always available, especially for a new disease. In such a context, other prophylactic measures can be deployed, such as social distancing,

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mask-wearing, hand-washing, or the use of condoms or lockdown for example. In the case of Covid-19, various non-pharmaceutical interventions (NPI) have been evaluated, see for instance [42] for social distancing measures, [41] for face masks wearing, and more generally [52]) for an overview of evaluated NPI. Such interventions have received less attention than vaccines from the modelling literature so far. Let us mention for example [45, 36, 33, 85, 25] for agent-based models and [65, 64, 69, 57] regarding ordinary differential equation (ODE) models. The present study falls into this last category, focusing on mask-wearing, which specific features have been slightly overlooked so far.

More precisely, we present and analyse a SIS-type model coupled with behaviour by means of imitation dynamics. Similar existing models often assume that the NPI considered reduces disease transmission by a certain factor, *e.g.* [51, 13, 40]. Such an assumption is consistent with a reduction of the encounter rate, for instance by self-isolation, or with social distancing. However, in the case of mask wearing, both transmission and susceptibility can be reduced, and to different extents [66, 50, 49]. To our best knowledge, these dual effects of mask wearing were taken into account in three modeling studies so far: first [28] in an ODE setting and [63] on a contact network, in which individuals are not able to change their strategy, and then [81] in a SIS setting, yet in a situation where the disease is set at equilibrium. The main novelty of our approach lies in the mechanistic derivation of a behavioural epidemic model from first principles.

Epidemic models coupled with behavior usually assume that individuals are guided solely by their own interests. However, it has been highlighted that when coming to health-related decision, purely selfish choices are disregarded by experimental data [71, 76], even though such decisions come from a complex combination of various factors [39, 14, 19, 62]. We found one previous paper within the framework of ODEs that explicitly includes altruism. Indeed, in [29] the authors modelled a vaccination game in the context of a single wave of influenza. They included altruism as a numeric parameter that was then related to the payoffs of each strategy (being vaccinated or not), and finally deduced a probability of vaccination in the population. We reuse this idea of altruism, in a different setting. We assume that people are unaware of their current health status, but have access to the prevalence at the national scale, which matches the ‘early epidemic’ situation in which NPI are the only leverage for action and tests are scarce. Thus, before choosing their strategy, individuals weigh the risk to get infected or infecting other, depending on the mean degree of altruism in the population.

This paper is organized as follows. The next section is devoted to the derivation of the model. In Section 3.2, we present in details the dynamics of a simplified version of the model, in the sense that we consider that masks can only act on reducing one of the two risks (infecting others). Although simpler, this particular case enables to capture the main picture of the model. To explore the implications of the model, we next assume a positive correlation between the perceived cost and the effectiveness of mask-wearing. Section 3.3 is devoted to the study of the full model, in particular the impact of the correlation between the cost and effectiveness. Finally, we discuss our results, as well as their limitations, and point out some directions for future research.

2 A coupled epidemics-human behavior model

In this section, we introduce and study our behavioral epidemic model. It is based on a classical SIS model, coupled with an equation that accounts for the ability for individuals to freely adopt or drop a NPI, such as social distancing, hand washing or mask wearing, in order to fight against a chronic disease. This translates into a coupling between the disease dynamics and human behavior. Individuals who do not comply with the NPI are called defectors.

2.1 Mechanistic derivation

Borrowing from [65] in the context of behavioural epidemiology or [16] regarding social contagion, we first introduce a simpler version of our model in which people cannot change their strategy. Consider a population of N individuals, and denote S_C , S_D , I_C and I_D respectively the density of complying susceptible individuals, defecting susceptible ones, cooperating infectious and defecting infectious.

The force of infection for defecting susceptible is made of the sum of two terms: first the risk of becoming infected after encountering a defecting infected, and then the risk of becoming infected after encountering a cooperating infected. In the later case, a discount of $(1 - e_T)$ is applied to the infection rate, due to the infectious individual adopting the protective measure considered. This results in the following force of infection for defectors

$$+\beta \left[\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right]$$

in which β is the natural infection rate. We make the assumption that adopting the control measure can only reduce the risk of infection, and is ineffective about the duration of the infectious period. Thus, the recovery rate is the same regardless of whether the individual complies or not. Finally, the equations on the cooperating population are obtained by multiplying the force of infection in each of the previous equations by $(1 - e_S)$, due to the protection provided by the NPI. The system reads

$$\begin{cases} \frac{dS_D}{dt} = -\beta \left[\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right] S_D + \gamma I_D \\ \frac{dI_D}{dt} = \beta \left[\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right] S_D - \gamma I_D \\ \frac{dS_C}{dt} = -\beta(1 - e_S) \left[\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right] S_C + \gamma I_C \\ \frac{dI_C}{dt} = \beta(1 - e_S) \left[\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right] S_C - \gamma I_C \end{cases}$$

We easily check that the relation $S_C + S_D + I_C + I_D = N$. This property will remain at the next step of the derivation of the model, in which we will allow individuals to change their strategy.

As mentioned earlier, our model relies on imitation dynamics. In this approach, we make the assumption that people challenge their opinion on a regular basis by comparing their payoff to that of individuals adopting the opposite strategy. In our case, we make the pivotal assumption that people are unaware of their health status, due to a lack of tests. We also assume the prevalence at the global scale is extrapolated from the few tests available, hence we neglect those individuals who know exactly whether or not they are infected. As a consequence, people make their decision based solely on the general prevalence, plus the degree of altruism and the characteristics of the NPI. The latter parameters are assumed to be independent of the dynamics of the disease.

Denoting π_C and π_D the payoffs associated with cooperating and defecting respectively, individuals compute the difference $\Delta E := \pi_C - \pi_D$ and possibly change their current strategy based on the sign of this quantity. For instance, susceptible defectors observe a sample of the population, and take into account only cooperators, who are drawn with probability $\frac{S_C + I_C}{N}$, and become susceptible cooperators at rate

$$\sigma(\Delta E)_+ \frac{S_C + I_C}{N},$$

while susceptible cooperators also observe all the cooperators and become susceptible defectors at rate

$$\sigma(\Delta E)_- \frac{S_C + I_C}{N},$$

where σ is the opinion update rate, and in which subscripts $+$ and $-$ denote positive and negative parts, respectively. Finally, the system is given by

$$\begin{cases} \frac{dS_D}{dt} = -\beta \left[\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right] S_D + \gamma I_D - \sigma \left[(\Delta E)_+ \frac{S_C + I_C}{N} S_D - (\Delta E)_- \frac{S_D + I_D}{N} S_C \right] \\ \frac{dI_D}{dt} = \beta \left[\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right] S_D - \gamma I_D - \sigma \left[(\Delta E)_+ \frac{S_C + I_C}{N} I_D - (\Delta E)_- \frac{S_D + I_D}{N} I_C \right] \\ \frac{dS_C}{dt} = -\beta(1 - e_S) \left[\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right] S_C + \gamma I_C + \sigma \left[(\Delta E)_+ \frac{S_C + I_C}{N} S_D - (\Delta E)_- \frac{S_D + I_D}{N} S_C \right] \\ \frac{dI_C}{dt} = \beta(1 - e_S) \left[\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right] S_C - \gamma I_C + \sigma \left[(\Delta E)_+ \frac{S_C + I_C}{N} I_D - (\Delta E)_- \frac{S_D + I_D}{N} I_C \right]. \end{cases} \quad (1)$$

We will need to provide an explicit formulation for the term ΔE , which we postpone to the next section. Figure 1 depicts a flowchart corresponding to the relations between the different compartments.

2.2 Payoffs, inclusion of altruism and perception bias

Most of previous works in behavioral epidemiology only account for purely selfish individuals. However, there are evidence that altruism plays a role in the decision-making process, see for example [71, 76, 27] or [72], chapter 3. Such an altruistic behavior was modelled in [29] by including an altruistic part in the payoff of the vaccinating strategy. This additional term depends on the so called *degree of altruism* in the population. In the present work, we keep the idea of degree of altruism, and we model the fraction of cooperators in the population as a dynamic quantity, drawing inspiration from imitation dynamics. Indeed, individuals are interested in protecting themselves and protecting others, combining both interests based

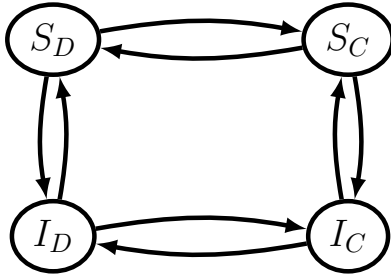


Figure 1: Flowchart associated with the model. Vertical transitions stand for a change in the health status, while horizontal ones result from a modification of the strategy adopted.

on the degree of altruism in the population. To include these somewhat opposite interests, we take into account the ability of a control measure to reduce either, or both, of susceptibility or transmission, see for example [74, 56, 63], or on related considerations [82, 37]. The ability of a relevant face mask to reduce susceptibility and/or transmission have been studied in [48, 46], and in [31] the impact of mask-wearing in the two parts of Ireland has been evaluated.

As mentioned earlier, individuals can either cooperate or defect, with associated payoffs π_C and π_D , respectively. Since people are unaware of their health status, these payoffs depend on the total prevalence

$$i := \frac{I_C + I_D}{N}. \quad (2)$$

The control measure considered provides a certain level of protection for the adopters and for people (both cooperators or defectors) they encounter, so the payoffs depend on the frequency such encounters. We denote

$$c := \frac{S_C + I_C}{N} \quad (3)$$

the fraction of cooperators in the population. The formulas associated to payoffs are thus

$$\begin{aligned} \pi_C(i, c) &= c\pi_{C,C}(i) + (1-c)\pi_{C,D}(i) \\ \pi_D(i, c) &= c\pi_{D,C}(i) + (1-c)\pi_{D,D}(i) \end{aligned}$$

with $\pi_{A,B}$ the payoff of an individual adopting strategy A encountering someone adopting strategy B , with $A, B \in \{C, D\}$. It remains to define such ‘pairwise encountering payoffs’.

We now wish to include both a ‘selfish’ and an ‘altruistic’ cost. To do so, we define the payoff of a defector meeting another defector as

$$\pi_{D,D}(i) = -r_S i - r_A i.$$

The first term accounts for a purely selfish part of the payoff, *i.e.* related to the perceived cost of catching the disease, such as feeling sick, missing school or work, and so on. The non negative parameter r_S provides the intensity of such concern. The second term stands for the altruistic part, namely the fear to infect the other. A defector meeting a cooperator benefits from its protection without paying any cost, so we assume a reduction in the perceived risk of being infected from the other, as well as that of infecting them. If the defector had a purely rational perception of the reduction in the probability of infection, the payoff would be $-r_D(1 - e_T)i - r_A(1 - e_S)i$. However, we want to allow a proportion of irrationality (or imperfect information) in the population. We then define the payoff of a defector encountering a cooperator as

$$\pi_{D,C}(i) = -r_S(1 - \varphi_T(e_T))i - r_A(1 - \varphi_S(e_S))i$$

with $\varphi_S, \varphi_T : [0, 1] \rightarrow [0, 1]$ two increasing functions accounting for a level of irrationality, in the sense that they reduce the fear of being infected or transmit the disease by a factor that can differ from the actual effectiveness of the control measure. Examples of such bias functions are given in the Appendix C. Now we define the payoffs for cooperators. Since they are complying with a costly control measure, there is a constant cost k included. Such an individual encountering a defector benefits from their own protection only, both to reduce their fear of being infected and of transmitting the disease, so the payoff is

$$\pi_{C,D}(i) = -k - r_S(1 - \varphi_S(e_S))i - r_A(1 - \varphi_T(e_T))i.$$

In contrast, when two cooperators meet, they benefit from both inward and outward protections, so the payoff is

$$\pi_{C,C}(i) = -k - r_S(1 - \varphi_S(e_S))(1 - \varphi_T(e_T))i - r_A(1 - \varphi_S(e_S))(1 - \varphi_T(e_T))i.$$

2.3 Dimensionless compact model

In this section, we reduce System (1) to an equivalent, more convenient set of three differential equations. First, we derive the equation giving the dynamics of the fraction of cooperators in the population by computing

$$\begin{aligned}\frac{dc}{dt} &= \frac{d}{dt} \frac{S_C + I_C}{N} \\ &= \sigma \left[(\Delta E)_+ \frac{S_C + I_C}{N} \frac{S_D + I_D}{N} - (\Delta E)_- \frac{S_D + I_D}{N} \frac{S_C + I_C}{N} \right] \\ &= \sigma c(1-c) \Delta E(i, c)\end{aligned}$$

and it remains to provide a tractable expression for ΔE . We compute

$$\begin{aligned}\Delta E &= \pi_C(i, c) - \pi_D(i, c) \\ &= ([r_S \varphi_S(e_S) (1 - \varphi_T(e_T) c) + r_A \varphi_T(e_T) (1 - \varphi_S(e_S) c)] i - k) \\ &= (r_S + r_A) ([(1 - \alpha) \varphi_S(e_S) + \alpha \varphi_T(e_T) - \varphi_S(e_S) \varphi_T(e_T) c] i - \rho)\end{aligned}$$

where $\alpha := \frac{r_A}{r_S + r_A}$ can be interpreted as the mean level of altruism in the population and $\rho := \frac{k}{r_S + r_A}$ the relative cost of the control measure. Low values of α mean that individuals are more afraid of getting infected themselves than of infecting others, and thus are more receptive to a reduction in susceptibility than transmission. In contrast, if α is close to 1, the reduction in the risk of getting infected by others plays a minor role, whereas the term in $\varphi_T(e_T)$ is emphasized. In addition, if $e_S = e_T = 0$, then the prophylactic measure is actually useless, and one has $\pi_C > \pi_D$ no matter the state of the epidemic, so no one complies. We denote

$$\mathcal{A} = \mathcal{A}(e_S, e_T, \alpha) := (1 - \alpha) \varphi_S(e_S) + \alpha \varphi_T(e_T)$$

an arithmetic mean of the perceived values of the characteristics of the control measure, and also

$$\mathcal{H} = \mathcal{H}(e_S, e_T, \alpha) := \frac{1}{\frac{\alpha}{\varphi_S(e_S)} + \frac{1-\alpha}{\varphi_T(e_T)}}$$

a harmonic mean, so that $\Delta E(i, c)$ can be rewritten in a compact form as

$$\Delta E(i, c) = (r_S + r_A) (\mathcal{A}(1 - \mathcal{H}c) i - \rho).$$

In this last term the prevalence i appears, whose dynamics is given by the equation

$$\begin{aligned}\frac{di}{dt} &= \frac{d}{dt} \frac{I_C + I_D}{N} \\ &= \beta \left(\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right) \left(\frac{S_D}{N} + (1 - e_S) \frac{S_C}{N} \right) - \gamma \left(\frac{I_C}{N} + \frac{I_D}{N} \right) \\ &= \beta \left(\frac{I_D + I_C}{N} - e_T \frac{I_C}{N} \right) \left(\frac{S_D + S_C}{N} - e_S \frac{S_C}{N} \right) - \gamma \frac{I_C + I_D}{N} \\ &= \beta \left(1 - e_S \frac{S_C}{S_C + S_D} \right) \left(1 - e_T \frac{I_C}{I_C + I_D} \right) \frac{S_C + S_D}{N} \frac{I_C + I_D}{N} - \gamma \frac{I_C + I_D}{N} \\ &= \beta \left(1 - e_S c \left(\frac{1}{c} \frac{S_C}{S_C + S_D} \right) \right) \left(1 - e_T c \left(\frac{1}{c} \frac{I_C}{I_C + I_D} \right) \right) (1 - i) i - \gamma i\end{aligned}$$

where we used the notations previously introduced i and c . It remains to deal with two terms, that we denote

$$x := \frac{1}{c} \frac{S_C}{S_C + S_D} = \frac{1}{c} \frac{1}{1 - i} \frac{S_C}{N} \quad \text{and} \quad y := \frac{1}{c} \frac{I_C}{I_C + I_D} = \frac{1}{c} \frac{1}{i} \frac{I_C}{N}.$$

Straightforward computations lead to the relation

$$(1 - i)x + iy = 1. \tag{4}$$

In the very particular situation of the events ‘being infected’ and ‘cooperating’ being independent, *i.e.* if

$$\mathbb{P}(\text{cooperating and being infected}) = \mathbb{P}(\text{cooperating}) \times \mathbb{P}(\text{being infected}),$$

then one has

$$\frac{I_C}{N} = \frac{S_C + I_C}{N} \frac{I_C + I_D}{N} = c \times i$$

which leads to $y = 1$, and thus $x = 1$ thanks to Equation (4). This case is obtained as the limit in large time when $e_S = 0$, as stated and studied in Section 3.2. For any other case, we need an additional equation, since System (1) has four equations. We compute the time derivative of the y variable

$$\frac{dy}{dt} = \frac{d}{dt} \left(\frac{1}{ci} \frac{I_C}{N} \right) = \frac{1}{ci} \frac{d}{dt} \frac{I_C}{N} - y \left(\frac{1}{c} \frac{dc}{dt} + \frac{1}{i} \frac{di}{dt} \right)$$

and compute each term separately. First, using extensively Equation (4), we obtain

$$\begin{aligned} \frac{1}{ci} \frac{d}{dt} \frac{I_C}{N} &= \frac{1}{ci} \left(\beta(1 - e_S) \left[\frac{I_D}{N} + (1 - e_T) \frac{I_C}{N} \right] S_C - \gamma I_C + \sigma \left[(\Delta E)_+ \frac{S_C + I_C}{N} I_D - (\Delta E)_- \frac{S_D + I_D}{N} I_C \right] \right) \\ &= \frac{1}{ci} \left(\beta(1 - e_S) [1 - e_T cy] \frac{i S_C}{N} - \gamma \frac{I_C}{N} + \sigma \left[(\Delta E)_+ c \frac{I_D}{N} - (\Delta E)_- (1 - c) \frac{I_C}{N} \right] \right) \\ &= \beta(1 - e_S)(1 - e_T cy)(1 - iy) - \gamma y + \sigma [(\Delta E)_+(1 - cy) - (\Delta E)_-(1 - c)y] \end{aligned}$$

which has to be combined with

$$\frac{1}{c} \frac{dc}{dt} + \frac{1}{i} \frac{di}{dt} = \sigma(1 - c)\Delta E + \beta(1 - e_S c x)(1 - e_T cy)(1 - i) - \gamma.$$

As a result, we obtain that System (1) is equivalent to

$$\begin{cases} \frac{di}{dt} = \beta \left(1 - e_S c \frac{1 - iy}{1 - i} \right) (1 - e_T cy) (1 - i) i - \gamma i \\ \frac{dc}{dt} = \sigma c(1 - c)\Delta E(i, c) \\ \frac{dy}{dt} = \beta(1 - e_T cy)((1 - e_S(1 - cy))(1 - iy) - (1 - i)y) + \sigma(1 - y)(\Delta E(i, c))_+. \end{cases}$$

We further simplify it by giving a dimensionless version of the equation. Specifically, we introduce three new dimensionless parameters:

- γt , still denoted t (since the dimension of γ is the inverse of a time);
- the basic reproduction number $\mathcal{R}_0 := \frac{\beta}{\gamma}$;
- the modified opinion updating rate $\kappa = \frac{\sigma(r_s + r_a)}{\gamma}$.

Finally, we obtain the following system that we will study

$$\begin{cases} \frac{di}{dt} = \mathcal{R}_0(1 - e_S c - i(1 - e_S cy)) (1 - e_T cy) i - i \\ \frac{dc}{dt} = \kappa c(1 - c) (\mathcal{A}(1 - \mathcal{H}c)i - \rho) \\ \frac{dy}{dt} = \mathcal{R}_0(1 - e_T cy)(1 - y - e_S(1 - iy)(1 - cy)) + \kappa(1 - y) (\mathcal{A}(1 - \mathcal{H}c)i - \rho)_+ \end{cases} \quad (5)$$

completed with the relation $(1 - i)x + iy = 1$. Parameters and related pieces of information are collected in Table 1. We prove in the Appendix that the flow drives this three dimensional system into the cube $[0, 1]^3$, which is invariant. Since we are interested in asymptotic behaviors, we assume for simplicity that $y(0) \leq 1$. An example of a dynamics generated by this model is depicted on Figure 2, with both sets of variables. From the graph on the right, we observe that untangling the respective contribution of each group to the disease dynamics seems complicated. However, in the (i, c, y) variables, the interpretation is clearer: the rise of the prevalence motivates people to adopt the protective behavior, which reduces transmissions and thus the prevalence, so in turn some individuals start to free-ride, enabling the disease to spread again.

If the function $(i, c) \mapsto \Delta E(i, c)$ has a constant sign on the square $[0, 1] \times [0, 1]$, then the fraction of compliers can only evolve monotonously and as a result, the behaviour part of the model becomes trivial. Thus, we impose a condition on ρ so that this function changes sign. Since $\Delta E(0, c) = -\rho$ is negative, we want the maximum of ΔE to be positive, which is equivalent to the condition

$$\rho < \mathcal{A}. \quad (6)$$

The fraction $\frac{\rho}{\mathcal{A}}$ can be understood as a kind of ‘price-quality ratio’. We show in the next section that it has to be compared with two explicit bounds to obtain the large time dynamics of the system.

Name	Symbol	Range
Basic reproduction number	\mathcal{R}_0	$(0, \infty)$
Effectiveness to reduce susceptibility	e_S	$[0, 1]$
Effectiveness to reduce transmission	e_T	$[0, 1]$
Dimensionless opinion update rate	κ	$(0, \infty)$
Dimensionless perceived cost	ρ	$[0, \infty)$

Table 1: Parameters.

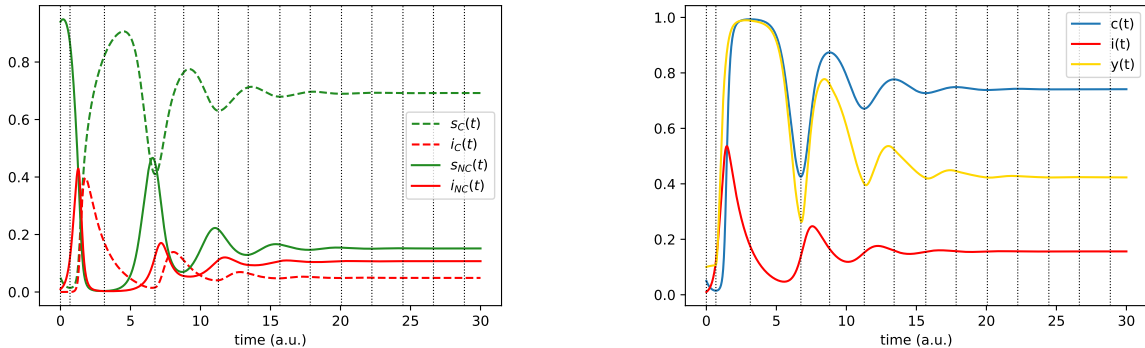


Figure 2: Time evolution of the system with unbiased perception of the effectiveness of the control measure. The parameters are $\mathcal{R}_0 = 5$, $e_S = 0.9$, $e_T = 0.3$, $\kappa = 30$, $\rho = 0.1$ and $\alpha = 0.1$, so that the selected equilibrium is the Endemic with Partial Compliance. Vertical dashed lines indicate times when ΔE changes sign. Left: system in the (i, c, y) variables. Right: system in the initial variables.

3 The equilibria and their stability conditions

In this section, we present different steady states and their associated stability. The system of coupled non-linear ODEs we study has five possible equilibria, with the notable property that only one at a time can be locally stable. In the first subsection, we state the equilibria as well as their local stability conditions. In the second, we focus on a particular case in which the model reduces to a planar system with every formula being explicit, and provide a rule of thumb to minimize prevalence at equilibrium, with and without perception bias. Finally in the last subsection, we conduct the same kind of study in the general case, building on the previous particular one to give an intuition of the phenomena at work.

3.1 Equilibria and stability

The system studied has five equilibria: two in a disease-free state, and three in which the disease is endemic. The Disease-Free with No Compliance equilibrium, labelled (DFN)

$$(i_*, c_*, y_*) = (0, 0, 1 - e_S),$$

exists whatever the values of the parameters, and is locally asymptotically stable (LAS) if and only if $\mathcal{R}_0 \leq 1$. The Disease-Free with Full Compliance equilibrium (DFF) also always exists and is defined as

$$(i_*, c_*, y_*) = (0, 1, 1)$$

is never stable. In contrast, when the disease is endemic, three equilibria can coexist, with a fraction of cooperators ranging from 0 to 1. The Endemic with No Compliance equilibrium (ENC)

$$(i_*, c_*, y_*) = \left(1 - \frac{1}{\mathcal{R}_0}, 0, \frac{1 - e_S}{1 - e_S \left(1 - \frac{1}{\mathcal{R}_0} \right)} \right)$$

Name	Abbr.	Values	Existence	Local stability
Disease-Free with No Compliance	DFN	$(0, 0, 1 - e_S)$	Always	$\mathcal{R}_0 \leq 1$
Disease-Free with Full Compliance	DFC	$(0, 1, 1)$	Always	Never
Endemic with No Compliance	ENC	$\left(1 - \frac{1}{\mathcal{R}_0}, 0, \frac{1 - e_S}{1 - e_S \left(1 - \frac{1}{\mathcal{R}_0}\right)}\right)$	$\mathcal{R}_0 > 1$	$1 - \frac{1}{\mathcal{R}_0} < \frac{\rho}{\mathcal{A}}$
Endemic with Partial Compliance	EPC	(i_*, c_*, y_*)	$(1 - \mathcal{H}) \left(1 - \frac{1}{\mathcal{R}_1}\right) < \frac{\rho}{\mathcal{A}} < 1 - \frac{1}{\mathcal{R}_0}$	
Endemic with Full Compliance	EFC	$\left(1 - \frac{1}{\mathcal{R}_1}, 1, 1\right)$	$\mathcal{R}_1 > 1$	$\frac{\rho}{\mathcal{A}} < (1 - \mathcal{H}) \left(1 - \frac{1}{\mathcal{R}_1}\right)$

Table 2: Equilibria and local stability of the system. We denote $\mathcal{R}_1 := \mathcal{R}_0(1 - e_S)(1 - e_T)$.

exists whenever $\mathcal{R}_0 > 1$ and is LAS roughly when the cost ρ is too large compared to the perceived effectiveness of the control measure and the severity of the disease, or more precisely when

$$1 - \frac{1}{\mathcal{R}_0} < \frac{\rho}{\mathcal{A}}.$$

On the opposite, it can occur that the whole population will comply with the prophylactic measure, leading to the Endemic with Full Compliance equilibrium (EFC)

$$(i_*, c_*, y_*) = \left(1 - \frac{1}{\mathcal{R}_1}, 1, 1\right)$$

with the modified reproduction number $\mathcal{R}_1 := \mathcal{R}_0(1 - e_S)(1 - e_T)$. This equilibrium exists if and only if $\mathcal{R}_1 > 1$, and is LAS when the control measure is cheap enough, namely

$$\frac{\rho}{\mathcal{A}} < (1 - \mathcal{H}) \left(1 - \frac{1}{\mathcal{R}_1}\right).$$

Lastly, we conjecture that when none of the two previous stability conditions is satisfied, an additional equilibrium exists and is stable. This equilibrium occurs when only a fraction of the population adopts the control measure, as a result of the ratio $\frac{\rho}{\mathcal{A}}$ having an intermediate value. We label it Endemic with Partial Compliance equilibrium (EPC), with the associated existence and stability condition

$$(1 - \mathcal{H}) \left(1 - \frac{1}{\mathcal{R}_1}\right) < \frac{\rho}{\mathcal{A}} < 1 - \frac{1}{\mathcal{R}_0}.$$

All these properties are summarized in Table 2.

3.2 Special case of a protective measure acting solely on transmission

Surgical mask wearing is a protective behavior that can be freely adopted by the population, in the absence of mandate. From a modelling point of view, this case corresponds to $e_S \ll 1$ and $e_T > 0$, meaning that the NPI proposed to the population reduces almost only transmission.

3.2.1 Mathematical analysis of the dynamical system

In this section, we study the particular case $e_S = 0$, which can be seen as an approximation of the aforementioned situation. A single equilibrium is possible for the third equation, which is $y_* = 1$. We show in the Appendix that in this particular case, the y variables converges exponentially fast toward 1. As a consequence, the equation on the prevalence reduces to

$$\frac{di}{dt} = \mathcal{R}_0(1 - e_T c)(1 - i)i - i$$

and that on cooperators to

$$\frac{dc}{dt} = \kappa c(1 - c)(\alpha \varphi_T(e_T)i - \rho).$$

¹The stability condition for the EPC equilibrium is conjectured from numerical simulations.

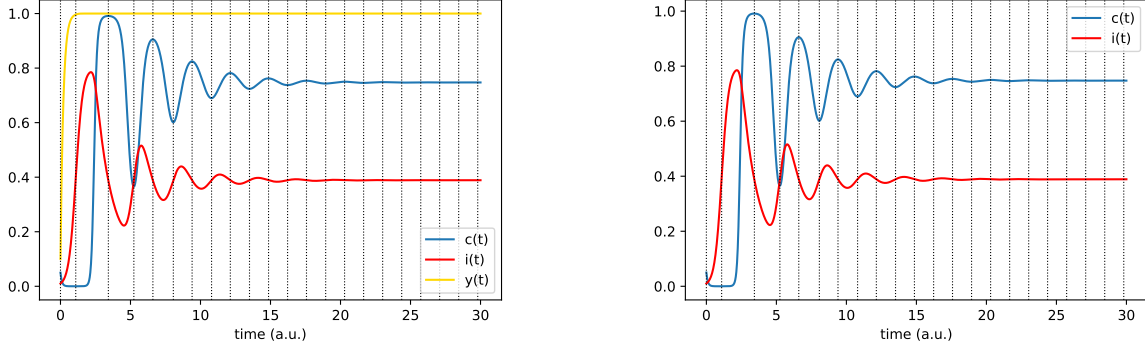


Figure 3: Time evolution of the system in the case $e_S = 0$ with unbiased perception of the effectiveness of the control measure. Parameters not mentioned previously are $e_T = 0.9$, $\kappa = 30$, $\rho = 0.35$ and $\alpha = 1$, so that the selected equilibrium is the EPC equilibrium. Vertical dashed lines marks times when ΔE changes sign. Left: system in the (i, c, y) variables. Right: planar system in the $i - c$ variables.

System (5) reduces in this case to the planar system given by

$$\begin{cases} \frac{di}{dt} = \mathcal{R}_0(1 - e_T c)(1 - i)i - i =: f(i, c) \\ \frac{dc}{dt} = \kappa c(1 - c)(\alpha \varphi_T(e_T)i - \rho) =: g(i, c) \end{cases} \quad (7)$$

which can be fully studied. Figure 3 illustrates the previous statements, we note in particular a close match between the asymptotic behaviors of the two models. An explicit expression for the EPC equilibrium can be given, namely

$$i_* = \frac{\rho}{\alpha \varphi_T(e_T)} \quad \text{and} \quad c_* = \frac{1}{e_T} \left(1 - \frac{1}{\mathcal{R}_0 \left(1 - \frac{\rho}{\alpha \varphi_T(e_T)} \right)} \right).$$

In addition, damped oscillations around this equilibrium can occur, but a limit cycle is impossible. Indeed, Dulac's criterion applied to the function

$$d(i, c) = \frac{1}{ic(1 - c)}$$

provides

$$\frac{\partial(fd)}{\partial i}(i, c) + \frac{\partial(gd)}{\partial c}(i, c) = -\frac{\mathcal{R}_0(1 - e_T c)}{c(1 - c)} < 0$$

in the semi-open square $(0, 1] \times (0, 1)$ which establishes the absence of limit cycle in this set. The remaining borders are easily dealt with by hand. Finally, for a large enough opinion update rate κ , this equilibrium is a stable spiral. This phenomenon of damped oscillations no matter how large the social learning rate is contrasts with [7], in which there exists a certain threshold above which the oscillations are sustained.

3.2.2 Correlating between cost and effectiveness

So far, we have been considering the perceived relative cost ρ and effectiveness on transmission e_T as independent parameters. In real life though, the effectiveness and cost of a control measure are seldom uncorrelated, as grounded by experimental studies [59, 73]. In the two following paragraphs, we explore two kinds of relations between the effectiveness and the cost. The most straightforward is a linear correlation, that is studied in the first part. Next, we postulate a quadratic correlation, that accounts for increasing marginal costs, which seems more realistic than the linear correlation studied in the previous section. This function is used as a prototype for any cost ρ that is a convex function of the effectiveness e_T . Concave functions, standing for decreasing marginal costs, seem unrealistic and are not studied.

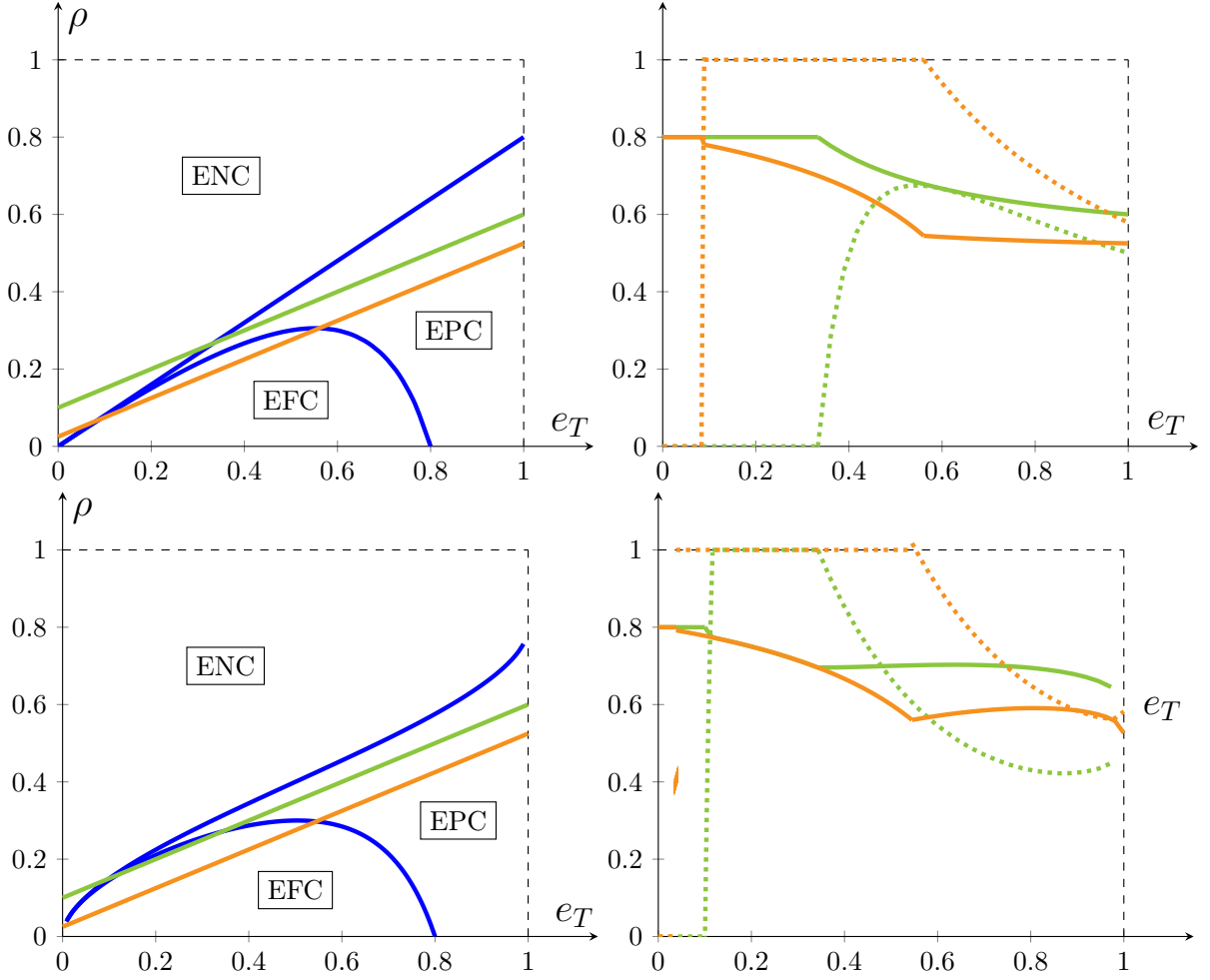


Figure 4: Selected equilibrium and implications of a linear correlation $\rho(e_T) = 0.5 \times e_T + \rho_0$ in case of unbiased (Top) or biased (Bottom) perception of the effectiveness. The bias function used is $e_T \mapsto \frac{1}{2}(\phi_\eta(e_T) + 1 - \phi_\eta(1 - e_T))$ with $\phi_\eta(e_T) = e^{-(-\log(e_T))^\eta}$. Without bias, we take $\eta = 1$. The biased case is illustrated with $\eta = \frac{2}{3}$. Left: two possible linear correlations and how they cross the stable equilibrium regions as e_T increases, with $\rho_0 = 0.1$ (green) and $\rho_0 = 0.025$ (orange). Right: corresponding fraction of compliers (dotted lines) and infected (plain lines) at equilibrium. Other parameter value: $\mathcal{R}_0 = 5$.

Linear correlation Let us consider in this paragraph that the most straightforward correlation formula

$$\rho(e_T) = \rho_0 + \ell \times e_T$$

holds. First we assume that the population accurately assesses the effect of the NPI on the reduction of the transmission, and thus adapts its choice accordingly. Mathematically, it corresponds to φ_T being the identity function. If the cost grows too fast compared with the effectiveness (ie $\rho_0 + \ell > 1 - \frac{1}{\mathcal{R}_0}$), then no one will adopt the NPI, no matter how protective it is. If, in contrast, it is moderately growing only, partial control is reachable, and if in addition ρ_0 is small enough, then full control is reachable too. We refer to Figure 4, top left, for a visualisation of these properties. In both cases, the formulae for prevalence at equilibrium are decreasing functions of the effectiveness e_T . As a consequence, to minimize prevalence, one only has to aim at the most effective NPI possible even though this leads to free riders, see Figure 4, top right.

If we include perception bias, the choice of the best NPI might be tougher. On Figure 4, bottom left, the previous three regions, modified by the perception bias, are depicted. We note in particular that unlike in the previous case, aiming at the most effective NPI is optimal for e_T close to 1, otherwise the best choice is an intermediate value, which is obtained as the largest e_T such that the whole population complies, see Figure 4, bottom right. A bias function favoring high effectiveness NPIs would lead to similar qualitative, but quantitatively different, results as in the no bias case.

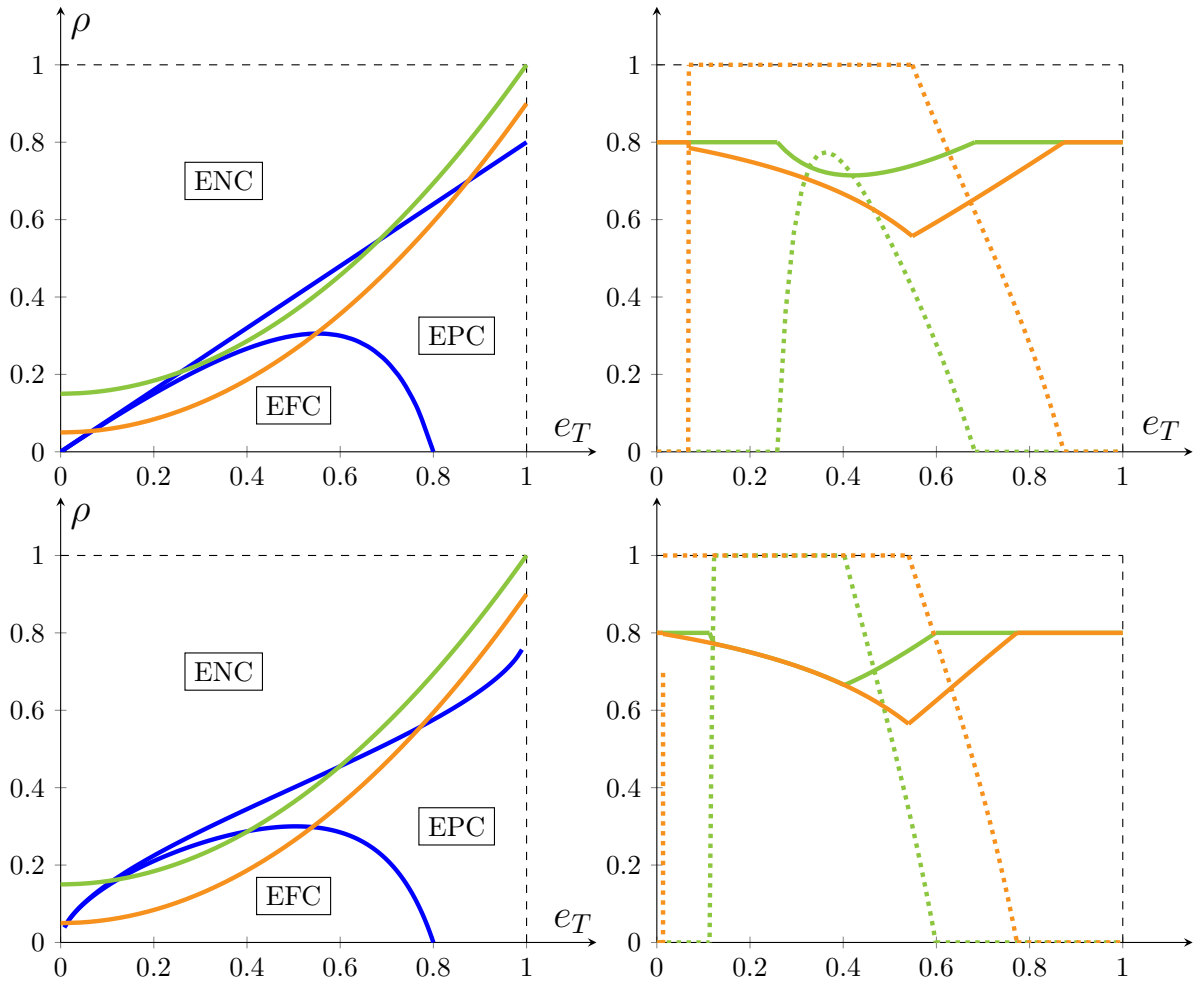


Figure 5: Selected equilibrium and implications of a quadratic correlation $\rho(e_T) = 0.85 \times e_T^2 + \rho_0$ in case of unbiased (Top) or biased (Bottom) perception of the effectiveness. The bias function used is $e_T \mapsto \frac{1}{2}(\phi_\eta(e_T) + 1 - \phi_\eta(1 - e_T))$ with $\phi_\eta(e_T) = e^{-(-\log(e_T))^\eta}$. Without bias, we take $\eta = 1$. The biased case is illustrated with $\eta = \frac{2}{3}$. Left: two possible linear correlations and how they cross the stable equilibrium as e_T increases, with $\rho_0 = 0.15$ (green) and $\rho_0 = 0.05$ (orange). Right: corresponding fraction of cooperators (plain lines) and infected (dotted lines) at equilibrium. Other parameter value: $\mathcal{R}_0 = 5$.

Quadratic correlation In this paragraph, we postulate a quadratic correlation between the effectiveness and cost, namely

$$\rho(e_T) = \rho_0 + q \times e_T^2.$$

As before, we start with the particular situation in which the population perfectly assesses the effectiveness of the prophylactic measure to reduce transmission, and makes their decision accordingly. On Figure 5 top left we depict which equilibrium is LAS, leading to three separated regions. We also depict the cost as a function of the effectiveness. If the characteristics of the NPI are small compared to the basic reproduction number, ie if $4q\rho_0 < \left(1 - \frac{1}{\mathcal{R}_0}\right)^2$, then partial control is reachable. Then again, if ρ_0 is small enough, full control is reachable. In all cases, a NPI with intermediate effectiveness and thus intermediate cost minimizes prevalence at equilibrium. If full control is achievable, we can state the following rule of thumb to find the best possible characteristics for a NPI: increase effectiveness so that the NPI is protective enough for the whole population to comply, and stop right before some individuals decide to free ride. If only partial control is possible though, the precise effectiveness to target is unclear.

As in the linear correlation case, the inclusion of perception bias modifies the boundaries between the different equilibria, see Figure 5 bottom left. In the case depicted, it enables full control for NPIs that would be otherwise too costly. In contrast, if the bias favors highly protective NPIs with respect to less protective ones, the opposite scenario could be observed, making the desirable situation of full control unachievable.

3.3 Back to the full model

In this section, we build on the intuition developed in the previous one on a simplified version on our model. We consider a general NPI acting both on transmission and susceptibility. Analogously with the 1D case, we make the assumption that its characteristics are correlated through the formula

$$\rho(e_S, e_T) = \rho_0 + q_S \times e_S^2 + q_T \times e_T^2, \quad (8)$$

which leads to various situations, depending on the relative values of the parameters and the type of bias included. In order to produce situations that can be compared, we set $q_S + q_T = 1$, meaning that decreasing marginal costs in terms of transmission increases marginal costs in terms of susceptibility and the other way round.

We note that prevalence at equilibrium can be dramatically reduced, see the bottom left and top right panels from Figure 6. In each case, the characteristics of the mask that is cheap to improve matches the mean level of altruism in the population. For instance, in the bottom left panel displays the situation for a rather selfish population, and the quadratic coefficient q_S in (8) has a low value, so improving self-protection has a limited impact on the overall cost of the mask considered. As a result, the ‘best mask’, as marked by the green cross, has a very high self-protection, and low outward protection, and prevalence at equilibrium can be reduced to less than half its value in the absence of protective behaviour. Interestingly, full compliance is not required for this minimal prevalence to be reached, see the corresponding panels from Figure 7.

The worst case scenario is represented in the central panel. This corresponds to a ‘medium’ situation, in which people are not particularly altruistic nor selfish, and it is equally expansive to increase any of the mask’s conferred protection. In this case, the population is hardly motivated to comply with mask wearing, resulting in a prevalence that is merely reduced.

4 Discussion

Including human behaviour in models of disease dynamics is a timely exciting scientific challenge. This field is expanding rapidly, as shown by the regular publication of meta-analyses [34, 35, 78, 75, 15, 8, 38, 32, 67]. The present study, which is part of this trend, provides a mechanistic derivation of a SIS epidemic model in which people can adopt a certain health behaviour or NPI, typically wear a face mask in public. A feature seldom modeled that we account for is a possible asymmetric effect of the control measure on the force of infection, with agents freely adopting or not a control measure. The decision-making process is based on imitation dynamics, in which individuals compare the payoff of the each strategy ‘defect’ or ‘comply’, both including terms related to the current state of the disease. In addition, both payoffs contain a term related to the selfish fear of becoming infected and to the altruistic one of infecting others. This peculiarity stems from the central assumption that people are unaware of their current health status. This hypothesis means infected and susceptible individuals behave in the same way, which results in a single equation accounting for payoff differences. Even with this simplifying assumption, the model cannot be reduced to a less than three-dimensional ODE system, which, up to our best knowledge, has been overlooked so far [51, 13].

Once the model is established, we performe a change of variables to obtain a more tractable set of equations, and then reduce it to a dimensionless system. The resulting system of ODEs contains a non smooth part, leading to an unusual method to analyse the local stability of the five equilibria. For any given set of parameters, only a single equilibrium can be both within the square $[0, 1] \times [0, 1]$ and be locally asymptotically stable. The three disease-free equilibria can be described by the share of the population that comply with the NPI considered. Bifurcations occur when the relative cost of the NPI crosses two bounds, that are explicitly given by parameters related to the disease, characteristics of the NPI and the degree of altruism in the population. We then study thoroughly the special case of a NPI that reduces transmission only. In this case, the model can be simplified to a system of two smooth equations. In particular, we obtain that the partial compliance equilibrium is locally stable when it exists, with no sustained oscillations possible, which contrasts with previous results [6]. This property is however only conjectured on the full model, as local asymptotic stability seems hardly amenable to a mathematical proof. The effectiveness of the NPI is then correlated to its cost, which impacts substantially the dynamics of the model, even in the special case we presented first. We postulate that the cost of the NPI is a strictly convex function of the effectiveness parameters, which accounts for increasing marginal costs to improve the NPI. As the protective ability of the NPI increases, a part of the population starts to free-ride, relying on the other’s efforts to mitigate the endemic disease. As a result, our model predicts that the best NPI to reduce disease prevalence is a moderately protective and thus moderately costly that is widely accepted. In particular, if

Prevalence at equilibrium

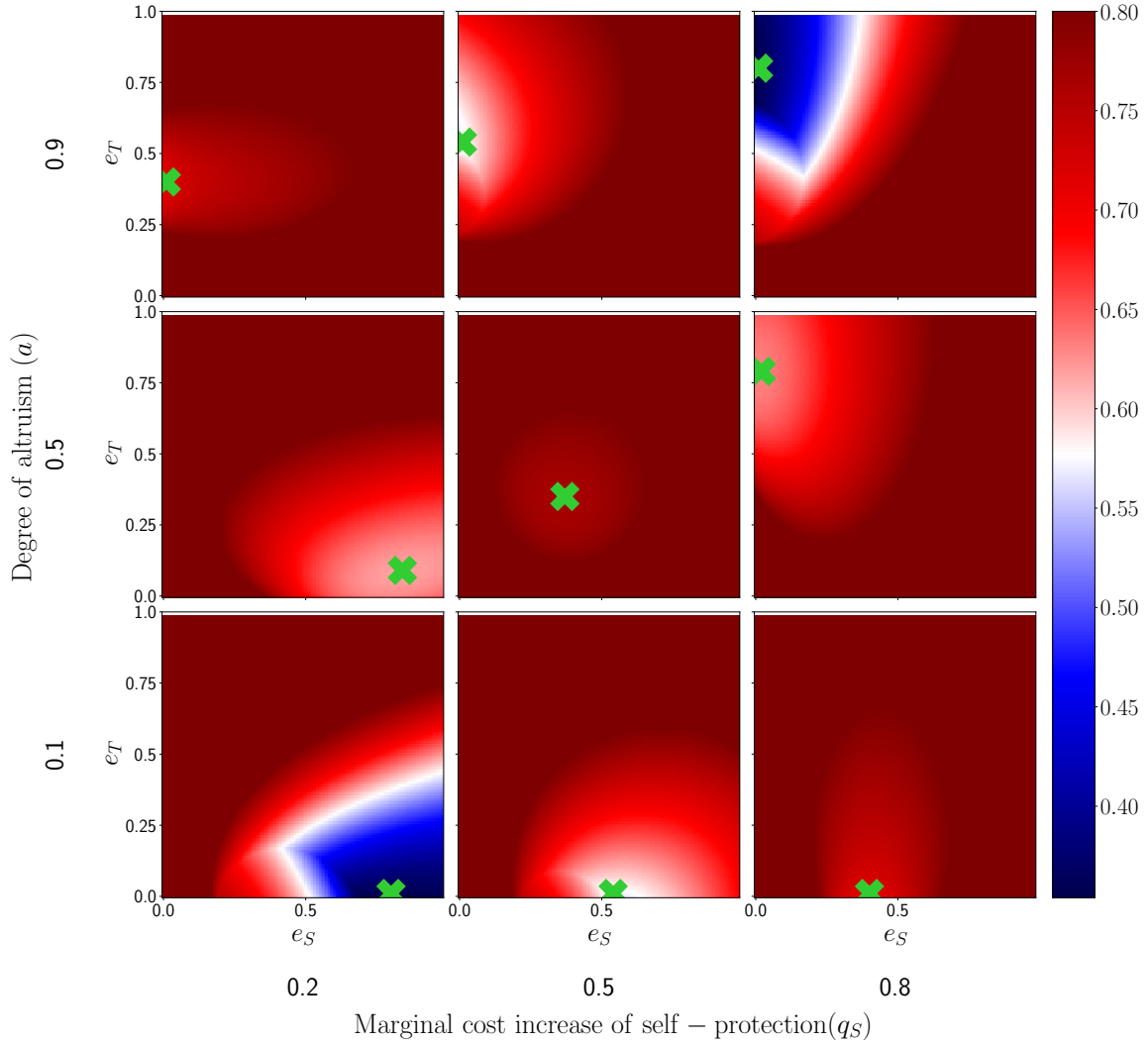


Figure 6: Prevalence in the full model for various sets of parameters. The effectiveness parameters e_S and e_T lie in $[0, 1]$ and the cost of the mask is given by the relation (8). The marginal cost increases of self- and outward-protection balance each other through the relation $q_S + q_T = 1$. Other parameters values $\mathcal{R}_0 = 5$ and $\eta = 1$ (meaning that no perception bias were included). Green crosses: effectiveness parameters that minimizes prevalence at equilibrium.

Fraction of cooperators at equilibrium

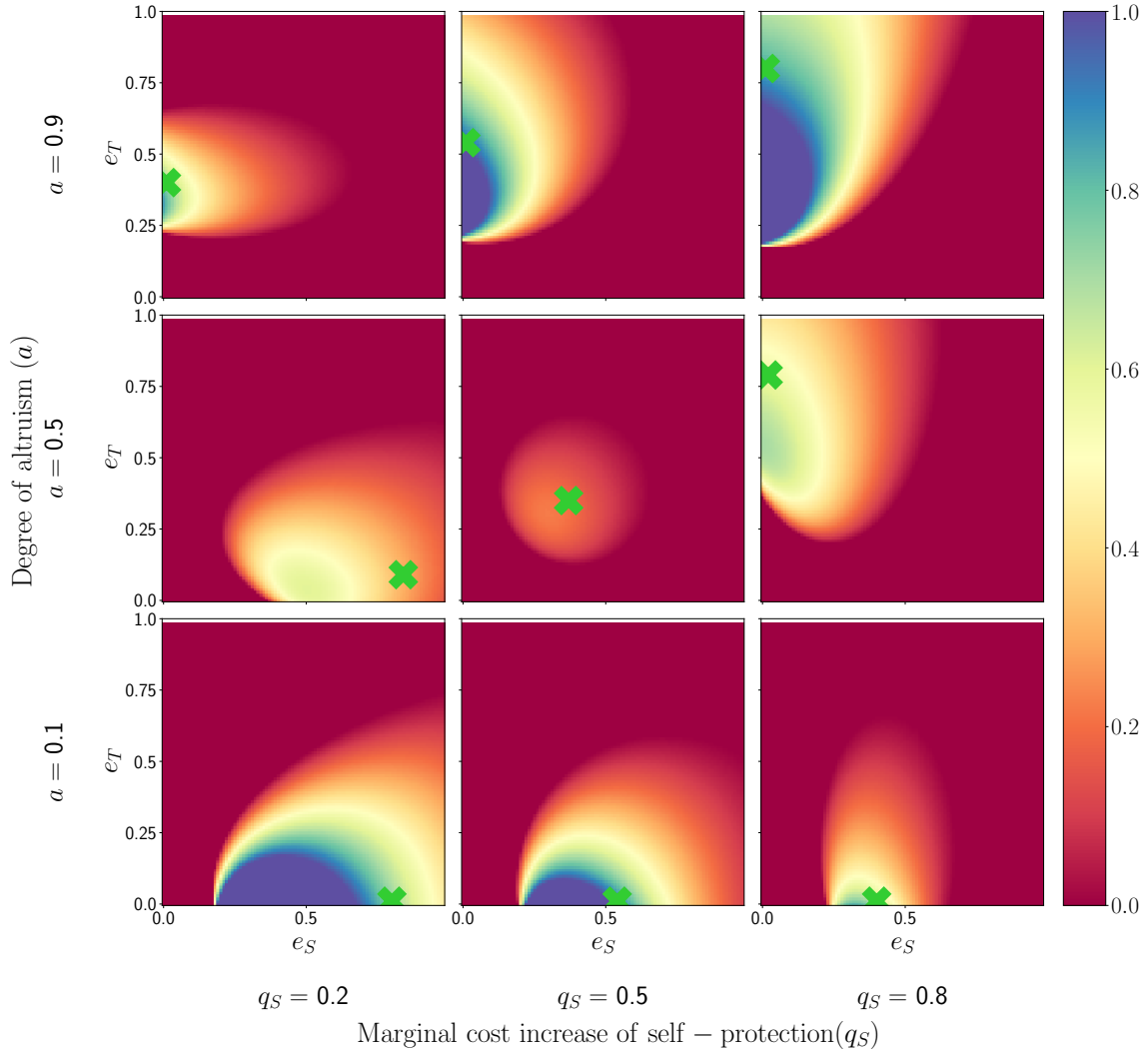


Figure 7: Fraction of compliers in the full model for various sets of parameters. The effectiveness parameters e_S and e_T lie in $[0, 1]$ and the cost of the mask is given by the relation (8). The marginal cost increases of self- and outward-protection balance each other through the relation $q_S + q_T = 1$. Other parameters values $\mathcal{R}_0 = 5$ and $\eta = 1$ (meaning that no perception bias were included). Green crosses: effectiveness parameters that minimizes prevalence at equilibrium.

the population is homogeneous in term of altruism – mostly selfish or altruistic – then the prevalence at equilibrium can be substantially reduced, provided the NPI can be improved on the related characteristic at a limited cost.

The closest model to ours is probably the one developed in [63]. In their work, Pastor-Satorras and Castellano studied an epidemic propagating on a complex network. Among the models they considered is an SIS-type one with asymmetric protection provided by the use of face masks in public. The authors concluded that if we are able to choose between inbound or outbound protection for a single type of mask to be available for the population, it is collectively better to chose for a good self protection in order to diminish the epidemic burden. As the reader may have noticed, we do not share their conclusion in the present paper. Let us mention two possible explanations – that are not mutually exclusive – for this discrepancy. First, the type of modelling we use is different from theirs. Although the authors provide a mean-field analysis of their model, it was for heterogeneous networks only. A mean-field analysis for a homogeneous network would have made the comparison straightforward, as a complete network with a large number of nodes is mimicked reasonably well with a corresponding ODE model. It is unclear to us whether our model posed on a heterogeneous network would lead to a different our conclusion. Second, and probably mostly, Pastor-Satorras and Castellano considered that a constant fraction f of the population, are made of mask-wearer. We expect the inclusion of the ability to change one’s strategy to have a tremendous effect on their model’s prediction.

In order to obtain this relatively simple model, we have to make a set of simplifying assumptions which should be relaxed in future works. For instance, a common feature in imitation dynamics is the inclusion of social pressure, that we ignored for the sake of simplicity. This would modify the payoff functions and certainly lead to the loss of monostability, see [84, 61, 51, 1, 3]. Similarly, we consider a homogeneous host population, although there exists a growing literature on how to include heterogeneity among individuals. Let us mention in particular [5], in which the authors have shown that older people may be more concerned about protecting themselves, while younger people may be more interested in protecting others. Therefore, an age-structured model would be a relevant extension of the present model, which could in addition account for heterogeneity in contact with respect to age [58] and thus on transmission [43]. When faced to decision-making, individuals have to rely on the available information. However, the same data can be interpreted differently by two people, leading to different choices. Thus, perception bias are a straightforward inclusion in game theory models [60, 26]. Another way to take such bias into account would have been to include an error in the estimation of one’s risk of a catching the disease [77, 23, 55]. Finally, the main assumption of our study is that individuals are unaware of their health-status (infected or not). Relaxing this assumption (as in, *e.g.*, [81]) would challenge the simplifications we are allowed to perform to arrive at the model we analyzed in the present study, and is therefore left for future research.

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Declarations

Conflict of interest We have no conflict of interest.

Ethical approval Not applicable.

CRedit authorship contribution statement **Hugo Martin**: Conceptualization, Methodology, Investigation, Writing – Original Draft, Writing – Reviewing and Editing, Visualization, Funding acquisition **François Castella** Conceptualization, Methodology, Writing – Reviewing and Editing **Frédéric Hamelin** Conceptualization, Methodology, Writing – Reviewing and Editing, Funding acquisition

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A Analysis of the dynamics of the model

We recall model (5) for convenience. It reads

$$\begin{cases} \frac{di}{dt} = \mathcal{R}_0(1 - e_{Sc} - i(1 - e_{Scy}))(1 - e_{Tcy})i - i =: F(i, c, y) \\ \frac{dc}{dt} = \kappa c(1 - c)(\mathcal{A}(1 - \mathcal{H}c)i - \rho) =: G(i, c, y) \\ \frac{dy}{dt} = \mathcal{R}_0(1 - e_{Tcy})(1 - y - e_s(1 - iy)(1 - cy)) + \kappa(1 - y)(\mathcal{A}(1 - \mathcal{H}c)i - \rho)_+ =: H(i, c, y). \end{cases}$$

A.1 General considerations on the model

Lemma 1. *The biologically admissible subset of \mathbb{R}^3 for Model (5) is included in*

$$\left\{ (i, c, y) \mid i, c \in [0, 1], y \in \left[0, \min\left(\frac{1}{c}, \frac{1}{i}\right)\right] \right\}$$

and for any i, c and $y > 1$ in this set, one has $H(i, c, y) < 0$.

Proof. Using (2) and (3), we readily obtain that i and c belong to $[0, 1]$, since all of S_C, S_D, I_C and I_D lie in the interval $[0, N]$, with $N = S_C + S_D + I_C + I_D$. In particular, the non negativity condition on I_D provides

$$\begin{aligned} i - \frac{I_C}{N} \geq 0 &\iff i - icy \geq 0 \iff y \leq \frac{1}{c} \\ c - \frac{I_C}{N} \geq 0 &\iff c - icy \geq 0 \iff y \leq \frac{1}{i} \end{aligned}$$

which proves our claim on the admissible set. In turn, this leads to the second claim since for such (i, c, y) one has $(1 - iy)(1 - cy) > 0$ and thus the corresponding $H(i, c, y)$ is negative whenever $y > 1$. \square

Lemma 2. *The cube $[0, 1] \times [0, 1] \times [0, 1]$ is stable under the flow associated with System (5).*

Proof. The equation on the variable c yields that the sides corresponding to $c = 0$ and $c = 1$ are stable under the flow, so starting in $[0, 1]$, the variable c remains in this interval. The same type of argument prevents the variable i to cross the side $i = 0$. Now on the side $i = 1$, the flow in the direction i is entering the cube if

$$(1 - e_{Sc} - (1 - e_{Scy}))(1 - e_{Tcy}) < \frac{1}{\mathcal{R}_0}$$

which is equivalent to

$$Q_c(y) := e_{Tcy}^2 - (1 + e_{Tc})y + 1 + \frac{1}{e_{Sc}\mathcal{R}_0} > 0$$

on $[0, 1]$, uniformly on $c \in (0, 1]$. To see this, we compute the derivative

$$Q'_c(y) = 2e_{Tcy} - (1 + e_{Tc}) = e_{Tc}(y - 1) + (e_{Tcy} - 1) < 0$$

and combine it with $Q_c(1) = \frac{1}{e_{Sc}\mathcal{R}_0} \geq \frac{1}{e_S\mathcal{R}_0} > 0$ which allows to conclude that the interval $[0, 1]$ for i is conserved by the flow. Finally for the sides $y = 0$ and $y = 1$, we compute

$$H(i, c, 0) \geq \mathcal{R}_0(1 - e_S) > 0 \quad \text{and} \quad H(i, c, 1) = -\mathcal{R}_0(1 - e_{Tc})e_S(1 - i)(1 - c) \leq 0 \quad \text{on } [0, 1] \times [0, 1]$$

so the proof is complete. \square

These two lemmas combined lead us to consider initial conditions $(i(0), c(0), y(0))$ in the square $[0, 1]^3$.

A.2 Equilibria and their stability

In this section, we provide the mathematical proofs for existence and stability of the equilibria. To study whether these equilibria are locally stable, it is usual to perform linearization by mean of the jacobian matrix and then check its eigenvalues. This standard procedure cannot apply in our case, due to the positive part that is not differentiable. Instead, we consider any equilibrium (i^*, c^*, y^*) and introduce the variables (z_1, z_2, z_3) such that $i = i^* + z_1$, $c = c^* + z_2$ and $y = y^* + z_3$. They satisfy

$$\left\{ \begin{array}{l} \frac{dz_1}{dt} = F(i^* + z_1, c^* + z_2, y^* + z_3) \approx \nabla F(i^*, c^*, y^*) \cdot \begin{pmatrix} z_1 \\ z_2 \\ z_3 \end{pmatrix} + H.O.T. \\ \frac{dz_2}{dt} = G(i^* + z_1, c^* + z_2, y^* + z_3) \approx \nabla G(i^*, c^*, y^*) \cdot \begin{pmatrix} z_1 \\ z_2 \\ z_3 \end{pmatrix} + H.O.T. \\ \frac{dz_3}{dt} = \mathcal{R}_0 Q(i^* + z_1, c^* + z_2, y^* + z_3) + \kappa(1 - (y^* + z_3))(\mathcal{A}(1 - \mathcal{H}(c^* + z_2))(i^* + z_1) - \rho)_+ \\ \approx \mathcal{R}_0 \nabla Q(i^*, c^*, y^*) \cdot \begin{pmatrix} z_1 \\ z_2 \\ z_3 \end{pmatrix} + \kappa(1 - (y^* + z_3))(\mathcal{A}(1 - \mathcal{H}(c^* + z_2))(i^* + z_1) - \rho)_+ + H.O.T. \end{array} \right. \quad (9)$$

in which we do not specify higher order terms (H.O.T.) involving functions of (z_1, z_2, z_3) having degree ≥ 2 , and we do not differentiate the term with the positive part in the third equation. Local stability near $(z_1, z_2, z_3) = (0, 0, 0)$ hence provides the local stability of System (5) near $(i, c, y) = (i^*, c^*, y^*)$. For the two disease-free equilibria, the following proposition holds.

Proposition 3. *The disease-free equilibrium with full cooperation is never locally stable. The disease-free with no cooperation is globally asymptotically stable if and only if $\mathcal{R}_0 \leq 1$.*

Proof. Plugging the equilibrium $(i^*, c^*, y^*) = (0, 1, 1)$ into the second line of System (9) leads to

$$\frac{dz_2}{dt} \approx \kappa \rho z_2 + H.O.T.$$

so this equilibrium is never stable. Next we turn to equilibrium $(i^*, c^*, y^*) = (0, 0, 1 - e_S)$. We can considered an initial data close enough to $(z_1, z_2, z_3) = (0, 0, 0)$ so that $\mathcal{A}(1 - \mathcal{H}z_2(0))z_1(0) - \rho \leq 0$. In such a case, the positive part in the equation on z_3 vanishes and the system to study reduces to

$$\left\{ \begin{array}{l} \frac{dz_1}{dt} \approx (\mathcal{R}_0 - 1)z_1 \\ \frac{dz_2}{dt} \approx -\kappa \rho z_2 \\ \frac{dz_3}{dt} \approx \mathcal{R}_0(e_S(1 - e_S)(z_1 + z_2) - z_3). \end{array} \right.$$

When $\mathcal{R}_0 < 1$, both z_1 and z_2 are decreasing exponential functions, hence the method of variation of constants states that z_3 is a sum of such functions. As a result, $(0, 0, 1 - e_S)$ is locally asymptotically stable. In the case $\mathcal{R}_0 = 1$, we rewrite the first line of System (5) as

$$\frac{di}{dt} = (1 - e_S c x)(1 - e_T c y)(1 - i)i - i \leq (1 - i)i - i = -i^2$$

so we obtain by direct integration

$$i(t) \leq \frac{1}{\frac{1}{i(0)} + t}$$

so i decreases toward 0. Since the disease-free with full cooperation equilibrium is never stable, the only possible equilibrium is $(0, 0, 1 - e_S)$. \square

We study now the local stability of endemic equilibria, so from now on we assume $\mathcal{R}_0 > 1$.

Lemma 4. *The endemic with no compliance equilibrium is stable if and only if $\frac{\rho}{\mathcal{A}} > 1 - \frac{1}{\mathcal{R}_0}$. In addition, its expression reduces to*

$$\left(1 - \frac{1}{\mathcal{R}_0}, 0, \frac{1}{1 + \frac{e_S}{\mathcal{R}_0(1 - e_S)}} \right).$$

Proof. For the above mentioned equilibrium, System (9) provides

$$\begin{cases} \frac{dz_1}{dt} \approx (1 - \mathcal{R}_0)(z_1 + K_1 z_2) + H.O.T. \\ \frac{dz_2}{dt} \approx \kappa \left(\mathcal{A} \left(1 - \frac{1}{\mathcal{R}_0} \right) - \rho \right) z_2 + H.O.T. \\ \frac{dz_3}{dt} \approx \mathcal{R}_0(e_S y^* z_1 + K_2 z_2 - (1 - e_S i^*) z_3) + \kappa(1 - (y^* + z_3)) (\mathcal{A}(1 - \mathcal{H} z_2)(i^* + z_1) - \rho)_+ + H.O.T. \end{cases}$$

with K_1 and K_2 constants given by

$$K_1 = e_S(1 - i^* y^*) + e_T y^*(1 - i^*) > 0 \quad \text{and} \quad K_2 = y^*((e_S + e_T)(1 - i^* y^*) - e_T(1 - y^*)) > 0.$$

Since $\frac{\rho}{\mathcal{A}} > 1 - \frac{1}{\mathcal{R}_0}$, z_2 is a decreasing exponential function, and thus z_1 is a sum of such functions, since $\mathcal{R}_0 > 1$. Due to the expressions of z_1 and z_2 , for any $\varepsilon > 0$, there exists a time $T(\varepsilon) > 0$, where $T(\varepsilon)$ is of the order of magnitude of ε such that for any $t \geq T(\varepsilon)$, one has $|z_1(t)|, |z_2(t)| < \varepsilon < 1$. As a result, for such times t , one has

$$(*) := \mathcal{A}(1 - \mathcal{H} z_2)(i^* + z_1) - \rho < \underbrace{\mathcal{A} \left(1 - \frac{1}{\mathcal{R}_0} \right) - \rho}_{< 0} + \varepsilon \mathcal{A} \left(1 + \mathcal{H} \left(1 - \frac{1}{\mathcal{R}_0} \right) + \mathcal{H} \varepsilon \right)$$

so for ε small enough, $(*)$ is negative. Thus, starting from this time, the equation on z_3 reduces to

$$\frac{dz_3}{dt} \approx \mathcal{R}_0(e_S y^* z_1 + K_2 z_2 - (1 - e_S i^*) z_3) + H.O.T.$$

which allows us to write z_3 as the sum of three decreasing exponential functions, so the equilibrium is locally asymptotically stable. \square

Now, we examine the endemic equilibrium with full cooperation.

Lemma 5. *The endemic with full compliance equilibrium is locally asymptotically stable whenever*

$$\frac{\rho}{\mathcal{A}} < (1 - \mathcal{H}) \left(1 - \frac{1}{\mathcal{R}_0(1 - e_S)(1 - e_T)} \right).$$

In particular, this condition ensures existence of the equilibrium, for which the minimal assumption for existence is

$$\mathcal{R}_0(1 - e_S)(1 - e_T) > 1.$$

Proof. The local stability of this equilibrium is provided by the behavior of the system

$$\begin{cases} \frac{dz_1}{dt} \approx (1 - \mathcal{R}_0(1 - e_S)(1 - e_T))(z_1 + K_1 z_2 + K_2 z_3) + H.O.T. \\ \frac{dz_2}{dt} \approx -\kappa \left(\mathcal{A}(1 - \mathcal{H}) \left(1 - \frac{1}{\mathcal{R}_0(1 - e_S)(1 - e_T)} \right) - \rho \right) z_2 + H.O.T. \\ \frac{dz_3}{dt} \approx \mathcal{R}_0(1 - e_T)[e_S(1 - i^*) z_2 - (1 - e_S(1 - i^*)) z_3] - \kappa z_3 (\mathcal{A}(1 - \mathcal{H}(1 + z_2))(i^* + z_1) - \rho)_+ + H.O.T. \end{cases}$$

with

$$K_1 = \left(\frac{e_S}{1 - e_S} + \frac{e_T}{1 - e_T} \right) (1 - i^*) > 0$$

$$K_2 = \left(\frac{e_S}{1 - e_S} + \frac{e_T}{1 - e_T} \right) i^* + \frac{e_T}{1 - e_T} > 0.$$

Since $\frac{\rho}{\mathcal{A}} < (1 - \mathcal{H}) \left(1 - \frac{1}{\mathcal{R}_0(1 - e_S)(1 - e_T)} \right)$, z_2 is a decreasing exponential function. Thus, for z_1 small enough, $\mathcal{A}(1 - \mathcal{H}(1 + z_2))(i^* + z_1) - \rho$ is negative, and as a result z_3 is a sum of decreasing exponential functions. Finally, z_1 is also a sum of decreasing exponential functions. \square

Finally, we move on the equilibrium with partial cooperation.

Proposition 6. *Under the condition*

$$(1 - \mathcal{H}) \left(1 - \frac{1}{\mathcal{R}_0(1 - e_S)(1 - e_T)} \right) < \frac{\rho}{\mathcal{A}} < 1 - \frac{1}{\mathcal{R}_0} \quad (10)$$

there exists at least one endemic equilibrium with partial compliance.

Proof. Let (i^*, c^*, y^*) denote the EPC equilibrium, thus with $c^* \in (0, 1)$. The condition $G(i^*, c^*, y^*) = 0$ leads to the relation $\mathcal{A}(1 - \mathcal{H}c^*)i^* - \rho = 0$ which is equivalent to

$$i^* = \frac{\rho}{\mathcal{A}(1 - \mathcal{H}c^*)}.$$

Due to this relation, cancelling function H is equivalent to

$$1 - y^* - e_S(1 - i^*y^*)(1 - c^*y^*) = 0$$

which can be rewritten as

$$i^* = \frac{1}{y^*} \left(1 - \frac{1 - y^*}{e_S(1 - c^*y^*)} \right).$$

Equating these two expressions provides

$$e_S \frac{\rho}{\mathcal{A}} c^* (y^*)^2 + \left((1 - e_S c^*)(1 - \mathcal{H}c^*) - e_S \frac{\rho}{\mathcal{A}} \right) y^* - (1 - e_S)(1 - \mathcal{H}c^*) = 0$$

which has a single positive solution given as a function of c^* by $Y(c^*)$ defined as

$$Y(c) = \frac{\sqrt{\left((1 - e_S c)(1 - \mathcal{H}c) - \frac{\rho}{\mathcal{A}} e_S \right)^2 + 4e_S(1 - e_S) \frac{\rho}{\mathcal{A}} c(1 - \mathcal{H}c) - (1 - e_S c)(1 - \mathcal{H}c) + \frac{\rho}{\mathcal{A}} e_S}}{2 \frac{\rho}{\mathcal{A}} e_S c}.$$

In addition, one has the equivalences

$$Y(c) < 1 \iff \frac{\rho}{\mathcal{A}(1 - \mathcal{H}c)} < 1$$

and

$$Y(c) = 1 \iff \left(\frac{\rho}{\mathcal{A}(1 - \mathcal{H}c)} = 1 \quad \text{or} \quad c = 1 \right).$$

It remains to prove that there exists $c^* \in (0, 1)$ such that

$$\frac{\rho}{\mathcal{A}(1 - \mathcal{H}c^*)} \leq 1 \quad \text{and} \quad F \left(\frac{\rho}{\mathcal{A}(1 - \mathcal{H}c^*)}, c^*, Y(c^*) \right) = 0.$$

This last condition is equivalent to

$$\tilde{F}(c^*) := \left(1 - e_S c^* - \frac{\rho}{\mathcal{A}(1 - \mathcal{H}c^*)} (1 - e_S c^* Y(c^*)) \right) (1 - e_T c^* Y(c^*)) = \frac{1}{\mathcal{R}_0}.$$

We compute

$$\tilde{F}(0) = \left(1 - \frac{\rho}{\mathcal{A}} \right) > \frac{1}{\mathcal{R}_0}$$

and

$$\tilde{F}(1) = (1 - e_S) \left(1 - \frac{\rho}{\mathcal{A}(1 - \mathcal{H})} \right) (1 - e_T) < \frac{1}{\mathcal{R}_0}$$

thanks to Assumption (10) so the claim is proved. \square

Conjecture 1. *The EPC equilibrium is unique.*

This conjecture has been explored numerically by testing the decay of function \tilde{F} for a wide set of parameters, see the code provided on the GitHub page related to this study.

Conjecture 2. *The EPC equilibrium is locally asymptotically stable whenever it exists.*

B Local stability for the special case of outbound protection only

When $e_S = 0$, the equation on y reads

$$\frac{dy}{dt} = (1 - y) (\mathcal{R}_0(1 - e_T c y) + \kappa (\mathcal{A}(1 - \mathcal{H}c)i - \rho)_+).$$

For $y(0) \leq 1$, let us introduce $z := 1 - y$. One has

$$\frac{dz}{dt} \leq -z\mathcal{R}_0((1 - e_T) - e_T z) \leq -z\mathcal{R}_0(1 - e_T)$$

since both c and y are bounded from above by 1, and Grönwall's lemma ensures that

$$z(t) \leq z(0)e^{-\mathcal{R}_0(1 - e_T)t}.$$

Now if $y(0) \in \left(1, \frac{1}{e_T} - 1\right)$, we introduce $z := y - 1$, and obtain the estimate

$$\frac{dz}{dt} \leq -z\mathcal{R}_0((1 - e_T) - e_T z) \leq -z\mathcal{R}_0((1 - e_T) - e_T z(0))$$

so again once Grönwall's lemma ensures exponential decay toward 0. As a result, after short time, the full system is expected to behave like the reduced system

$$\begin{cases} \frac{di}{dt} = \mathcal{R}_0(1 - e_T c)(1 - i)i - i \\ \frac{dc}{dt} = \kappa c(1 - c)(\alpha\varphi_T(e_T)i - \rho) \end{cases}$$

that is now completely smooth. Thus, this one can be studied with a classical method of Jacobian matrix. For the sake of completeness, we also include the proofs for equilibria that were already studied in the general case. For any $(i, c) \in [0, 1]^2$, this matrix reads

$$J_{(i,c)} = \begin{pmatrix} \mathcal{R}_0(1 - e_T c)(1 - 2i) - 1 & -\mathcal{R}_0 e_T(1 - i)i \\ \kappa \mathcal{A} c(1 - c) & \kappa(1 - 2c)(\mathcal{A}i - \rho) \end{pmatrix}$$

and we have to evaluate it at each equilibrium point. Note that in this case, one has the simpler expressions for \mathcal{A} and \mathcal{H} given by

$$\mathcal{A} = \alpha\varphi_T(e_T) \quad \text{and} \quad \mathcal{H} = 0.$$

Let us now review all equilibria in the planar case.

- Disease Free equilibrium with No compliance (DFN):

One has

$$J_{(0,0)} = \begin{pmatrix} \mathcal{R}_0 - 1 & 0 \\ 0 & -\kappa\rho \end{pmatrix}$$

thus *DFC* is stable when $\mathcal{R}_0 < 1$. The case $\mathcal{R}_0 = 1$ can be treated as in the full system case and leads to the same stability. In any other case, it is a saddle point. In addition,

$$\text{Tr}(J_{(0,0)})^2 - 4 \det(J_{(0,0)}) = (\mathcal{R}_0 - 1 + \kappa\rho)^2 > 0$$

so when $\mathcal{R}_0 \leq 1$, *DFN* is a stable node.

- Disease Free equilibrium with full Compliance (DFC):

$$J_{(0,1)} = \begin{pmatrix} \mathcal{R}_0(1 - e_T) - 1 & 0 \\ 0 & \kappa\rho \end{pmatrix}$$

thus *DFC* is never stable.

- Endemic equilibrium with No Compliance (ENC):

The first endemic equilibrium *ENC* has jacobian matrix

$$J_{\left(1 - \frac{1}{\mathcal{R}_0}, 0\right)} = \begin{pmatrix} 1 - \mathcal{R}_0 & * \\ 0 & \kappa \left(\mathcal{A} \left(1 - \frac{1}{\mathcal{R}_0} \right) - \rho \right) \end{pmatrix}$$

with * some coefficients that have no influence on the stability of the equilibrium. This matrix has two negative eigenvalues if and only if

$$\mathcal{R}_0 > 1 \quad \text{and} \quad \frac{\rho}{\mathcal{A}} > 1 - \frac{1}{\mathcal{R}_0}.$$

In addition, the Jacobian matrix is upper triangular, so as in the *DFN* case, when it exists, *ENC* is a stable node.

- Endemic equilibrium with Full Compliance (EFC):

Then the *EFC* equilibrium provides the jacobian matrix

$$J_{\left(1 - \frac{1}{\mathcal{R}_0(1-e_T)}, 1\right)} = \begin{pmatrix} 1 - \mathcal{R}_0(1 - e_T) & * \\ 0 & -\kappa \left(\mathcal{A} \left(1 - \frac{1}{\mathcal{R}_0(1-e_T)} \right) - \rho \right) \end{pmatrix}$$

so this equilibrium is a stable node if and only if

$$\mathcal{R}_0(1 - e_T) > 1 \quad \text{and} \quad \frac{\rho}{\mathcal{A}} < 1 - \frac{1}{\mathcal{R}_0(1 - e_T)}.$$

In addition, the Jacobian matrix is upper triangular, so as in the *ENC* case, when it exists, *EFC* is a stable node.

- Endemic equilibrium with Partial Compliance (EPC):

The last equilibrium is the only one that lies in the interior of the square $[0, 1] \times [0, 1]$ and has no explicit formulation in the general case. Yet, in this specific case $e_S = 0$, we can obtain such an expression. It is obtained as the intersection of the nullclines of the system

$$\begin{cases} 0 = \mathcal{R}_0(1 - e_T c_*)(1 - i_*) - 1 \\ 0 = \mathcal{A}i_* - \rho \end{cases}$$

which leads to the formulae

$$i_* = \frac{\rho}{\mathcal{A}} \quad \text{and} \quad c_* = \frac{1}{e_T} \left(1 - \frac{1}{\mathcal{R}_0 \left(1 - \frac{\rho}{\mathcal{A}} \right)} \right)$$

and the existence and stability condition

$$1 - \frac{1}{\mathcal{R}_0(1 - e_T)} < \frac{\rho}{\mathcal{A}} < 1 - \frac{1}{\mathcal{R}_0}.$$

Observe that these bounds match those of the *ENC* and *EFC* equilibria, so only one equilibrium at a time can be stable. The jacobian matrix at *EPC* is given by

$$J_{(i_*, c_*)} = \begin{pmatrix} -\frac{i_*}{1 - i_*} & -\mathcal{R}_0 e_T (1 - i_*) i_* \\ \kappa \mathcal{A} c_* (1 - c_*) & 0 \end{pmatrix}$$

whose trace is always negative. Its determinant is

$$\det(J_{(i_*, c_*)}) = \kappa \mathcal{A} \mathcal{R}_0 e_T c_* (1 - c_*) (1 - i_*) i_*$$

which is positive, so *EPC* is always stable whenever it exists. Unlike the other equilibria that can be only nodes when they are stable, *EPC* might be a stable spiral. This happens when κ is large enough, *i.e.* when the population update their opinions fast enough. Indeed, we can compute

$$Tr(J_{(i_*, c_*)})^2 - 4 \det(J_{(i_*, c_*)}) = \left(\frac{i_*}{1 - i_*} \right)^2 - 4 \kappa \mathcal{A} \mathcal{R}_0 e_T c_* (1 - c_*) (1 - i_*) i_*$$

which can be negative for κ large enough.

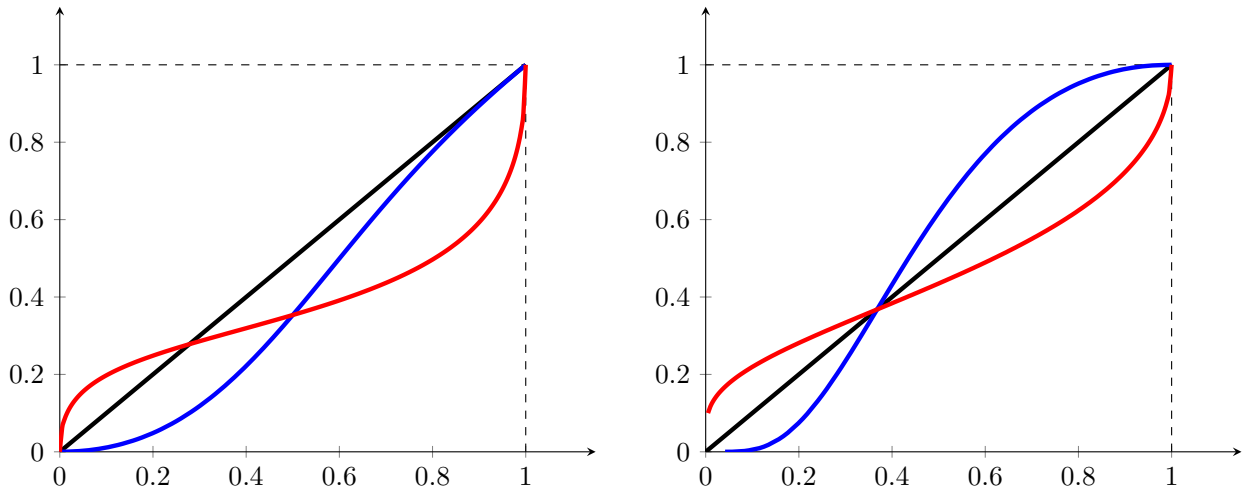


Figure 8: Plots of possible perception bias functions for $\eta = 2$ (blue), $\eta = 1$ (black) and $\eta = 0.5$ (red). Left: plots of φ_η . Right: plots of ϕ_η .

C Bias functions

As mentioned in the main text, Prospect Theory usually considers that people tend to overestimate the chance of rare events and underestimate that of common ones. As a result, some functions mapping the actual probability to its perceived probability were proposed. For example, in [60] the authors use functions of the form

$$\varphi_\eta(x) = \frac{x^\eta}{(x^\eta + (1-x)^\eta)^{\frac{1}{\eta}}}$$

with $\eta < 1$. Another possibility is proposed in a recent paper [26] that also mingles Prospect Theory with epidemiology to obtain a rich model, using functions of the form

$$\phi_\eta(x) = \exp(-(-\log x)^\eta)$$

with $\eta < 1$. Depictions of such functions for three values of η are given in Figure 8. In our model, we use these functions for a different purpose: we want to account for a biased perception of the actual capability of a given NPI to reduce transmission. This bias can be either an overestimation of moderate protection and underestimation of higher ones or the opposite. In addition, we assume that the effect of a protection that reduces 0% or 100% of the transmission is accurately evaluated, so the bias function must satisfy $\phi(0) = 0$ and $\phi(1) = 1$. For simplicity, we make the assumption that the switch between under- and overestimation occurs at $e = 0.5$. These conditions are fulfilled for instance by a function of the form

$$\phi(e) = \frac{1}{2} (\phi_\eta(e) + 1 - \phi_\eta(1 - e)) \quad (11)$$

with ϕ_η introduced above. Corresponding plots are displayed on Figure 9, right. For completeness, we plotted the combined bias formula using φ_η instead of ϕ_η on Figure 9, left.

D Code availability

The Python codes used throughout this paper are available at the following GitHub repository: <https://github.com/Hugo-Martin/SIS-imitation.git>.

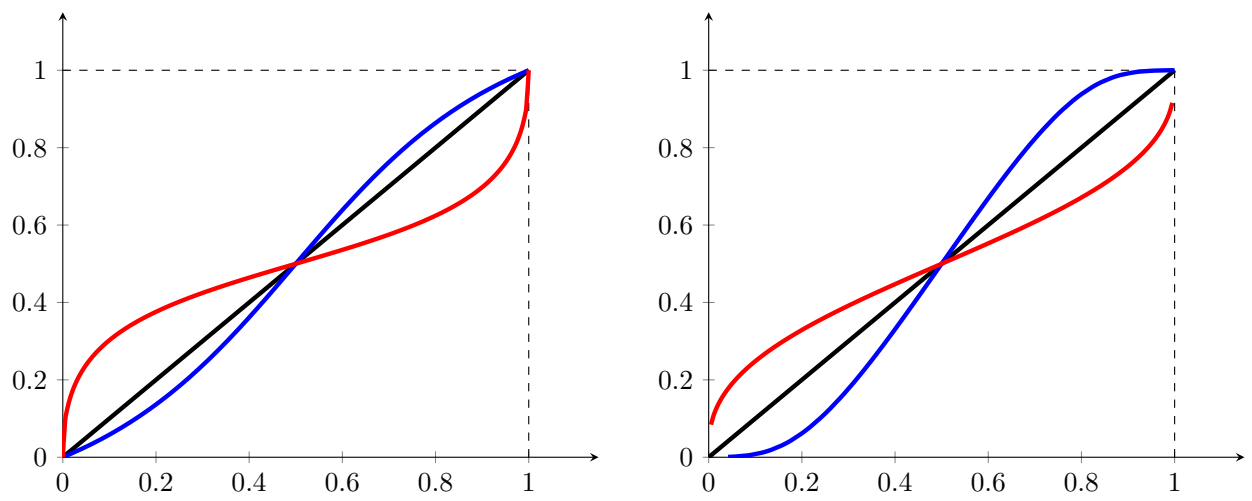


Figure 9: Plots of possible perception bias functions of the form (11) for $\eta = 2$ (blue), $\eta = 1$ (black) and $\eta = 0.5$ (red). Left: plots of combined bias using φ_η . Right: plots of combined bias using ϕ_η .