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Peer effects and peer avoidance: epidemic diffusion in coevolving networks*

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Abstract

We study the long-run emergence of behavioral patterns in dynamic complex networks. Individuals display two kinds of behavior: G ("good") or B ("bad"). We assume that agents have an innate tendency towards G, but can also be led towards B through the influence of peer bad behavior. We model the implications of those peer effects as an epidemic process in the standard SIS (Susceptible-Infected-Susceptible) framework. The key novelty of our model is that, unlike in received epidemic literature, the network is taken to change over time within the same time scale as behavior. Specifically, we posit that links connecting two G agents last longer, reflecting the idea that B agents tend to be avoided. The main concern of the paper is to understand the extent to which such biased network turnover may play a significant role in supporting G behavior in a social system. And indeed we find that network coevolution has nontrivial and interesting effects on long-run behavior. This yields fresh insights on the role of (endogenous) peer pressure on the diffusion of (a)social behavior as well as on the traditional study of disease epidemics.

Keywords: Coevolutionary networks, diffusion of behavior, social dilemma, epidemics.

JEL Classification: C71, D83, D85.

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"Company, villainous company,
hath been the spoil of me."
W. Shakespeare, Henry IV, Part I.

1 Introduction

We study the long-run emergence of behavior in dynamic complex networks. Individuals display two kinds of behavior: G ("good") or B ("bad") which, depending on the context, can be respectively conceived as behavior that is either social/cooperative or asocial/hostile. We suppose that the population of agents is large, but each of them interacts with a typically small group. As customary, therefore, the prevailing pattern of interaction is modelled as a large social network. The objective is to understand under what conditions B or G will spread and dominate the overall population. More specifically, we address the question of whether the adjustment of the network – which is taken to reflect the individual efforts to avoid "villainous company" – may prevent or contain the spread of bad behavior. The leading interpretation of our model is social, but it obviously admits an alternative one in terms of disease epidemics as well. Indeed, as we shall explain below, our approach addresses concerns that are also at the center of modern epidemiological theory.

The model couples two dynamics. First, the dynamics by which agents change their behavior. This dynamics is conceived as a diffusion process, in which B spreads through the local contact and *influence of peers*. Formally, we adopt one of the paradigmatic frameworks in epidemiological theory: the so-called SIS (susceptible-infected-susceptible) model.[1] In this setup, the strength of the peer effects inducing the switch to B is linear in the number of B neighbors a G agent has. B agents, in turn, have an internal tendency to switch back to G, independently of their neighborhood conditions. In a stylized sense, one could interpret this feature as reflecting a Rousseaunian view of human nature, which is taken to intrinsically lead to good behavior unless spoiled by bad social influence. Second, the dynamics by which the network evolves. New links are randomly created while on going contacts' decay depends on partners' behavior. Specifically, we posit that links connecting two G agents last longer, reflecting the idea that B agents tend to be avoided.

Let us now summarize our conclusions. The model has three main parameters:

- the rate λ at which peer effects turn G into B;

[1] See, for example, Newman (2002) [14] and Pastor-Satorras and Vespignani (2001) [17] for a detailed discussion of this model in the framework provided by the modern theory of complex networks.

- the differential rate γ at which peer avoidance destroys links connecting to B agents;
- the rate τ of "social turnover" at which the network adjusts, thus modulating the relative speed of peer effects and peer avoidance.

For the sake of focus, we fix γ at different levels and explore in detail the interplay between λ and τ. Our main concern, in particular, is to understand how these parameters affect the long-run frequency ρ of agents displaying B – i.e. what, using the terminology common in epidemiological models, we shall often call *prevalence.* As a first step, we obtain a conclusion that is formally akin to that obtained in standard SIS models. Given τ (and γ), there is a critical value λ_c marking a continuous transition in the long-run prevalence of bad behavior, i.e. its limit value ρ is positive iff (the strength of) peer influence exceeds that critical value *and* the gradient changes discontinuously at λ_c.

The more interesting results concern the effect of varying the pace at which the network adjusts. In particular, we study the implications on two different (endogenous) magnitudes. First, we consider how τ affects the critical rate λ_c that establishes the onset of long-run prevalence of bad behavior. And we find, at first sight somewhat surprisingly, that the effect is not always monotone. This indeed depends on the value of γ. If γ is high (we shall refer to the situation as Regions I and II, since there are also other parameters involved), then as τ grows so happens with λ_c. This reflects the natural idea that faster turnover checks the prevalence of bad behavior. But, interestingly, we also find that the conclusion is not always so clear-cut. For in the complementary region in parameter space (Region III), the behavior turns out to be non-monotone, with an interior value of τ where λ_c is maximal.

To understand why such non-monotonicity may arise, it is useful to bear in mind that a higher degree of social turnover (network adjustment) has a two-fold implication. On the one hand, of course, it reinforces the creation and maintenance of GG links at the expense of other type of links, thus tending to prevent B agents from exerting their detrimental peer pressure. But this, in turn, creates the network conditions (i.e. long paths of G agents) on which a "seed" of B behavior can rapidly expand. Thus, it seems intuitive that, under suitable parameter conditions, the complex interplay of those considerations can produce non-monotone effects.

In fact, a further interesting manifestation of such complexity can be found concerning the effect of the turnover rate τ on the extent of long-run prevalence ρ (of course, assuming that $\lambda > \lambda_c$). In this case we find that higher turnover τ always implies lower prevalence ρ when the peer-influence rate λ is low enough, but it implies higher ρ when λ is sufficiently high. This is indeed intuitive since higher turnover means not only faster link destruction (which tends to isolate B agents) but also faster link creation (which may enhance the spread of action B). Whether one effect or the other should dominate must depend on the value of λ. Complex patterns arise, however, when λ is at an intermediate level and neither of those effects dominates. Then we find that the effect is no longer monotone and it crucially depends on whether the underlying parameters lie in one of the aforementioned regions (so, in particular, it depends on γ). In Region I, the value of ρ attains an interior maximum as a function of τ, so there is a (finite) "worst level of turnover" that maximizes the prevalence of bad behavior and the best performance is achieved for high turnover. Instead, in Regions II and III, ρ achieves an interior minimum, so that the best level of turnover lies at some intermediate point.

Our analysis of the model relies on so-called mean-field techniques, widely used in statistical physics for the study of large interacting systems with a substantial (and little correlated) random component in the behavior of individual entities. In fact, since a key feature of the model revolves around the correlation of behavior between linked agents, our mean-field description of the network is not centered, as it is common in modern network analysis, on its degree distribution. Rather, we focus on the distribution of the different types of links (GG, BB, or GB), building on what is known as multisite (or cluster) mean-field analysis.[2] This approach, of course, still abstracts from correlations that, to some extent, must play some role in the dynamics of the system. The mean-field theory must be viewed, therefore, as an approximate description of the situation. This leads us to check its validity through extensive Monte Carlo simulations, and we find that both (theory and simulations) are in very close match. In particular, the theory provides a quite accurate prediction of how the critical points as well as the induced long-run prevalence of bad behavior depends on the different parameters of the model.

Our model provides an abstract theoretical framework for the study of how local diffusion of behavior (peer effects) and the underlying pattern of interaction (peer selection/avoidance) coevolve in many social environments. Admittedly, the setup is very stylized and thus can afford only a very simplified representation of how those processes unfold in real-world environments. It is, however, that simple formulation which allows us to gain a good analytical handle of the overall dynamics and obtain the insights that could be applied to more specific scenarios. There are, specifically, two natural realms of application, which themselves have generated a very substantial body of literature: the evolution of cooperative behavior in social systems, and the study of disease (or electronic) epidemics in large populations of people (or interconnected computers). In both contexts, the need to allow for the simultaneous evolution of behavior and the underlying social network appears of crucial importance to understand the eventual outcome. A thorough review of these two strands of literature is conducted in our working-paper version, Fosco, Marsili, and Vega-Redondo (2007) [10]. Here we only provide a brief sketch of each of them in turn.

[2] See ben-Avraham and Köhler (1992) [3] for a clear description of the mean-field cluster method.

The fact that the combination of local interaction and simple (but natural) rules of behavior can jointly contain the spread of opportunistic/noncooperative behavior in social systems was highlighted by the influential work of Nowak and May (1992) [16], Nowak, Bonhoeffer and May (1994) [15], and Eshel, Samuelson, and Shaked (1998) [11]. These papers studied the problem for a fixed structure of interaction, but others have followed suit by letting the interaction structure evolve endogenously over time. Interesting research in this vein has been conducted by Ebel and Bornholdt (2002) [9], Zimmermann, Eguíluz and San Miguel (2004) [21], and Hanaki, Peterhansl, Dodds and Watts (2007) [13]. Relying on numerical analysis alone, all of them identify corresponding conditions (each reflecting the specific features of their setup) under which some extent of cooperative behavior arises. An important difference between their approach and that of the present paper hinges upon the nature of the long run outcome. In their case, it represents an absorbing situation that is eventually reached within a stable underlying environment. Our model, in contrast, contemplates an ever changing environment, where the links and behavior of agents are subject to persistent volatility.

The study of epidemics, on the other hand, has a long and well-established tradition.[3] But only recently has it tried to account for the local-interaction effects embodied by a (possibly complex) social network, or consider contexts (such as internet) where the internode diffusion goes well beyond the spread of disease-like phenomena.[4] Most of this recent work, however, has presumed that the underlying network either remains fixed throughout or changes very slowly. This is now recognized as an important limitation of the analysis, and some interesting efforts have been devoted to allowing for a genuinely coevolving network. Out of this starting line of research,[5] we want to single the recent paper by Gross, Dommar D'Lima and Blasius (2006) [12] which is closest to our own concerns.[6] These authors also study how a SIS epidemics unfolds on a large network, which itself adapts over time as healthy/susceptible (i.e. G) nodes attempt to avoid/isolate infected (B) neighbors. The network adjustment dynamics, however, is quite different: healthy nodes *rewire* their links from infected neighbors to other healthy nodes. Hence, over time, only links between healthy and infected nodes decay and new contacts arise only between two healthy nodes.[7] The total average degree is preserved but healthy nodes tend to accumulate links (with other healthy ones) while infected nodes loose them (only keeping those with their own kind). Their model, therefore, differs substan-

[3] See e.g. Bailey (1975) [2] for a classic discussion of mathematical epidemiology.

[4] For a survey on how the complex-network literature has approached the study of epidenmics, see the references mentioned in Footnote 1. On the other hand, for a useful elaboration on how epidemic processes bear on the operation of the internet network, see Pastor-Satorras and Vespignani (2004) [18].

[5] See e.g. Ball, Mollison and Scalia-Tomba (1997) [1], Boots and Sasaki (1999) [4], and Saramäki and Kaski (2004) [19].

[6] This work is independent of ours and its first working-paper version appeared after the first version of our paper was completed.

[7] Nevertheless, Zanette (2007) [20] studies a variation where susceptible nodes rewire their links to *any* other node and obtains similar results.

tially from ours and so do their conclusions. Firstly, in their model, a sufficient large increase in the rewiring rate causes the infection to be completely suppressed, i.e. a zero level of prevalence. In contrast, in our setup, for a given rate of avoidance the final effect on prevalence depends on the peer rate – if it is too high, increasing social turnover does not decrease prevalence but has the opposite effect.[8] Secondly, they observe that, within a certain range of parameter values (i.e. between some lower and upper thresholds of the spreading rate – the counterpart of our peer-influence rate), the endemic *and* the healthy population state can be stable simultaneously. Thus, their system displays transitions across different levels of prevalence that can also be discontinuous and between those two thresholds a hysteresis loop is formed. Instead, in our model, both of those thresholds are the same and the transition is always continuous.[9]

The paper is organized as follows. In Section 2, we present the framework, the assumptions, the derivation of the mean-field model and its long run solutions. In Section 3, we analyze and discuss the results and present some simulations. Finally, in Section 4 we sum up and discuss possible avenues of future research. For the sake of clarity, we have relegated the main bulk of mathematical derivations and proofs to the Appendix.

2 The Model

There is a population of N individuals (implicitly supposed large) whose relationships and behavior continuously coevolve. Relationships, or social contacts, are represented by a network of undirected links. At each time $t \in [0, \infty)$, each agent i interacts with her immediate neighborhood, i.e. the set of individuals connected to her. Individual behavior is a binary variable with two states: G ("good") or B ("bad"), conceived generally as representing behavior that can be associated to cooperation, social-mindedness, law-abidance, etc. and their opposite. Each individual displays the same behavior with all her neighbors – hence, at each t, every agent may be simply characterized by her current behavior and her neighbors.

The dynamics driving the coevolutionary process of agents' behavior and the social network consists of the following two components.

- *Behavior dynamics: peer effects*

Bad behavior spreads by bilateral contact. Specifically, there is some given *peer-influence rate* $\lambda > 0$ such that a G-node turns to B at a base rate that is proportional

[8] Naturaly, if we fix the rate at which changes in the network take place (social turnover τ), bad behavior can always be suppressed by increasing the differential rate at which bad peers are avoided, γ.

[9] In the language of statistical physics, such discontinuous (continuous) transitions are called first (second) order phase transitions.

to λ and the number of B neighbors. In addition, it is useful to allow for a small supplementary rate ε, conceived as noise, at which this change occurs, independently of neighborhood conditions. In total, therefore, the rate at which a G node switches to B is given by $(\lambda k_{GB} + \varepsilon)$, where k_{GB} is its number of B-neighbors. Reciprocally, any B-agent is assumed to change her behavior to G at a rate ν, independently of her neighbors' behavior.

Overall, the process by which individuals change behavior may be understood as one of stimulus/response operating on "resilient G fundamentals." That is, we may view individuals as naturally inclined to G but responding to bad external stimuli by switching temporarily from G to B. In a sense, this formulation reflects a reinforcement model of individual behavior, similar in spirit to that commonly posited in modern learning literature.[10]

- *Network dynamics: peer avoidance*

First, as a base framework, we consider a subprocess of link creation and destruction that is independent of agents' behavior and *by itself* would generate a random Poisson network. Thus, suppose that each possible link not currently in place is created at a rate $\tau > 0$, while each existing link is destroyed at the rate $\tau\eta > 0$. If only this subprocess were operating, it is straightforward to see that the induced social network would be a random network with a Poisson degree distribution and average degree $2/\eta$, independently of τ. The parameter τ quantifies the speed of network adjustment relative to that of behavioral adjustment, and will be called the rate of *social turnover*.

But, of course, since we do want to relate network adjustment to agents' behavior, we superimpose on the former subprocess another one (for simplicity, operating only on link destruction) by which there is a supplementary pressure for the removal of any link that connects pairs of agents in which *at least one* of them displays B. Formally, we posit that any link whose corresponding behavior profile is BB,GB, or BG vanishes at a rate $\tau(\eta + \gamma)$, where γ is the differential rate capturing the strength of the peer-avoidance component of the model.

One possible interpretation of the whole subprocess of network adjustment is as follows. First, there is a purely random component in the creation and destruction of links, which is totally unrelated to behavior. In fact, *all new* links are taken to be formed in this fashion, possibly because partner's behavior is not observed until a relationship has been established (and thus such behavior cannot have any effect on the creation of the link). Random forces, on the other hand, are also involved in link destruction as a reflection of what could be called volatility (i.e. unmodelled reasons for which

[10] These models can be traced back to the models of mathematical sociology originated by Bush and Mostellar (1955) [6], later developed in the economic literature by authors such as Cross (1983) [8], Borgers and Sarin (1997) [5], or Camerer and Ho (1999) [7].

links become obsolete or unfeasible and then vanish). But, in addition to those random subprocesses, there is also an additional route for link destruction that is contingent on the behavior currently displayed and thus *observed* by partners. And, in this respect, the implicit assumption we make is a standard one, namely, that in order for a link to persist there must be mutual consent. Hence both parties must be satisfied, which is something that is supposed to occur only if both display G behavior.

Mean-field equations

In order to derive the system of dynamic equations that govern the expected co-evolution of structure (social network) and behavior (node's states), we introduce two binary variables: the behavioral variable s_i that assumes the value 1 (0) if the node i is B (G); and the network variable $a_{i,j}$ that adopts the value of 1 (0) if the link between i and j is present (missing). The dynamic equations for the *expected* values of these variables are:

$$\dot{s}_i = (\varepsilon + \lambda \sum_j a_{i,j} s_j)\,(1 - s_i) - \nu s_i \tag{1}$$

$$\dot{a}_{i,j} = \frac{2\tau}{N}(1 - a_{i,j}) - \tau\,[\eta + \gamma\,(s_i + s_j - s_i s_j)]\,a_{i,j} \tag{2}$$

At any t, $\sum_i s_i$ is the number of B nodes, and $[GB] = \sum_{i,j}(1 - s_i)\,a_{i,j} s_j$, $[BB] = \frac{1}{2}\sum_{i,j} s_i a_{i,j} s_j$ and $[GG] = \frac{1}{2}\sum_{i,j}(1-s_i)a_{i,j}(1-s_j)$, the number of pairs (links) GB/BG, BB and GG, respectively. For N large, the ratios $\frac{\sum_i s_i}{N}$, $\frac{[GB]}{N}$, $\frac{[BB]}{N}$ and $\frac{[GG]}{N}$ approach the deterministic limits ρ (which we call prevalence), $[gb]$, $[bb]$ and $[gg]$, respectively. From equations (1, 2), standard methods lead to a dynamic model based on multi-site (or cluster) mean-field theory. In its general formulation, this approach yields an *infinite* hierarchy of equations for multi-site correlation functions. However, in the case of a large population connected by a sparse random network, this hierarchy can be closed in terms of the density of triplets obtained from local densities and pair densities. And in the end, one arrives at a dynamical system defined on the average conditional connectivities $\langle k_{GB}\rangle$, $\langle k_{GG}\rangle$, $\langle k_{BB}\rangle$ as follows:[11]

$$\dot{\rho} = -\nu\rho + (\varepsilon + \lambda\,\langle k_{GB}\rangle)\,(1 - \rho) \tag{3}$$

$$\begin{aligned} \dot{\langle k_{GB}\rangle} = 2\tau\rho - (\nu + \varepsilon + \tau\,(\eta + \gamma))\,\langle k_{GB}\rangle + \varepsilon\,\langle k_{GG}\rangle + \nu\,\langle k_{BB}\rangle\,\tfrac{\rho}{1-\rho} \\ +\lambda\,\langle k_{GB}\rangle\,(\langle k_{GG}\rangle - \langle k_{GB}\rangle - 1) + \tfrac{\dot{\rho}}{1-\rho}\,\langle k_{GB}\rangle \end{aligned} \tag{4}$$

[11] See the Appendix for the analytical derivation of the following equations.

$$\dot{\langle k_{BB} \rangle} = 2\tau\rho - (2\nu + \tau(\eta + \gamma)) \langle k_{BB} \rangle + 2\varepsilon \langle k_{GB} \rangle \frac{1-\rho}{\rho} + 2\lambda \frac{1-\rho}{\rho} \langle k_{GB} \rangle (\langle k_{GB} \rangle + 1) - \frac{\dot{\rho}}{\rho} \langle k_{BB} \rangle \quad (5)$$

$$\dot{\langle k_{GG} \rangle} = 2\tau(1-\rho) - (2\varepsilon + \tau\eta) \langle k_{GG} \rangle + 2\nu \langle k_{GB} \rangle - 2\lambda \langle k_{GB} \rangle \langle k_{GG} \rangle + \frac{\dot{\rho}}{1-\rho} \langle k_{GG} \rangle \quad (6)$$

The interpretation of these equations is straightforward. Equation (3), for example, reflects that the density of B agents decreases whenever B-agents spontaneously return to good behavior, while it increases every time G agents switch to bad behavior due to the influence of peers (in addition to the base transition occurring at the small rate ε). In turn, the average conditional connectivity changes when links *of the type considered* (GB, BB, or GG) are created and decay and when the appropriate agents maintaining other kind of links *change their behavior*. For instance, in (4), $\langle k_{GB} \rangle$ increases when a new encounter between a B and a G occurs, when one of the agents connected by a GG link is "spoiled" by peer effects (or by exogenous reasons, at the small rate ε), or when one of two agents connected by a BB link changes to G. It decreases, on the other hand, when links GB vanish, when a B partner changes exogenously to G at the rate ν, or when the G agent switches to B due to peer effects.[12]

Parameter configurations

The model has the following parameters: ν, λ, and ε that shape the behavioral dynamics; η and γ that bear on the relative speeds of link destruction and creation; and finally, τ that models the relative pace of behavioral and network adjustment. We reduce the range of parameters under considerations as follows.

(a) First, we set $\nu = 1$.

(b) Then, we set the range of possible values for η and γ such that the *long run* total average connectivity is relatively high if all nodes were G-agents ($\rho \to 0$), while the opposite happens if all agents were of the B type ($\rho \to 1$). Specifically, we assume that η and γ are such that the connectivity in each limit case lie in separate sides on the key threshold of unity, i.e.[13]

$$\langle k|\rho \to 0\rangle > 1 \text{ and } \langle k|\rho \to 1\rangle < 1 \quad (7)$$

[12] The last terms in eqs. (4-6) embody the effect on average conditional connectivities induced by the fact that changes in ρ alter the probabilities of the conditioning events.

[13] Bear in mind that the condition $\langle k \rangle = 1$ is the threshold above which a random network has a giant component, i.e. a non-negligible fraction of connected nodes when $N \to \infty$.

The role of (a) is simply that of a normalization, which fixes the time scale of the process. The motivation of (b), on the other hand, is twofold. On one hand, we want to consider a scenario where extremely polarized behavior would have significant effects on the overall connectivity of the social network. For only in this case may the social network be expected to have a significant effect on the overall spreading of bad behavior. We also find, on the other hand, that the restriction of η and γ to satisfy condition (b) greatly sharpens the focus of the analysis on the two remaining parameters, i.e. the rate of social turnover τ and the strength of peer effects λ.

To understand, more precisely, what conditions on the parameters η and γ are entailed by (7), note that both when $\rho \to 0$ and when $\rho \to 1$, we must have $\langle k_{GB} \rangle \to 0$. Therefore, from (5) and (6) (and $\dot{\rho} = \dot{\langle k_{BB} \rangle} = \dot{\langle k_{GG} \rangle} = 0$) we can write

$$\begin{array}{l} \langle k|\rho \to 0\rangle > 1 \Leftrightarrow \langle k_{GG}|\rho \to 0\rangle = \frac{2}{\eta} > 1 \\ \langle k|\rho \to 1\rangle < 1 \Leftrightarrow \langle k_{BB}|\rho \to 1\rangle = \frac{1+\tau}{1+\frac{\tau(\eta+\gamma)}{2}} < 1 \iff \eta + \gamma > 2 \end{array}$$

so that the implied parameter range is given by the conditions

$$\eta \in (0, 2)\,; \qquad \gamma \in (2 - \eta, \infty)\,. \tag{8}$$

Long-run behavior

We are interested in characterizing stable stationary states of the dynamics given by (3)-(6). In a stationary state, $\dot{\rho} = \dot{\langle k_{GB} \rangle} = \dot{\langle k_{BB} \rangle} = \dot{\langle k_{GG} \rangle} = 0$. Thus, assuming a negligible $\varepsilon \approx 0$, we obtain the following non-linear system in the variables ρ, $\langle k_{GB} \rangle$, $\langle k_{GG} \rangle$, $\langle k_{BB} \rangle$:

$$\rho = \frac{\lambda \langle k_{GB} \rangle}{1 + \lambda \langle k_{GB} \rangle} \tag{9}$$

$$\langle k_{GB} \rangle = \frac{2\tau\rho + \lambda \langle k_{GB} \rangle (\langle k_{GG} \rangle + \langle k_{BB} \rangle)}{1 + \tau(\eta + \gamma) + \lambda(\langle k_{GB} \rangle + 1)} \tag{10}$$

$$\langle k_{BB} \rangle = \frac{\tau\rho + \langle k_{GB} \rangle + 1}{1 + \frac{\tau(\eta+\gamma)}{2}} \tag{11}$$

$$\langle k_{GG} \rangle = \frac{\tau(1-\rho) + \langle k_{GB} \rangle}{\lambda \langle k_{GB} \rangle + \frac{\tau\eta}{2}} \tag{12}$$

To characterize the solutions of the system, it is useful to define the following function of the parameters:

$$\Phi_c(\eta, \gamma, \lambda, \tau) := \frac{\lambda}{\eta} \frac{(2 + 2\tau\eta - \eta)(\tau(\eta + \gamma) + 2) + 2\eta}{(\tau(\eta + \gamma) + 1)(\tau(\eta + \gamma) + 2)} \tag{13}$$

Then we can first establish the following result.

Proposition 1
(i) Let the parameters satisfy $\Phi_c(\eta, \gamma, \lambda, \tau) < 1$*. Then, the mean-field dynamics has a unique stationary state and it has* $\rho^* = 0$*.*
(ii) Let the parameters satisfy $\Phi_c(\eta, \gamma, \lambda, \tau) > 1$*. Then, the mean-field dynamics has two stationary states. One of them has* $\rho^* = 0$*, while the other has* $\rho^* > 0$*.*

Proof. See the Appendix. ■

The previous result indicates that a stationary state with only G behavior always exists. It is unique if $\Phi_c(\eta, \gamma, \lambda, \tau) < 1$, while another one with positive prevalence also exists if $\Phi_c(\eta, \gamma, \lambda, \tau) > 1$. In view of this possible multiplicity, it is important to evaluate the stability of the different configurations. This issue is addressed by the following result.

Proposition 2
(i) Let the parameters satisfy $\Phi_c(\eta, \gamma, \lambda, \tau) < 1$*. Then, the unique stationary state (that has* $\rho^* = 0$*) is asymptotically stable.*
(ii) Let the parameters satisfy $\Phi_c(\eta, \gamma, \lambda, \tau) > 1$*. Then, only the stationary state with* $\rho^* > 0$ *is asymptotically stable.*

Proof. See the Appendix. ■

The previous result indicates that the model exhibits a continuous transition at the threshold $\Phi_c(\eta, \gamma, \lambda, \tau) = 1$ that separates a phase of overall G behavior from a phase in which a positive fraction of B agents is expected to prevail in the long run. As we shall amply illustrate in the subsequent section, these theoretical conclusions represent a very good prediction, not only qualitatively but also quantitatively, of the results that are obtained from extensive numerical simulations.[14]

Remark 1 Note that $\lim_{\gamma \to \infty} \Phi_c(\eta, \gamma, \lambda, \tau) = 0$. This means that given the peer-influence rate $\lambda > 0$, the social turnover $\tau > 0$ and the basic volatility of relationships, $\eta > 0$, it is always possible to suppress the prevalence of bad behavior by sufficiently increasing the rate at which bad agents are avoided. If, in the limit, agents are allowed to cut BG/GB and BB links immediately (i.e. $\gamma \to \infty$) bad behavior will never invade the population.

[14] It should be stressed, however, that even if a steady state with $\rho^* = 0$ is not asymptotically stable for the mean-field dynamics, such a configuration is always absorbing in systems for $\varepsilon = 0$. That is, eventually and with probability one, all nodes will become G agents, and misbehavior can no longer spread thereafter whatever is the spreading rate. In this light, one can understand the role of a positive (albeit small) ε as a way to expose the fragility of such situations in actual implementations of the system through (finite) simulations.

3 Peer influence, social turnover and prevalence

Our main concern here is to study how the different forces at work affect the long-run prevalence of bad behavior, ρ. As explained above, the parameters ν, η, and γ are either fixed by normalization ($\nu = 1$) or we choose to have their range restricted in a convenient way – cf. (8). Thus, the focus of our analysis will be on the effect on prevalence ρ of the interplay between the strength of peer influence parametrized by λ and the speed of the network turnover as captured by τ. We start by studying the effect of λ for a given social turnover τ, thus in effect "slicing" the function $\rho(\lambda, \tau)$ in the τ dimension.

Peer influence and prevalence

The effect of the peer-influence rate λ on ρ is *qualitatively* the same for *any* given social turnover τ *and link decay rates* (η, γ). For any given social turnover τ, the threshold $\Phi_c(\eta, \gamma, \lambda, \tau) \gtreqless 1 \iff \lambda \gtreqless \lambda_c$, where:

$$\lambda_c = \frac{\eta\left(\tau\left(\eta+\gamma\right)+1\right)\left(\tau\left(\eta+\gamma\right)+2\right)}{\left(2+2\tau\eta-\eta\right)\left(\tau\left(\eta+\gamma\right)+2\right)+2\eta}. \tag{14}$$

Thus, if $\lambda \leq \lambda_c$, $\rho(\lambda) = 0$, while if $\lambda > \lambda_c$, $\rho(\lambda) > 0$ and $\rho(\cdot)$ is increasing. This outcome is standard in the analysis of SIS models. The critical threshold λ_c – a point uniquely determined once τ, γ and η are fixed – separates the phases of null and positive (but only partial) materialization of B behavior. To obtain closed analytical expressions for the evolution of ρ, we consider extreme values of τ, where it is easy to find:

$$\rho\left(\lambda|\tau=0\right) = \begin{cases} \frac{1}{2} + \frac{2\lambda+\gamma-\sqrt{4(\gamma+\lambda(\eta+\gamma))(\eta+\gamma)+\lambda^2(\eta+\gamma-2)^2}}{2(\gamma+\lambda(\eta+\gamma))} > 0 & \Leftrightarrow \lambda > \frac{\eta}{2} \\ 0 & \Leftrightarrow \lambda \leq \frac{\eta}{2} \end{cases} \tag{15}$$

$$\rho\left(\lambda|\tau\to\infty\right) = \begin{cases} 1 - \frac{\eta+\gamma}{2\lambda} > 0 & \Leftrightarrow \lambda > \frac{\eta+\gamma}{2} \\ 0 & \Leftrightarrow \lambda \leq \frac{\eta+\gamma}{2} \end{cases} \tag{16}$$

When $\tau \to 0$, the network doesn't evolve and the model is reduced to the propagation of misbehavior on a fixed pattern of connections with average connectivity $\approx \frac{2}{\eta}$. The critical threshold is akin to that found in epidemic models when it is assumed that the connectivity is approximately homogeneous (i.e. $k_i \approx \langle k \rangle$) and the homogeneous mixing hypothesis holds (i.e. $\lambda_c = 1/(\frac{2}{\eta})$). On the other extreme, when $\tau \to \infty$, all the relationships are extremely volatile and the social structure has no real bearing on behavior. Thus the model approaches one where *every* individual choosing G faces an essentially symmetric situation, typically confronting the same number $\frac{2}{\eta+\gamma}$ of B agents. (This follows from the fact that every GB link is formed at the rate of 2 while it vanishes at the rate $\eta + \gamma$.) Under these conditions, B behavior can spread if $\lambda\frac{2}{\eta+\gamma} > 1$, where recall that $\nu = 1$ is the spontaneous recovery rate. This leads to a critical threshold for positive prevalence equal to $\lambda_c = 1/(\frac{2}{\eta+\gamma})$.

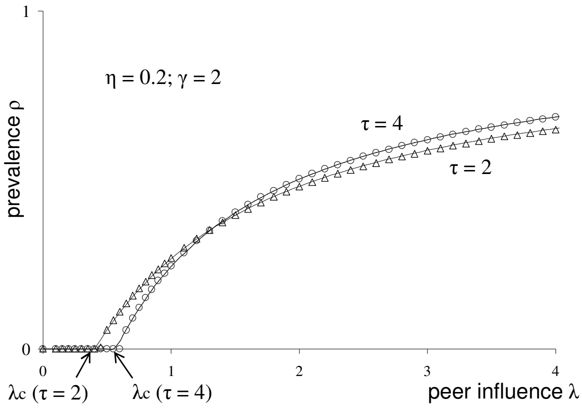

Figure1: Prevalence ρ as a function of strength peer influence λ. Lines represent the mean-field predictions; circles ($\tau = 4$) and triangles ($\tau = 2$), MC simulations ($N = 10^4$, $\rho(t = 0) = 1\%$, $\varepsilon = 10^{-5}$).

For finite social turnover τ, the situation can be quite complex, as the examples displayed in Figures 1 and 2 indicate. In these examples, the lines trace our theoretical prediction, while circles/triangles represent observations derived Monte Carlo (MC) simulations.[15]

These examples suggest the possibility of a nonmonotonic effect of turnover on prevalence, its specific direction depending on the *peer-influence rate* λ under consideration (see Figure 1). Such contrasting effects also seem to arise concerning the effect of turnover on the critical threshold λ_c for peer influence, the direction in this case depending on the particular value of the remaining two parameters, η and γ (cf. Figures 1 and 2). The aim of the subsequent subsections is to explore systematically such pattern of cross dependencies.

[15] All the simulations presented in this paper were performed with initial random networks of $N = 10^4$; initial number of B-agents 1%; and $\varepsilon = 10^{-5}$. Long run outcomes are (almost) invariant to moderate changes in the initial number of bad agents and noise. It is also highly robust to N, down to a minimum of about $N = 2000$ nodes (with initial bad agents and ε accordingly adjusted). Increases in N above this lower bound do not have any significant bearing on the results.

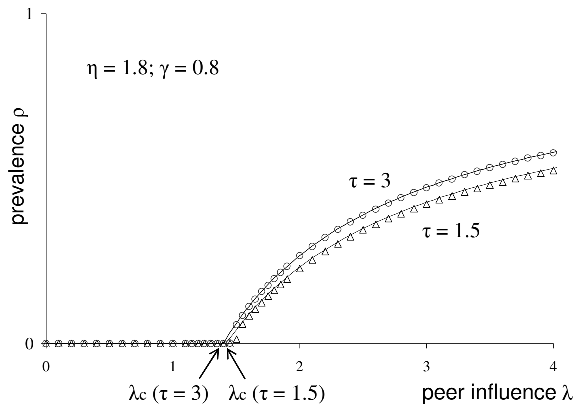

Figure 2: Prevalence ρ as a function of strength peer influence λ. Lines represent the mean-field predictions; circles ($\tau = 3$) and triangles ($\tau = 1.5$), MC simulations ($N = 10^4$, $\rho(t = 0) = 1\%$, $\varepsilon = 10^{-5}$).

Social turnover, critical threshold and prevalence

As we explained in Section 2, the onset of positive prevalence is marked by the locus of points in the parameter space that satisfy $\Phi_c(\eta, \gamma, \lambda, \tau) = 1$. For given η, γ and λ, it is easy to see that the set $\{\tau \in \mathbb{R}_{++} : \Phi_c(\eta, \gamma, \lambda, \tau) = 1\}$ may be empty, a singleton, or consist of two points. The latter case would correspond to a situation where one could speak of two "critical" values for social turnover. This means, in particular, that for some given values of η and γ, the mapping $\tau_c(\lambda)$ defined by $\Phi_c(\eta, \gamma, \lambda, \tau_c(\lambda; \eta, \gamma)) = 1$ is not a (single-valued) function of τ. Instead, if we fix η, γ and focus on the critical value $\lambda_c(\tau; \eta, \gamma)$ defined by $\Phi_c(\eta, \gamma, \lambda_c(\tau; \eta, \gamma), \tau) = 1$ we find that it always specifies a well-defined and continuous function, a feature that significantly facilitates the analysis. We pursue, therefore, this latter approach in what follows.

Critical threshold λ_c

Denote the critical-threshold function by $\lambda_c(\tau)$ to highlight its dependence on τ. For any η,γ satisfying (8), this function is strictly positively valued in its domain, $\mathbb{R}_{++}$, with $\lim_{\tau\to 0} \lambda_c(\tau) = \frac{\eta}{2} < 1$ and $\lim_{\tau\to\infty} \lambda_c(\tau) = \frac{\eta+\gamma}{2} > 1$, which follow directly from (15) and (16), respectively. It displays two different behaviors, which allows us to divide the parameter space $\{(\eta, \gamma) : \eta \in (0, 2) ; \gamma \in (2 - \eta, \infty)\}$ into two regions (see Figures 3 and 4 for typical examples). A precise description of the situation is contained in the following result.

Proposition 3 *Let $\lambda_c(\tau)$ denote the function that specifies the critical threshold for peer influence that marks the materialization of positive prevalence.*
(a) If $\gamma \geq \frac{\eta^2}{2-\eta}$ (and $\gamma > 2-\eta$ from (8)), the function $\lambda_c(\tau)$ is increasing and concave in the whole range of $\tau \geq 0$, its asymptote being $\lambda_c(\tau \to \infty) = \frac{\eta+\gamma}{2}$.
(b) If $\gamma < \frac{\eta^2}{2-\eta}$ (and $\gamma > 2-\eta$ from (8)), the function $\lambda_c(\tau)$ has a unique interior maximum for some positive (and finite) τ^* with $\lambda_c(\tau^*) > \lambda_c(\tau \to \infty) = \frac{\eta+\gamma}{2}$.

Proof. See the Appendix. ■

For future use, it will be convenient to refer to the parameter conditions specified in (a) above as the union of Regions I and II (or *Region I+II* for short), while we shall refer to the parameter conditions specified in (b) by *Region III.* The situation is quite different in each case. In Region I+II, higher turnover can just help in attaining the full dominance of G behavior. Instead, in Region III, there is a range of values for λ, $\frac{\eta+\gamma}{2} < \lambda < \lambda_c(\tau^*)$, where higher rates of turnover can lead to fresh coexistence of G and B behavior.

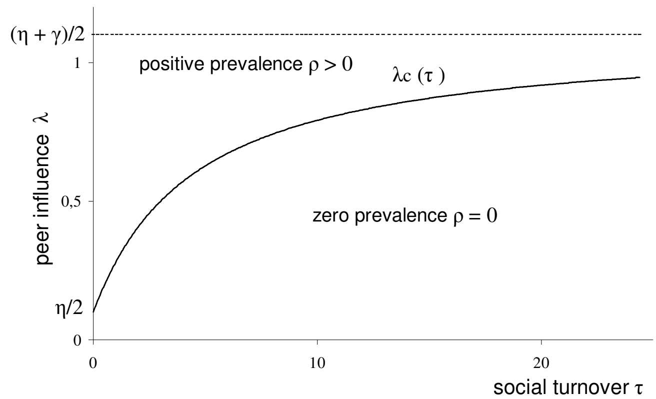

Figure 3: Critical threshold λ_c as a function of social turnover τ.
Example Region I+II ($\gamma > \eta^2/(2-\eta)$): $\eta = 0.2; \gamma = 2$.
$\lambda_c(\tau)$ increasing up to asymptote $(\eta + \gamma)/2$.

The contrasting implications of the two parameter regions are an intuitive reflection of the twin role that network turnover has in our context. On the one hand, turnover acts as a disciplining mechanism that tends to isolate B agents and thus checks the spread of bad behavior. But, on the other hand, faster turnover can also have a detrimental effect on the preservation of good behavior. This happens because, as the links are adjusted at a brisker pace, the wider become as well the possibilities afforded to B agents in spreading their behavior though peer influence. In this light, Proposition 3 simply establish the intuitive fact that whether the first consideration will come to dominate throughout or not depends on the relative magnitudes of the base and differential rates of link destruction, respectively given by η and γ. In particular, if γ is not large enough relative to η (Region III), we find that, beyond a certain point (the rate τ^*), higher turnover has the "counterproductive" effect of lowering the critical rate λ_c.

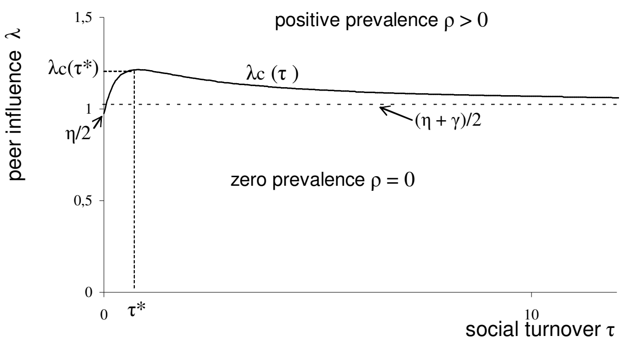

Figure 4: Critical threshold λ_c as a function of social turnover τ.
Example for Region III ($\gamma < \eta^2/(2-\eta)$): $\eta = 1.95; \gamma = 0.1$.
$\lambda_c(\tau)$ non-monotonic; $\tau^* = 0.795\,52$; $\lambda_c(\tau^*) = 1.2138$.

Having settled by Proposition 3 what is the effect of turnover on the threshold λ_c that marks the consolidation of some extent of bad behavior in the long run, a natural follow-up question arises. Given the prevailing turnover rate τ, suppose that $\lambda > \lambda_c(\tau)$ so that the induced extent of long run prevalence $\rho(\tau) > 0$. How is this prevalence of bad behavior affected by changes in τ? This is the question addressed in the next subsection.

Prevalence ρ

Given η and γ, and the prevailing rate of turnover τ, assume that $\lambda > \frac{\eta}{2} = \lambda_c(\tau \to 0)$ so that the induced prevalence $\rho(\tau)$ is positive for (at least) some $\tau \in \mathbb{R}_{++}$.[16] First of all, note that for λ given, the limiting behavior of $\rho(\tau)$ as τ grows or falls to its extreme values, 0 and ∞, is respectively given by (15) and (16). Now denote by $\hat{\lambda}$ the value of λ such that if $\lambda > \hat{\lambda}$ ($< \hat{\lambda}$) the slope of $\rho(\tau)$ at $\tau = 0$ is strictly positive (negative). We can then first characterize the evolution of ρ as a function of τ for two polar cases: when λ is relatively low and when it is relatively high, given the value of η, γ, and $\hat{\lambda}$ (which is itself, of course, a function of η and γ). In those cases, the effect of τ on

[16] Note that for any η and γ satisfying 8, the minimum value of $\lambda_c(\tau)$ is $\frac{\eta}{2} = \lim_{\tau \to 0} \lambda_c(\tau)$, thus if $\lambda \leq \frac{\eta}{2}$, there is null prevalence ($\rho = 0$) for any social turnover τ.

ρ is always monotone (decreasing and increasing, respectively), as established by the following result.

Proposition 4 *Let $\rho(\tau)$ denote the function that specifies the prevalence of bad behavior as a function of τ, for given values of λ, η and γ.*
(a) If $\lambda \in (\frac{\eta}{2}, \min\{\hat{\lambda}, \max\{\frac{\gamma}{\eta}, \frac{\eta+\gamma}{2}\}\}]$, the function $\rho(\cdot)$ is monotonically non-increasing. Moreover, if $\lambda < \frac{\eta+\gamma}{2}$, $\rho(\cdot)$ decreases monotonically until it vanishes at a finite critical value τ_c.
(b) If $\lambda > \max\{\hat{\lambda}, \frac{\gamma}{\eta}\}$, the function $\rho(\cdot)$ is strictly positive and increasing for all τ.

Proof. See the Appendix. ■

Again, the former conclusions are intuitive, and can be understood along the same lines as before. When peer influence is weak, faster social turnover is a crucial mechanism for checking bad behavior. Indeed, as its pace accelerates, a definite point is eventually reached when no B behavior persists in the long run, i.e. a state with zero prevalence is attained. Instead, when peer influence is strong, the mechanism of social turnover works in a "perverse" manner: it rides on forceful peer effects to spread bad behavior ever faster and more widely. An illustration of this state of affairs is provided in panels (a) and (d) of Figures 5,6.

The situation, as one would expect, is much less transparent when λ displays an intermediate value. In this case, the interplay between positive and negative effects operate at different strengths for different values of τ, thus yielding nonmonotonic behavior. A formal description of matters is the object of the following result.

Proposition 5 *Let $\rho(\tau)$ denote the function that specifies the prevalence of bad behavior as a function of τ, for given values of η, γ, and $\lambda \in (\min\{\hat{\lambda}, \max\{\frac{\gamma}{\eta}, \frac{\eta+\gamma}{2}\}\}, \max\{\hat{\lambda}, \frac{\gamma}{\eta}\}]$.*
(a) If $\gamma > \frac{2\eta^2}{2-\eta}$, the function $\rho(\cdot)$ has a unique interior maximum.
(b) If $\gamma < \frac{2\eta^2}{2-\eta}$, the function $\rho(\cdot)$ has a connected set $[\tau_1, \tau_2]$ of interior minima. If $\tau_1 \neq \tau_2$, then $\rho(\tau) = 0$ for all $\tau \in [\tau_1, \tau_2]$.

Proof. See the Appendix. ■

The implications of the above result are illustrated by panels (b)-(c) of Figures 5, 6. In line with the region labelling used to present the results of Proposition 3, let us denote by *Region I* the parameter configurations induced by (a) in Proposition 5. It is the clear that Region I is a subset of what we called before "Region I+II" and, by comparison, we want to identify *Region II* with the inequality $\frac{\eta^2}{2-\eta} < \gamma < \frac{2\eta^2}{2-\eta}$.

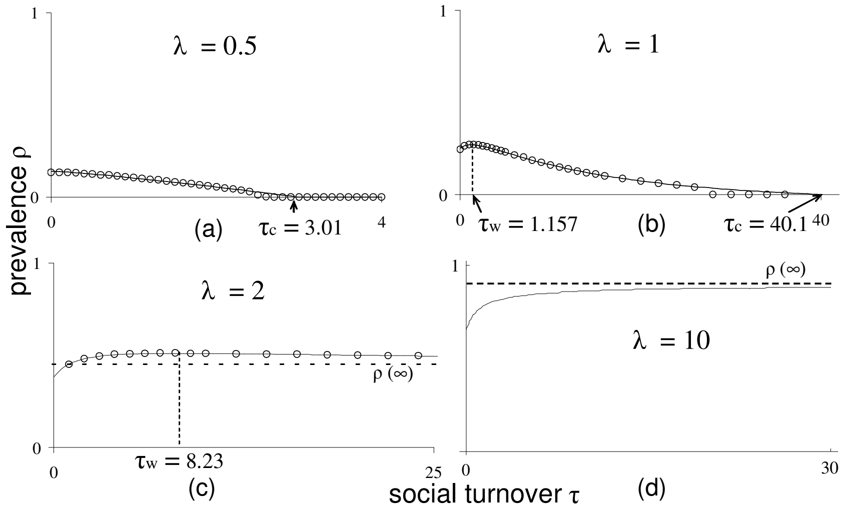

Figure 5: Prevalence ρ as a function of social turnover τ. Examples for Region I ($\gamma \geq 2\eta^2/(2-\eta)$): $\eta = 0.2; \gamma = 2$. Lines represent the mean-field predictions, circles, MC simulations ($N = 10^4$, $\rho(t=0) = 1\%$, $\varepsilon = 10^{-5}$).

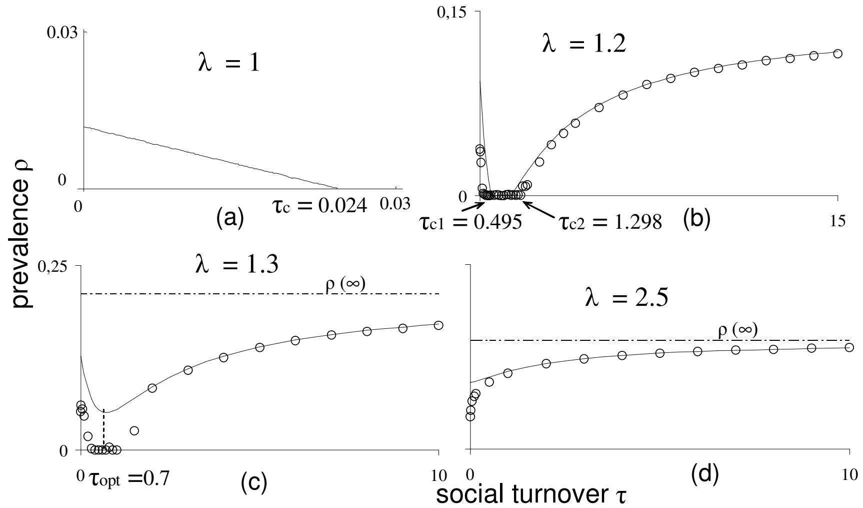

Figure 6: Prevalence ρ as a function of social turnover τ. Examples for Region III ($\gamma < 2\eta^2/(2-\eta)$): $\eta = 1.95; \gamma = 0.1$. Lines represent the mean-field predictions, circles, MC simulations ($N = 10^4$, $\rho(t=0) = 1\%$, $\varepsilon = 10^{-5}$).

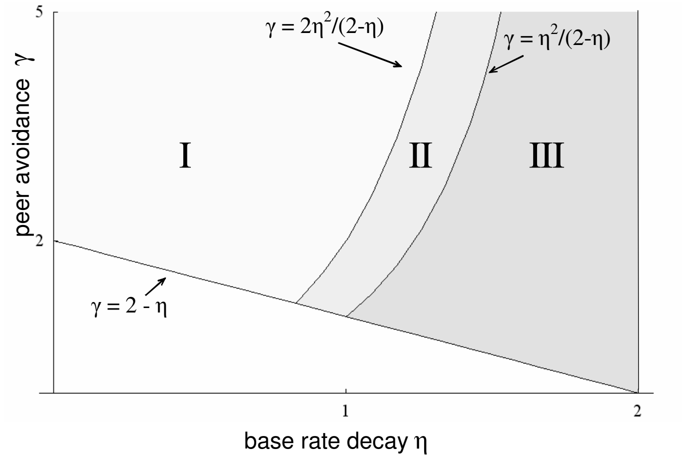

Figure 7: Regions in (η, γ)

<table>
<tr><td>γ,η:</td><td>$\gamma>\frac{2\eta^2}{2-\eta}$
$\eta \in(0,2)$;
(R. I)</td><td>$\frac{2\eta^2}{2-\eta}>\gamma>\frac{\eta^2}{2-\eta}$
$\gamma \in(2\text{-}\eta,\infty)$
(R. II)</td><td>$\gamma<\frac{\eta^2}{2-\eta}$

(R. III)</td></tr>
<tr><td rowspan="2">$\lambda_c(\tau)$:</td><td colspan="3">$\lim\limits_{\tau\to 0}\lambda_c=\frac{\eta}{2}$, $\lim\limits_{\tau\to\infty}\lambda_c=\frac{\eta+\gamma}{2}$</td></tr>
<tr><td colspan="2">$\frac{\partial\lambda_c(\tau)}{\partial\tau}>0$; $\forall\tau$</td><td>$\exists\tau^*<\infty$:
$\tau^*=$
argmax$_\tau\lambda_c$</td></tr>
<tr><td>λ:</td><td colspan="3">ρ (τ)</td></tr>
<tr><td>$[0,\frac{\eta}{2}]$</td><td colspan="3">ρ=0; $\forall\tau$</td></tr>
<tr><td>$(\frac{\eta}{2}$,
min$\{\hat{\lambda}$,max$\{\frac{\gamma}{\eta},\frac{\eta+\gamma}{2}\}\}]$</td><td colspan="3">$\frac{\partial\rho}{\partial\tau}<0$,$\forall\tau$; $\rho\to$ max$\{0$,1-$\frac{\eta+\gamma}{2\lambda}\}$</td></tr>
<tr><td rowspan="2">(min$\{\hat{\lambda}$,max$\{\frac{\gamma}{\eta},\frac{\eta+\gamma}{2}\}\}$,
max$\{\hat{\lambda},\frac{\gamma}{\eta}\}]$</td><td rowspan="2">$\exists\tau_w$:
τ_w=
argmax$_\tau\rho$</td><td colspan="2">$\exists\tau_{opt}$: τ_{opt}= argmin$_\tau\rho$</td></tr>
<tr><td></td><td>$\exists[\tau_1,\tau_2]$:
$\rho=0$</td></tr>
<tr><td>$>$ max$\{\hat{\lambda},\frac{\gamma}{\eta}\}$</td><td colspan="3">$\frac{\partial\rho}{\partial\tau}>0,\forall\tau$; $\rho\to 1-\frac{\eta+\gamma}{2\lambda}>0$</td></tr>
</table>

Table 1: Peer effects λ, peer avoidance γ and social turnover τ Synthesis of main effects on prevalence ρ.

With the aforementioned labelling conventions (see Figure 7), the conclusions of the Proposition 5 can be summarized as follows. Provided λ lies in the intermediate range being considered, prevalence in Region I is first increasing and then decreasing as turnover becomes faster, while in regions II+III, it is first decreasing and then increasing. This again is a reflection of the rich interplay between conflicting forces that arises when the peer effects are neither very strong nor very weak and the turnover rate operates at intermediate levels as well. In contrast, when turnover is high, we find once more (cf. Proposition 3) that the key consideration is whether peer avoidance is strong enough – or, more precisely, whether there is an enough significant wedge between the base and differential rates of link destruction.

4 Summary and Conclusions

The paper has proposed a simple model to study the interplay between peer effects and peer avoidance in the diffusion and prevalence of "bad" behavior in large social networks. Our analysis has focused on understanding how different speeds of network adjustment (i.e. what we have called social turnover) affect the long-run performance of the model. In particular, we have studied how it impinges on (a) the critical peer-influence rate required for positive prevalence of bad behavior, and (b) the precise magnitude of such prevalence. We have found that for a wide range of parameters, both magnitudes behave as intuitively expected– i.e. in a monotone fashion. For example, when the peer-influence rate is relatively low (respectively, high), higher turnover induces lower (respectively, higher) prevalence throughout. But for intermediate values of the peer-influence rate, conflicting tendencies operate and the picture is much more complex. In particular, prevalence can display either an interior local maximum or minimum, depending on the precise parameter configuration. These results point to the subtle effects at work when peer effects and peer avoidance interact in dynamic processes of behavioral diffusion. In a nutshell, the main insight we gain from the analysis is that such interaction is sharply affected by the speed at which the network adjusts, which crucially affects the final outcome.

The paper has focused on only one dimension of the problem, namely, the long run prevalence of bad behavior. Future research along these lines should address as well other important features of the system such as the entailed characteristics of the social network, e.g. its connectivity, degree distribution, clustering, etc. It would also be interesting to study certain variations of the present framework that might required to capture how different processes of diffusion operate in some social contexts. For example, one could introduce so-called frequency-dependent considerations in the formulation of peer effects. Specifically, it could be assumed that the switch to bad behavior by a particular agent depends on the *proportion* of her neighbors who choose B rather than their absolute number. In some setups, where behavior is shaped by strategic considerations, this formulation might indeed be more appropriate. Finally, another interesting variation would be to have B and G behavior play a symmetric role in the model, with the switch in either direction being dependent on the behavior being displayed by neighbors.

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A Appendix

System of dynamic equations

The evolution of the *average density* of B-agents for N large is approximated by:

$$\frac{\sum_i \dot{s}_i}{N} = \varepsilon \frac{\sum_i (1-s_i)}{N} + \lambda \frac{\sum_{i,j} (1-s_i) a_{i,j} s_j}{N} - \nu \frac{\sum_i s_i}{N} \rightarrow$$
$$\dot{\rho} = \varepsilon (1-\rho) + \lambda [gb] - \nu \rho$$

Replace by $\langle k_{GB} \rangle = \frac{[gb]}{(1-\rho)}$ and obtain (3). The average density of links GB/BG evolves as:

$$\begin{array}{c}\frac{[\dot{GB}]}{N} = 2\tau \frac{\sum_{i,j}(1-s_i)s_j}{N^2} - (\frac{2\tau}{N} + \tau(\eta+\gamma) + \nu + \varepsilon + \lambda)\frac{\sum_{i,j}(1-s_i)a_{i,j}s_j}{N} \\ +\varepsilon\frac{\sum_{i,j}(1-s_i)a_{i,j}(1-s_j)}{N} + \nu\frac{\sum_{i,j}s_i a_{i,j}s_j}{N} \\ +\lambda(\frac{\sum_{i,j,k}(1-s_i)a_{i,j}(1-s_j)a_{j,k}s_k}{N} - \frac{\sum_{i,j,k,i\neq k}s_i a_{i,j}(1-s_j)a_{j,k}s_k}{N})\end{array}$$

For $N \to \infty$ and always growing faster than τ ($\Longrightarrow \frac{\tau}{N}, \frac{2\tau}{N} \longrightarrow 0$):

$$\begin{array}{c}\frac{[\dot{GB}]}{N} \to [\dot{gb}] = 2\tau\rho(1-\rho) - (v + \varepsilon + \tau(\eta+\gamma) + \lambda)[gb] \\ +\varepsilon 2[gg] + \nu 2[bb] + \lambda(2[ggb] - [bgb])\end{array}$$

The last term is the difference between the expected number of triplets GGB and triplets BGB ($\frac{[GGB]}{N} \to [ggb]$ and $\frac{[BGB]}{N} \to [bgb]$).[17] Similarly, for N large the evolution of the average densities of links BB and GG approach:

$$\begin{aligned}[\dot{bb}] &= \tau\rho^2 - \left(v + \frac{\tau(\eta+\gamma)}{2}\right)2[bb] + (\varepsilon+\lambda)[gb] + \lambda[bgb] \\ [\dot{gg}] &= \tau(1-\rho)^2 - \left(\varepsilon + \frac{\tau\eta}{2}\right)2[gg] + \nu[gb] - \lambda 2[ggb]\end{aligned}$$

To close the model, we *condition on the state of the central site* (G) and approximate the triplets as follows. Under the assumption that the number of B and G neighbors of a G site are independent, $[ggb] \simeq \frac{[gg][gb]}{1-\rho}$ (in other words, the influence of an agent on the behavior of her second neighbor in a triplet is negligible). Next, we consider any (central) agent G influenced by a B neighbor provided that she already has another B neighbor and denote by $l_{(i)GB}$ the number of links GB of that G-agent, then $[bgb] \simeq \frac{E\left[l_{(i)GB}(l_{(i)GB}-1)\right]}{1-\rho} = \frac{E\left[l^2_{(i)GB}\right]-E\left[l_{(i)GB}\right]}{1-\rho}$; we assume then that $l_{(i)GB}$ is approximately Poisson distributed with mean $[gb]$ and second moment $E\left[l^2_{(i)GB}\right] = [gb]^2 + [gb]$, thus $[bgb] \simeq \frac{[gb]^2}{1-\rho}$. This same result can be obtained under the assumption that any two different B neighbors of the central agent G are independent: $[bgb] \simeq \frac{[gb][gb]}{1-\rho} = \frac{[gb]^2}{1-\rho}$ (as $[bg] = [gb]$). Finally, we simply rely on the expressions $\langle k_{GB} \rangle = \frac{[gb]}{(1-\rho)}$, $\langle k_{BB} \rangle = \frac{2[bb]}{\rho}$, and $\langle k_{GG} \rangle = \frac{2[gg]}{(1-\rho)}$ to obtain (4)-(6) after time differentiation and suitable algebraic manipulations. ■

[17] The term $-\lambda[gb]$ is the expected density of links GB that become BB because the node B "infects" her partner G at a rate λ; the term $-\lambda[bgb]$ denotes the expected density of triplets BGB where the pair BG becomes BB because the center node G is influenced by other B neighbor than the one involved in the first pair BG. By the same token, $2\lambda[ggb]$ is the expected density of triplets GGB where the pair GG becomes GB due to the influence of agent B at the end of the triplet (we must count it twice, as given any pair GG both end nodes can be connected to a B).

Proof of Proposition 1

By substitution, the system yields one equation in terms of $\langle k_{GB}\rangle$. We show under which conditions the solution of such equation is $\langle k_{GB}\rangle^* \geq 0$. Iff $\langle k_{GB}\rangle^* \geq 0$ $\rho^* \in [0,1]$. From eqs. 9, 11, and 12 in eq. 10 it follows that the long run solution $\langle k_{GB}\rangle^*$ is a zero of the function $f(\langle k_{GB}\rangle) = a\langle k_{GB}\rangle^4 + b\langle k_{GB}\rangle^3 + c\langle k_{GB}\rangle^2 + d\langle k_{GB}\rangle$ where:

$$a = \lambda^3 \frac{\tau(\eta+\gamma)}{2} \tag{17}$$

$$b = \lambda^2 \frac{\tau}{4}\left[(\eta+\gamma)(2\tau\gamma+3\tau\eta+6) + 2\lambda(\eta+\gamma-2)\right] \tag{18}$$

$$\begin{aligned} c = \lambda\tfrac{\tau}{4}[&(2\eta+(\eta+\gamma)(\tau\eta+2))(1+\tau(\eta+\gamma)) + (\eta+\gamma)(\tau\eta+2) \\ &-\lambda((4\tau-(\tau\eta+2))(\gamma+\eta)+2\tau\eta+8)] \end{aligned} \tag{19}$$

$$\begin{aligned} d = \tfrac{\tau}{4}[&(1+\tau(\eta+\gamma))(2+\tau(\eta+\gamma))\eta \\ &-\lambda((2-\eta+2\tau\eta)\tau(\gamma+\eta)+4+4\tau\eta)] \end{aligned} \tag{20}$$

The roots of the polynomial are four, of which $\langle k_{GB}\rangle^* = 0$ is always one solution (therefore, $\rho^* = 0$ is always a solution). The other three roots satisfy $a\langle k_{GB}\rangle^3 + b\langle k_{GB}\rangle^2 + c\langle k_{GB}\rangle + d = 0$. Given $\lambda > 0$, $\tau > 0$, $\eta \in (0,2)$ and $\gamma \in (2-\eta, \infty)$, $a > 0$ and $b > 0$, while $c \gtreqless 0$ and $d \gtreqless 0$. Next we show that there can be *at most one sign change* (in the polynomial of degree 3), thus by the Descartes' Rule of Signs there can be *at most* one strictly positive real root $\langle k_{GB}\rangle^* > 0$ ($\implies \rho^* > 0$). Consider eq. (19). The first term between brackets is strictly positive, while the factor multiplying λ (between the brackets):

$$\begin{aligned} &((4\tau-(\tau\eta+2))(\gamma+\eta)+2\tau\eta+8) \gtreqless 0 \\ \iff &\tau \gtreqless \tilde{\tau} = 2\tfrac{\eta+\gamma-4}{\gamma(4-\eta)+\eta(6-\eta)} > 0 \iff (\eta+\gamma) > 4 \end{aligned}$$

Hence, if $(\eta+\gamma) > 4$ and $\tau \in (0,\, 2\frac{\eta+\gamma-4}{\gamma(4-\eta)+\eta(6-\eta)}]$, the factor multiplying λ is ≤ 0 and therefore $\forall\lambda$, $c > 0$; otherwise $((\eta+\gamma) > 4 \wedge \tau > 2\frac{\eta+\gamma-4}{\gamma(4-\eta)+\eta(6-\eta)}$ or $(\eta+\gamma) \leq 4)$ it is strictly positive. In this latter case:

$$c \lesseqgtr 0 \iff \lambda \gtreqless \lambda_1 = \frac{(\eta+\gamma)\left[4(1+\tau\eta)+\tau(2+\tau\eta)(\eta+\gamma)\right]+2\eta}{2(4+\tau\eta)+(\gamma+\eta)(4\tau-\tau\eta-2)} > 0 \tag{21}$$

In the case of d (eq.20), for all $\tau > 0$:

$$d \lesseqgtr 0 \iff \lambda \gtreqless \lambda_2 = \eta\frac{2+3(\eta+\gamma)\tau+(\eta+\gamma)^2\tau^2}{4+(4\eta+(\eta+\gamma)(2-\eta))\tau+2\eta(\eta+\gamma)\tau^2} > 0$$

When condition (21) applies, $\lambda_1 > \lambda_2$:

$$\begin{aligned}
&\lambda_1 > \lambda_2 \iff \\
&2\eta^2\tau^4 (\eta+\gamma)^3 + 2\eta\tau^3 (6\eta+\gamma)(\eta+\gamma)^2 \\
&+\tau^2 (\eta+\gamma)\left(\eta^2 (26-\eta) + \gamma\eta (16-\eta) + 4\gamma^2\right) \\
&+2\tau\left(\gamma\eta (18+2\eta+\gamma) + 8\gamma^2 + \eta^2 (12+\eta)\right) \\
&+4\left((2+\eta)(\eta+\gamma) + 2\gamma\right) > 0
\end{aligned}$$

Therefore, the possible signs for c and d (for $\tau > 0$) are:

(i) $c > 0 \ \wedge \ d > 0 \iff \lambda < \lambda_2$

(ii) $c > 0 \wedge d = 0 \iff \lambda = \lambda_2$

(iii) $c > 0 \wedge d < 0 \iff \begin{cases} \left\{\lambda > \lambda_2 \wedge \eta+\gamma > 4 \wedge \tau \leq 2\frac{\eta+\gamma-4}{\gamma(4-\eta)+\eta(6-\eta)}\right\} \ \veebar \\ \left\{ \begin{array}{c} \lambda_1 > \lambda > \lambda_2 \wedge \\ \left\{\eta+\gamma \leq 4 \veebar \left\{\eta+\gamma > 4 \wedge \tau > 2\frac{\eta+\gamma-4}{\gamma(4-\eta)+\eta(6-\eta)}\right\}\right\} \end{array} \right\} \end{cases}$

(iv) $c = 0 \wedge d < 0 \iff \lambda = \lambda_1 > \lambda_2 \wedge \left\{ \begin{array}{c} \eta+\gamma \leq 4 \ \veebar \\ \left\{\eta+\gamma > 4 \wedge \tau > 2\frac{\eta+\gamma-4}{\gamma(4-\eta)+\eta(6-\eta)}\right\} \end{array} \right\}$

Cases (i) and (ii) imply there is *not* any strictly positive real root, (iii) and (iv), exactly one. Other combinations of signs are *not* possible because they are incompatible with $\lambda_1 > \lambda_2$. Therefore, the necessary and sufficient condition for the existence of a strictly positive solution is that $\lambda > \lambda_2$, which (after some algebraic manipulations) is equivalent to $\Phi_c(\eta, \gamma, \lambda, \tau) > 1$ in text. ■

Proof of Proposition 2

We show analytically the conditions under which $\rho^* = 0$ is asymptotically stable. The stability of the positive solution has been checked numerically, finding that it is stable whenever it exists. Given the dynamic system, the Jacobian evaluated in the long run solution, $\rho \to 0$,$\langle k_{GB} \rangle \to 0$,$\langle k_{BB} \rangle \to \frac{2}{2+\tau(\eta+\gamma)}$,$\langle k_{GG} \rangle \to \frac{2}{\eta}$, is:[18]

$$\begin{bmatrix} -1 & \lambda & 0 & 0 \\ 2\tau + \frac{2}{2+\tau(\eta+\gamma)} & \lambda\frac{2}{\eta} - \lambda - \tau(\eta+\gamma) - 1 & 0 & 0 \\ 2\tau - \frac{2}{\lambda} - 4 + \frac{4}{2+\tau(\eta+\gamma)} & 0 & -1-\tau(\eta+\gamma) & 0 \\ -2\tau - \frac{2}{\eta} & 2 - \lambda\frac{2}{\eta} & 0 & -\tau\eta \end{bmatrix}$$

The eigenvalues are:

$$\begin{aligned} r_1 &= -(1+\tau(\eta+\gamma)) < 0 \\ r_2 &= -\tau\eta < 0 \\ r_3 &= \tfrac{-m+\sqrt{m^2-4pq}}{2p} \\ r_4 &= \tfrac{-m-\sqrt{m^2-4pq}}{2p} \end{aligned}$$

with:

$$\begin{aligned} p &= (2+\tau(\eta+\gamma))\eta \\ m &= (2+\tau(\eta+\gamma))(\eta(2+\tau(\eta+\gamma)) - \lambda(2-\eta)) \\ q &= \eta(\eta+\gamma)(\eta+\gamma-2\lambda)\tau^2 + ((\eta+\gamma)(\lambda\eta+3\eta-2\lambda) - 4\lambda\eta)\tau - 2(2\lambda-\eta) \end{aligned}$$

Hence, if $r_3 < 0$ and $r_4 < 0$, $\rho^* = 0$ is stable, otherwise (if either $r_3 > 0$ or $r_4 > 0$) it is not.

First, note that given $p > 0$ and $\eta > 0$:

$$m^2 - 4pq = p^2\left(2 - \lambda + \tfrac{1}{\eta}(2\lambda - p)\right)^2 + 8p\lambda(\eta + \tau p) > 0.$$

Second, for $\eta > 0$, when $\Phi_c(\eta,\gamma,\lambda,\tau) < 1$, $q > 0$ and $m > 0$. To see this, denote by $\lambda_c : \Phi_c(\eta,\gamma,\lambda_c,\tau) = 1$ (uniquely determined and $\Phi_c(\eta,\gamma,\lambda_c,\tau) \gtreqless 1 \iff \lambda \gtreqless \lambda_c$), thus:

$$\begin{aligned} q \gtreqless 0 &\iff \lambda \lesseqgtr \lambda_c \text{ and} \\ m \gtreqless 0 &\iff \lambda \lesseqgtr \lambda_* = \tfrac{(2+\tau(\eta+\gamma))\eta}{2-\eta} \end{aligned}$$

with $\lambda_* > \lambda_c$ always ($\lambda_* > \lambda_c \iff 2 + 2\tau\eta(2+\tau(\eta+\gamma)) + \eta > 0$, trivially true). Therefore, when $\lambda < \lambda_c \implies \lambda < \lambda_* \implies q > 0$, $m > 0$. It follows that $\lambda < \lambda_c$ ($\Phi_c(\eta,\gamma,\lambda_c,\tau) < 1$) $\implies 0 < \sqrt{m^2-4pq} < \sqrt{m^2} \implies r_3 = \frac{-m+\sqrt{m^2-4pq}}{2p} < \frac{-m+\sqrt{m^2}}{2p} = 0$ and $r_4 = \frac{-m-\sqrt{m^2-4pq}}{2p} < 0$, hence $\rho^* = 0$ is stable.

On the other hand, if $\lambda > \lambda_c$ ($\Phi_c(\eta,\gamma,\lambda_c,\tau) < 1$), $q < 0$, while $m \gtreqless 0$. Whatever is the sign of m, $\sqrt{m^2-4pq} > 0$. If $m \le 0$, trivially, $r_3 = \frac{-m+\sqrt{m^2-4pq}}{2p} > 0$; if $m > 0$,

[18] The fact that analytically $\langle k_{BB} \rangle \to \frac{2}{2+\tau(\eta+\gamma)}$ is due to its asymptotic behavior; when $\rho \to 0$, $\lim_{\rho\to 0}\langle k_{BB} \rangle = \frac{2\ell_{BB}}{\rho N}$ is undetermined.

$r_3 = \frac{-m+\sqrt{m^2-4pq}}{2p} > \frac{-m+\sqrt{m^2}}{2p} = 0$, thus $\rho^* = 0$ is unstable. ∎

Proof of Proposition 3

First note that $\lambda_c(\cdot)$ is a continuous function of τ, as under our assumptions its denominator is always positive. Its first derivative is:

$$\frac{\partial \lambda_c}{\partial \tau} = \eta \frac{8\gamma + 2\eta(\eta+\gamma) + 8\gamma(\eta+\gamma)\tau + (\eta+\gamma)^2(2\gamma - \eta(\eta+\gamma))\tau^2}{(4 + 4\tau\eta + \tau(\eta+\gamma)(2-\eta) + 2\tau^2(\eta+\gamma)\eta)^2} \tag{22}$$

It follows that if $2\gamma - \eta(\eta+\gamma) \geq 0$ ($\iff \gamma \geq \frac{\eta^2}{2-\eta}$), $\frac{\partial \lambda_c}{\partial \tau} > 0 \; \forall \tau \geq 0$ up to its horizontal asymptote, $\lim_{\tau\to\infty} \lambda_c(\tau) = \frac{\eta+\gamma}{2}$. Its second derivative is:

$$\frac{\partial^2 \lambda_c}{\partial \tau^2} = -\frac{4\eta^2\left((2\gamma-\eta(\eta+\gamma))(\gamma+\eta)^3\tau^3 + 12\gamma(\gamma+\eta)^2\tau^2 + ((6\tau-1)(\gamma+\eta)+4)(4\gamma+\eta(\eta+\gamma)) + 2(\eta+\gamma)^2\right)}{(4+4\tau\eta+\tau(\eta+\gamma)(2-\eta)+2\tau^2(\eta+\gamma)\eta)^2} \tag{23}$$

Expression 23 is in general negative, unless γ is too large relatively to η, in which case it can be positive for τ very close to zero. For any value of η and γ, as a sufficient condition, it is strictly negative (thus $\lambda_c(\cdot)$ concave) for all $\tau > \frac{1}{6}$.

In the complementary region, $\gamma < \frac{\eta^2}{2-\eta}$, $\frac{\partial \lambda_c}{\partial \tau} = 0$ iff:

$$\tau^* = \frac{4\gamma + \sqrt{2\eta(\eta+\gamma)(\eta^2+\gamma\eta+2\gamma)}}{(\eta^2+\gamma\eta-2\gamma)(\eta+\gamma)} > 0$$

The second candidate is infeasible: it is always negative, since $\left(\eta^2 + \gamma\eta - 2\gamma\right) \gtrless 0 \implies 4\gamma - \sqrt{2\eta(\eta+\gamma)(\eta^2+\gamma\eta+2\gamma)} \lessgtr 0$. The second order condition (i.e. 23 evaluated at $\tau = \tau^*$) is:

$$\frac{\partial^2 \lambda_c}{\partial \tau^2}\Big|_{\tau=\tau^*} = \frac{\eta\left(2\gamma\left((\eta+\gamma)+2\tau^*\left(\frac{\eta+\gamma}{2}\right)^2\right) - \eta\left(\frac{\eta+\gamma}{2}\right)4\tau^*\left(\frac{\eta+\gamma}{2}\right)^2\right)}{\left(2+2\tau^*\eta+\tau^*\left(\frac{\eta+\gamma}{2}\right)(2-\eta)+\tau^{*2}(\eta+\gamma)\eta\right)^2} < 0$$
$$\iff 4\gamma - \tau^*(\eta+\gamma)\left(\eta^2+\gamma\eta-2\gamma\right) < 0$$

Replacing τ^*, the last inequality holds since:

$$4\gamma - \tau^*(\eta+\gamma)\left(\eta^2+\gamma\eta-2\gamma\right) = -\sqrt{2\eta(\eta+\gamma)(\eta^2+\gamma\eta+2\gamma)}$$

Therefore if $\gamma < \frac{\eta^2}{2-\eta}$, λ_c has a maximum for $\tau^* < \infty$. Trivially (because it is a global maximum), $\lambda_c(\tau^*) > \frac{\eta+\gamma}{2} = \lim_{\tau\to\infty} \lambda_c(\tau)$:

$$\lambda_c(\tau^*) - \frac{\eta+\gamma}{2} =$$
$$\frac{1}{2}\frac{\sqrt{2}\sqrt{\eta(\eta+\gamma)(\eta^2+\gamma\eta+2\gamma)}(\eta+\gamma)\left(\eta^2+\gamma\eta-2\gamma\right)^2}{\sqrt{2}\sqrt{\eta(\eta+\gamma)(\eta^2+\gamma\eta+2\gamma)}(((6-\eta)\eta^2+(4-\eta^2)\gamma)(\eta+\gamma)+4\gamma(\eta^2+\gamma\eta-2\gamma))+8\eta^2(\eta+\gamma)(\eta^2+\gamma\eta+2\gamma)} > 0.$$

∎

Proof of Proposition 4

We first describe the relevant values of λ that allow us to characterize the behavior of $\rho(\tau)$. Denote $\rho_0 = \lim_{\tau\to 0}\rho(\tau)$, $\rho_\infty = \lim_{\tau\to\infty}\rho(\tau)$.

(i) $\lambda = \frac{\eta}{2}$ is the minimum value of λ below which $\rho(\tau) = 0$ for any τ as for $\tau \to 0$, $\lambda_c(\tau)$ attains its minimum value, i.e. $\lim_{\tau\to 0}\lambda_c(\tau) = \frac{\eta}{2}$.

(ii) $\lambda = \hat{\lambda}$ is the value of λ such that $\frac{\partial\rho(\tau)}{\partial\tau}|_{\tau=0} \gtreqless 0$ iff $\lambda \gtreqless \hat{\lambda}$, where $\hat{\lambda}$ satisfies $\hat{\lambda} = \frac{(\eta+\gamma)}{2}\frac{2\left(3\eta+2\gamma\rho_0(\hat{\lambda})\right)}{\left((2-\eta)(\eta+\gamma)+4\eta+2\gamma\rho_0(\hat{\lambda})\right)\left(1-\rho_0(\hat{\lambda})\right)}$. This implicit condition is obtained by (implicit) differentiation of the following self-consistency condition:

$$\rho = 1 - \frac{\eta+\gamma}{2\lambda} + \frac{1}{2\tau}\left(\frac{\tau(1-\rho)+\frac{\rho}{\lambda(1-\rho)}}{\frac{\rho}{(1-\rho)}+\frac{\tau\eta}{2}} - \frac{1}{\lambda} + \frac{\tau\rho+\frac{\rho}{\lambda(1-\rho)}+1}{1+\frac{\tau(\eta+\gamma)}{2}} - 1 - \frac{\rho}{\lambda(1-\rho)}\right)$$

obtained from the long run system. Thus if $\lambda > \hat{\lambda}$ ($\lambda < \hat{\lambda}$), the slope of $\rho(\tau)$ at $\tau = 0$ is strictly positive (negative).

(iii) $\lambda = \frac{\eta+\gamma}{2}$ is the value of λ such that if $\lambda \le \frac{\eta+\gamma}{2}$, $\rho_\infty = 0$, otherwise, $\rho_\infty > 0$.

(iv) $\lambda = \frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2})$ is the value of λ such that $\rho_\infty \gtreqless \rho_0 \iff \lambda \gtreqless \frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2})$ (provided that $\lambda > \frac{\eta+\gamma}{2}$, so that both ρ_0 and ρ_∞ are strictly positive).

(v) $\lambda = \frac{\gamma}{\eta}$ together with the last value $\lambda = \frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2})$ define an interval of values of λ for which, provided that $\rho_\infty > 0$ ($\lambda > \frac{\eta+\gamma}{2}$), prevalence $\rho(\tau)$ is equal to ρ_∞ for some finite and strictly positive value of τ and the slope of $\rho(\tau)$ at such τ may be positive or negative. More precisely:

If $\lambda > \frac{\eta+\gamma}{2}$ and $\max\{\frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2}), \frac{\gamma}{\eta}\} > \lambda > \min\{\frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2}), \frac{\gamma}{\eta}\}$ then $\rho(\hat{\tau}) = \rho_\infty$ at $\hat{\tau} = 2\frac{\gamma+\lambda(\gamma+\eta)-2\lambda^2}{\eta(\eta+\gamma)\left(\lambda-\frac{\gamma}{\eta}\right)} > 0$.

As $\frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2}) \gtreqless \frac{\gamma}{\eta} \iff \gamma \lesseqgtr 2\frac{\eta^2}{2-\eta}$, we find that if $\gamma < 2\frac{\eta^2}{2-\eta}$, $\hat{\tau} > 0$ when $\frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2}) > \lambda > \frac{\gamma}{\eta}$ and the slope is $\frac{\partial\rho(\tau)}{\partial\tau}|_{\tau=\hat{\tau}} < 0$; if $\gamma > 2\frac{\eta^2}{2-\eta}, \hat{\tau} > 0$ when $\frac{\gamma}{\eta} > \lambda > \frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2})$ and the slope is $\frac{\partial\rho(\tau)}{\partial\tau}|_{\tau=\hat{\tau}} > 0$.

(vi) $\lambda = \lambda_c(\tau^*)$ is the maximum critical value of λ when function $\lambda_c(\tau)$ is non-monotonic, i.e. given that $\gamma < \frac{\eta^2}{2-\eta}$.

The order of these values of λ depends on the values of both decay rates η and γ. In particular:

If $\gamma < \frac{\eta^2}{2-\eta}$ (Region III):

$$\begin{aligned}&\min\{\tfrac{\eta}{2}, \tfrac{\gamma}{\eta}\} < \max\{\tfrac{\eta}{2}, \tfrac{\gamma}{\eta}\} < \tfrac{\eta+\gamma}{2} \\ &< \min\{\lambda(\tau^*), \tfrac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2})\} \\ &< \max\{\lambda(\tau^*), \tfrac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2})\} < \hat{\lambda}\end{aligned}$$

If $\frac{\eta^2}{2-\eta} < \gamma < 2\frac{\eta^2}{2-\eta}$ (Region II):

$$\frac{\eta}{2} < \frac{\eta+\gamma}{2} < \frac{\gamma}{\eta} < \frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2}) < \hat{\lambda}$$

If $\gamma > 2\frac{\eta^2}{2-\eta}$ (Region I):

$$\frac{\eta}{2} < \min\{\hat{\lambda}, \frac{\eta+\gamma}{2}\} < \max\{\hat{\lambda}, \frac{\eta+\gamma}{2}\} < \frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2}) < \frac{\gamma}{\eta}.$$

Note that what we call Region III coincides with the zone where $\lambda_c(\tau)$ is non-monotonic, while Regions I and II with the other, in which $\lambda_c(\tau)$ is always increasing. Next consider the two monotonic behaviors of $\rho(\tau)$.

For $\rho(\cdot)$ being *monotonically decreasing*, the following conditions should hold: $\lambda > \frac{\eta}{2}$ (s.t. $\rho_0 > 0$), $\lambda < \hat{\lambda}$ (s.t. the slope at $\tau = 0$ is negative). Additionally, if $\rho_\infty > 0$ something that occurs when $\lambda > \frac{\eta+\gamma}{2}$, necessarily $\rho_0 > \rho_\infty$ and $\rho(\tau)$ should *not* be equal to ρ_∞ for any finite τ (hence λ should not belong to the interval defined in (v) above). In this latter case, we would observe that $\rho(\cdot)$ decreases until $\rho_\infty > 0$. If instead $\rho_\infty = 0$ (i.e. $\lambda < \frac{\eta+\gamma}{2}$), $\rho(\tau)$ decreases until $\rho(\tau) = 0$ at $\tau = \tau_c$.

Now consider the order of these values within each region:

In Region III, $\rho(\cdot)$ decreases until $\rho(\tau_c) = 0$ when $\frac{\eta}{2} < \lambda < \frac{\eta+\gamma}{2}$. In Region II $\rho(\cdot)$ decreases until $\rho(\tau_c) = 0$ when $\frac{\eta}{2} < \lambda < \frac{\eta+\gamma}{2}$, and for $\frac{\eta+\gamma}{2} < \lambda < \frac{\gamma}{\eta}$ decreases until $\rho(\tau) \to \rho_\infty > 0$. At last, in Region I whenever $\min\{\hat{\lambda}, \frac{\eta+\gamma}{2}\} = \hat{\lambda}$ and $\frac{\eta}{2} < \lambda < \hat{\lambda}$, $\rho(\cdot)$ decreases until $\rho(\tau_c) = 0$; when otherwise for $\min\{\hat{\lambda}, \frac{\eta+\gamma}{2}\} = \frac{\eta+\gamma}{2}$, $\rho(\cdot)$ decreases until $\rho(\tau_c) = 0$ if $\frac{\eta}{2} < \lambda < \frac{\eta+\gamma}{2}$ and decreases until $\rho(\tau) \to \rho_\infty > 0$ for $\frac{\eta+\gamma}{2} < \lambda < \hat{\lambda}$. All together, $\rho(\cdot)$ monotonically decreases whenever $\lambda \in (\frac{\eta}{2}, \min\{\hat{\lambda}, \max\{\frac{\gamma}{\eta}, \frac{\eta+\gamma}{2}\}\}]$.

On the other hand, for $\rho(\cdot)$ being *monotonically increasing,* the following conditions should hold: $\lambda > \hat{\lambda}$ (s.t. the slope at $\tau = 0$ is positive), $\lambda > \frac{(\eta+\gamma)}{2}$ (s.t. $\rho_\infty > 0$); $\lambda > \frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2})$ (s.t. $\rho_\infty > \rho_0$), and $\rho(\tau)$ should *not* be equal ρ_∞ for any finite τ (λ should not belong to the interval defined in (v) above). It is easy to see that in Regions II and III this occurs when $\lambda > \hat{\lambda}$, while in Region I when $\lambda > \frac{\gamma}{\eta}$ (this is because if $\frac{(\eta+\gamma)}{4}(1+\sqrt{1+8\gamma/(\eta+\gamma)^2}) < \lambda < \frac{\gamma}{\eta}$, $\rho(\hat{\tau}) = \rho_\infty$). All together, $\rho(\cdot)$ monotonically increases if $\lambda > \max\{\hat{\lambda}, \frac{\gamma}{\eta}\}$. ■

Proof of Proposition 5

Consider the same relevant values of λ and their order within each region used in the Proof of Proposition 4. First of all note that in Region I, $\rho(\cdot)$ *cannot* display an interior *minimum* because when the slope $\frac{\partial\rho(\tau)}{\partial\tau}|_{\tau=0} < 0$ (i.e. $\lambda < \hat{\lambda}$), $\rho(\cdot)$ is *monotonically* decreasing. By the same token, in Regions II and III, $\rho(\cdot)$ *cannot* display an interior *maximum*, because when the slope $\frac{\partial\rho(\tau)}{\partial\tau}|_{\tau=0} > 0$ (i.e. $\lambda > \hat{\lambda}$), $\rho(\tau)$ is *monotonically* increasing.

The existence of maxima (minima) in Region I (II and III) can be then elicited following analogous arguments as in the Proof of Proposition 4. We provide here two examples.

First, consider the case of $\rho(\cdot)$ with a maximum, followed by the suppression of prevalence (as in Figure 5 panel b). The following conditions should hold: $\lambda < \frac{\eta+\gamma}{2}$ (s.t. $\rho_\infty = 0$) and the slope at $\tau = 0$ must be positive, i.e. $\lambda > \hat{\lambda}$. These conditions are necessary and sufficient to say that we will observe this behavior only in Region I whenever $\hat{\lambda} < \lambda < \frac{\eta+\gamma}{2}$ (which requires that $\min\{\hat{\lambda}, \frac{\eta+\gamma}{2}\} = \hat{\lambda}$).

Second, consider the existence of an interior minimum of such that $\rho_0 > \rho_\infty > \rho(\tau)_{\min} > 0$. In this case, we need $\lambda < \hat{\lambda}$. Also, we should observe that $\rho(\hat{\tau}) = \rho_\infty$ and that at $\hat{\tau} > 0$, with $\frac{\partial \rho(\tau)}{\partial \tau}|_{\tau=\hat{\tau}} < 0$, which only occurs if $\gamma < 2\frac{\eta^2}{2-\eta}$ when $\frac{(\eta+\gamma)}{4}(1 + \sqrt{1 + 8\gamma/(\eta+\gamma)^2}) > \lambda > \frac{\gamma}{\eta}$ (i.e. in Regions II and III). As $\hat{\lambda} > \frac{(\eta+\gamma)}{4}(1 + \sqrt{1 + 8\gamma/(\eta+\gamma)^2})$, the upper bound is given by $\frac{(\eta+\gamma)}{4}(1 + \sqrt{1 + 8\gamma/(\eta+\gamma)^2})$. But in Region III we need $\lambda > \lambda_c(\tau^*)$, otherwise we could observe the interval of $\rho(\tau) = 0$ (i.e. the minimum is not unique, but a connected set). All together, we observe this behavior in Region II for $\frac{(\eta+\gamma)}{4}(1 + \sqrt{1 + 8\gamma/(\eta+\gamma)^2}) > \lambda > \frac{\gamma}{\eta}$ and in Region III for $\frac{(\eta+\gamma)}{4}(1 + \sqrt{1 + 8\gamma/(\eta+\gamma)^2}) > \lambda > \lambda_c(\tau^*)$ (because $\lambda_c(\tau^*) > \frac{\gamma}{\eta}$).

Other cases (i.e. maximum without suppression of prevalence; minimum s.t. $\rho_\infty > \rho_0 > \rho(\tau)_{\min} > 0$, etc.) can be showed in the same way. Note that the principal difference between Regions II and III is that we would observe the interval of $\rho(\tau) = 0$ only in Region III for $\frac{\eta+\gamma}{2} < \lambda < \lambda_c(\tau^*)$. ■